

In the Clinic

Acute Gastrointestinal Bleeding

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CME Objective: To review current evidence for the prevention, presentation and diagnosis, treatment, and practice improvement of acute gastrointestinal bleeding.

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Acute gastrointestinal (GI) bleeding is common in both the outpatient setting and the emergency department. Annual U.S. incidence rates over the past decade are approximately 90–108 per 100 000 persons (1), leading to approximately 300 000 hospitalizations annually. Most cases are due to nonvariceal sources of bleeding (e.g., peptic ulcers) and continue to be associated with significant mortality (3–14%) and health economic burden (1–3). The incidence of nonvariceal bleeding may be decreasing in the West largely because of decreased incidence of *Helicobacter pylori* infection and increased awareness and implementation of ulcer-prevention strategies in users of nonsteroidal anti-inflammatory drugs (NSAIDs) (1). However, identifying patients with acute GI bleeding who are in danger of serious adverse events and establishing evidence-based treatment plans are essential in both primary and specialty care.

Prevention

Who is at risk for acute GI bleeding?

Risk factors for acute GI bleeding vary according to the site and cause. Upper GI bleeding most often results from peptic ulcer disease (approximately one quarter of all cases), the primary risk factors for which include NSAIDs and *H. pylori* infection and, less commonly, increased gastric acid production (e.g., the Zollinger-Ellison syndrome). Smoking (4–6), severe physiologic stress (7), and various host factors (e.g., genetic polymorphisms affecting cyclooxygenase and prostaglandin production) (8, 9) may increase risk for peptic ulcers even in persons without concomitant *H. pylori* infection or NSAID exposure. Although spicy foods may cause GI symptoms, there are no convincing data that they increase the risk for peptic ulcers. Other risk factors for acute upper GI bleeding include varices, esophagitis, vascular abnormalities (e.g., angioectasias, arteriovenous malformations, Dieulafoy lesions), Mallory-Weiss tear from protracted vomiting, and benign and malignant neoplasms (10–13).

Lower GI bleeding, historically defined as bleeding from a source distal to the ligament of Treitz, also results from several causes with distinct risk factors. Diverticulosis is the most common cause of hematochezia, accounting for up to half of all cases, particularly in patients older than 65 years. Other risk factors associated

with lower GI bleeding include inflammatory bowel disease, infectious colitis, neoplasia, angioectasias, and benign anorectal disease (14).

Approximately 10–20% of patients with GI bleeding have “obscure” bleeding, defined as an unknown cause despite evaluation with esophagogastroduodenoscopy (EGD), colonoscopy, and radiographic small bowel imaging. Approximately half of these patients have recurrent or persistent bleeding and are further subclassified as obscure-overt (passage of visible blood with melena or hematochezia) or obscure-occult (iron-deficiency anemia and/or positive for fecal occult blood) (15). Many such patients have bleeding sources in the small intestine, now sometimes referred to as “mid-GI bleeding” (between the ligament of Treitz and the ileocecal valve). Angioectasia is the most common cause of small-bowel bleeding in the West, accounting for 70–80% of cases (16).

Can acute GI bleeding be prevented?

Prevention of acute GI bleeding depends on the risk factors and causes. For example, reducing use of NSAIDs and administering antacid treatment with H₂-inhibitors or proton-pump inhibitors (PPIs) prevents peptic ulcer bleeding. Prophylactic acid suppression should be considered in selected hospitalized patients who are at increased risk for gastroduodenal ulceration and bleeding, including

1. Hreinnsson JP, Kalaitzakis E, Gudmundsson S, Björnsson ES. Upper gastrointestinal bleeding: incidence, etiology and outcomes in a population-based setting. *Scand J Gastroenterol*. 2013;48:439-47. [PMID: 23356751]
2. Yavorski RT, Wong RK, Maydonovitch C, et al. Analysis of 3,294 cases of upper gastrointestinal bleeding in military medical facilities. *Am J Gastroenterol*. 1995;90:568-73. [PMID: 7717312]
3. Targownik LE, Nabalamba A. Trends in management and outcomes of acute nonvariceal upper gastrointestinal bleeding: 1993-2003. *Clin Gastroenterol Hepatol*. 2006;4:1459-1466. [PMID: 17101296]
4. McColl KE, el-Nujumi AM, Chittajallu RS, et al. A study of the pathogenesis of *Helicobacter pylori* negative chronic duodenal ulceration. *Gut*. 1993;34:762-8. [PMID: 8314508]
5. Kurata JH, Nogawa AN. Meta-analysis of risk factors for peptic ulcer. Nonsteroidal antiinflammatory drugs, *Helicobacter pylori*, and smoking. *J Clin Gastroenterol*. 1997;24:2-17. [PMID: 9013343]
6. Talamini G, Zamboni G, Cavallini G. Antral mucosal *Helicobacter pylori* infection density as a risk factor of duodenal ulcer. *Digestion*. 1997;58:211-7. [PMID: 9243115]
7. Barkun AN, Bardou M, Pham CQ, Martel M. Proton pump inhibitors vs. histamine 2 receptor antagonists for stress-related mucosal bleeding prophylaxis in critically ill patients: a meta-analysis. *Am J Gastroenterol*. 2012;107:507-20. [PMID: 22290403]
8. Arisawa T, Tahara T, Shibata T, et al. Association between genetic polymorphisms in the cyclooxygenase-1 gene promoter and peptic ulcers in Japan. *Int J Mol Med*. 2007;20:373-8. [PMID: 17671743]

patients who are mechanically ventilated or who have coagulopathy or thrombocytopenia, traumatic brain or spinal cord injury, or a history of burns (7, 17). In patients with chronic liver disease, nonselective beta-blockers (such as propranolol or nadolol) to reduce portal hypertension and endoscopic interventions, such as band ligation (18), can effectively prevent variceal bleeding. No specific

measures can prevent diverticular bleeding or bleeding related to angioectasias. Although high-fiber diets are often recommended, the role of fiber in the pathogenesis or progression of diverticulosis is debatable. Surgical intervention for diverticulosis is not routinely done for prevention, because risk for diverticular bleeding is low relative to the morbidity of prophylactic surgical resection (19).

Prevention... Risk factors for, and therefore prevention of, acute GI bleeding depends on the site and cause of bleeding. In general, minimizing use and appropriate prescribing of NSAIDs, antiplatelet agents, and anticoagulants, as well as judicious primary and secondary prophylactic acid suppression in selected patients, are effective measures for ulcer-related upper GI bleeding. Nonselective beta-blockers and endoscopic therapy for esophageal and gastric varices are effective for primary and secondary prevention of variceal bleeding. Few measures are helpful in preventing lower GI bleeding, except for reducing exposure to NSAIDs, anticoagulants, and antiplatelets.

CLINICAL BOTTOM LINE

What are the symptoms and signs of acute GI bleeding? Can they help localize the site of bleeding?

GI bleeding can present with myriad signs and symptoms (Table 1). The most indolent forms may present as severe anemia. Manifestations include fatigue; dizziness; pallor; and rarely end-organ complications, such as unstable angina. Brisk bleeding from the upper or lower GI tract can present with more specific manifestations, such as visualized blood, syncope, or other symptoms of hypotension.

Distinguishing between upper and lower GI bleeding can help guide initial diagnosis and therapy. Upper GI bleeding is more likely to cause nausea and dyspepsia, whereas lower GI bleeding is more likely to result in altered bowel habits, lower abdominal pain, or rectal discomfort. Hematemesis is exclusive to upper GI bleeding. Melena, which can be caused by as little as 50 mL of blood, usually results from upper GI bleeding; however,

it can occur with small-bowel bleeding and even slowly bleeding right colonic lesions. The source of hematochezia is usually the colon. Up to 10% of upper GI bleeding episodes present with hematochezia, which is often associated with signs and symptoms of hemodynamic instability.

What are the common causes of upper and lower GI bleeding?

Major causes of GI bleeding are shown in the Box. Upper GI bleeding is approximately 5 times as common as lower GI bleeding, with mortality rates of 10%. Upper GI bleeding can be classified as non-variceal or variceal.

Methods allowing visualization and treatment of lesions deep in the small intestine have recently been developed. This has led to increasing use of the term “mid-GI bleeding” (MGIB) (20). MGIB can present with occult bleeding (iron-deficiency anemia and stools positive for occult

Presentation and Diagnosis

9. Malaty HM, Graham DY, Isaksson I, Engstrand L, Pedersen NL. Are genetic influences on peptic ulcer dependent or independent of genetic influences for *Helicobacter pylori* infection? *Arch Intern Med*. 2000;160:105-9. [PMID: 10632311]
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Table 1. History and Physical Examination Elements of Patients with Acute Upper Gastrointestinal Bleeding

Category	Physical Examination Finding	Notes
History	Hematemesis	Indicates bleeding proximal to the ligament of Treitz
	Melena	Should not be confused with dark stool of another etiology (test with guaiac to confirm the presence of blood)
	Bloody diarrhea	10% of patients will have bleeding sources proximal to the ligament of Treitz
Physical examination	Presyncope and/or syncope	Indicates loss of significant blood volume
	Hypotension (systolic blood pressure 90 mm Hg)	Indicates severe intravascular volume loss (≥50%), assuming normal baseline systolic blood pressures
	Tachycardia (≥120 beats/min)	Indicates severe intravascular volume loss (≥50%)
	Orthostatic changes in blood pressure (≥10 mm Hg) or heart rate (≥30/min)	Indicates loss of 20–25% of intravascular volume
	Nasogastric aspirate shows blood or coffee ground–like material	Indicates an upper gastrointestinal source of bleeding
	Pallor	Subjective/poor indicator without corroborative evidence
	Perioral telangiectasias	Suggestive of hereditary hemorrhagic telangiectasia syndrome
	Skin abnormalities	Stigmata of cirrhosis, pigmented lip lesions, acanthosis nigricans, vascular anomalies

From Bjorkman DJ, Eisen GM. Gastrointestinal bleeding, non-variceal upper. <http://pier.acponline.org/physicians/diseases/d184/tables/d184-thp.html>. (Date accessed 26 May 2009). PIER. Philadelphia: American College of Physicians; 2009.

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13. Cook DJ, Fuller HD, Guyatt GH, et al. Risk factors for gastrointestinal bleeding in critically ill patients. Canadian Critical Care Trials Group. *N Engl J Med*. 1994;330:377-81. [PMID: 8284001]
14. Strate LL. Lower GI bleeding: epidemiology and diagnosis. *Gastroenterol Clin North Am*. 2005;34:643-64. [PMID: 16303575]
15. Rondonotti E, Marano R, Petracchini M, de Franchis R, Pennazio M. The American Society for Gastrointestinal Endoscopy (ASGE) diagnostic algorithm for obscure gastrointestinal bleeding: eight burning questions from everyday clinical practice. *Dig Liver Dis*. 2013;45:179-85. [PMID: 22921043]
16. Pennazio M. *Techniques in Gastrointestinal Endoscopy*. 2012;14:94-99.
17. Alain BB, Wang YJ. Cushing's ulcer in traumatic brain injury. *Chin J Traumatol*. 2008;11:114-9. [PMID: 1837716]

blood) or overt bleeding (melena, hematochezia). More frequently encountered sources of MGIB are vascular lesions (e.g., angioectasias, Dieulafoy lesions, arteriovenous malformations), ulcerating disorders (e.g., NSAID–enteropathy and inflammatory bowel disease), and neoplasms (e.g., neuroendocrine tumors, GI stromal tumors, lymphoma, and primary or metastatic carcinomas).

The most common source of lower GI bleeding is diverticulosis, reflective of the high prevalence of this condition in the adult population; however, only a few patients with diverticulosis have diverticular bleeding. Other common causes of lower GI bleeding include colonic polyps, cancer, colitis (due to inflammatory bowel disease, infection, or ischemia), vascular lesions (angioectasia, radiation proctitis), and anorectal sources (hemorrhoids, anal fissures) (21).

Can risk for adverse outcomes be predicted in patients with acute GI bleeding? Which patients may be evaluated as outpatients, and which require the emergency department or hospitalization? Risk stratification can help determine the disposition of a patient

with acute GI bleeding (e.g., outpatient vs. inpatient management) and provide prognostic information. Predictors of rebleeding risk and mortality in patients vary according to the cause of bleeding; however, several factors portend a poorer prognosis. Adverse outcomes have also been associated with chronic alcoholism and active cancer (22).

The International Consensus Upper Gastrointestinal Bleeding Conference Group advocates use of validated risk-stratification tools to facilitate triage of patients with acute upper GI bleeding by prognostication based on risk for poor clinical outcomes (e.g., rebleeding, need for emergent surgery, or mortality) (22). The Rockall scoring system (23) and the Glasgow–Blatchford Scale (24) (Table 2) are 2 commonly used risk-stratification systems that incorporate clinical, laboratory, and/or endoscopic parameters. The Glasgow–Blatchford and the “Clinical” Rockall stratification systems are preendoscopic scoring systems and use clinical and laboratory information to help predict the need for hospitalization or endoscopic intervention. Additional risk stratification by incorporating

some endoscopic information to the Clinical Rockall scale leads to the Full Rockall scale, which helps predict risk for adverse outcomes from acute upper GI bleeding. Additional stratification schemes for upper and lower GI bleeding have been proposed (25–28). Outpatient management may be appropriate for persons with low risk scores (Rockall score of 0–2 or Glasgow–Blatchford score of 0), whereas those with high risk scores require inpatient care (24, 29–34). Admission to an intensive care unit (ICU) should be considered for patients with evidence of brisk, active bleeding, such as hemodynamic instability (systolic blood pressure [BP] <100 or pulse >100, orthostatic hypotension, evidence of shock), or other clinical parameters associated with high risk for rebleeding and mortality. To date, there are no widely accepted validated scoring systems specific to acute lower GI bleeding.

What should the initial diagnostic evaluation for possible acute GI bleeding include?

The initial evaluation of a patient with acute GI bleeding should include a focused history and physical examination. The history should include associated signs and symptoms; use of NSAIDs, antiplatelet agents, anticoagulants, selective serotonin reuptake inhibitors, and beta-blockers (because of issues of risk assessment with tachycardia and resuscitation implications); prior GI bleeding episodes; and comorbid conditions. Vital signs on postural changes should be assessed, and stool should be examined. Resting hypotension (systolic BP ≤90 mm Hg) or tachycardia (≥120 bpm) indicates severe intravascular volume loss (≥50%). An increase of ≥30/min in the pulse or severe lightheadedness when rising from a supine position also indicates significant volume loss (35, 36).

Immediate laboratory tests should include a complete blood count, prothrombin and partial thromboplastin times, platelet count, blood type and

Table 2. Risk Stratification Tools for Upper Gastrointestinal Bleeding

Scoring System	Points
<i>Rockall Risk: Total score = sum of all category scores; risk category: high (>5), intermediate (3–4), low (0–2)</i>	
Age, y	
>80	2
60–79	1
<60	0
Shock	
SBP <100 mm Hg	2
SBP >100 mm Hg	1
No shock	0
Comorbid conditions	
Renal failure, liver failure, widespread cancer	2
Cardiac failure, ischemic heart disease	1
No other comorbid conditions	0
Diagnosis on EGD	
Upper gastrointestinal cancer	2
All other diagnoses	1
No lesion on EGD	0
Stigmata of recent hemorrhage	
Blood in upper gastrointestinal tract, adherent clot, visible vessel	2
No stigmata	0
<i>Glasgow–Blatchford: Total score = sum of all category scores; a score of zero suggests low risk and the safety of outpatient care</i>	
Blood urea nitrogen (mmol/L)	
>25	6
10–<25	4
8–<10	3
6.5–<8	2
<6.5	0
Hemoglobin (g/dL)	
<10 in men and women	6
10–<12 in men	3
10–<12 in women/12–<13 in men	1
≥12 in women or ≥13 in men	0
SBP (mm Hg)	
<90	3
90–99	2
100–109	1
>110	0
Other markers	
Cardiac failure	2
Hepatic disease	2
Presentation with syncope	2
Presentation with melena	1
Pulse >100/min	1

EGD = esophagogastroduodenoscopy; SBP = systolic blood pressure.

Major Causes of Gastrointestinal Bleeding

- Inflammatory**
 - Peptic ulcer disease
 - Esophagitis or esophageal ulceration
 - Diaphragmatic hernia (Cameron erosions)
 - Inflammatory bowel disease
- Benign and malignant neoplasms**
 - Primary gastrointestinal tract neoplasms, at any site
 - Metastatic deposits in the gastrointestinal tract, at any site
- Vascular anomalies**
 - Gastroesophageal varices
 - Angioectasias
 - Dieulafoy lesion
 - Gastric antral vascular ectasia (GAVE, also known as watermelon stomach)
 - Radiation proctopathy
- Drug-induced**
 - Aspirin
 - Nonsteroidal anti-inflammatory drugs
- Miscellaneous**
 - Colonic diverticulosis
 - Postpolypectomy
 - Mallory–Weiss tear
 - Meckel diverticulum

From Rajan E, Ahlquist D. Gastrointestinal bleeding. In: Dale DC, Federman DD, eds. ACP Medicine, 3rd ed. New York: WebMD; 2007:863.

18. Garcia-Tsao G, Sanyal AJ, Grace ND, Carey W; Practice Guidelines Committee of the American Association for the Study of Liver Diseases. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology*. 2007;46:922–38. [PMID: 17879356]
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20. Okazaki H, Fujiwara Y, Sugimori S, et al. Prevalence of mid-gastrointestinal bleeding in patients with acute overt gastrointestinal bleeding: multi-center experience with 1,044 consecutive patients. *J Gastroenterol*. 2009;44:550–5. [PMID: 19360374]

crossmatch in anticipation of blood transfusion, and a routine chemistry panel. An increased ratio of blood urea nitrogen to creatinine (>25:1 if in mg/dL) suggests an upper GI source of bleeding (35, 36).

Nasogastric or orogastric aspiration may confirm an upper GI source of bleeding and provide prognostic information on bleeding activity and severity. However, the procedure is uncomfortable; yields false-negative results in approximately 15% of patients with active bleeding; and although it may lead to earlier performance of endoscopy, it has not been proven to improve clinical outcomes (30, 37). Some expert panels recommend it in selected patients but do not provide selection criteria (22, 38). Others state that it is not required in patients with suspected upper GI bleeding for diagnosis, prognosis, visualization, or therapeutic effect (39). As a result, use of gastric aspiration should be dictated by institutional preference and practice.

When should a gastroenterologist be consulted in the evaluation of acute GI bleeding?

A gastroenterologist should be consulted early to consider prompt endoscopy for patients with GI bleeding and to facilitate patient triage. EGD or colonoscopy or both are the initial diagnostic tests of choice for identification and treatment of specific bleeding lesions. Clinical presentation dictates which procedures are done. EGD is appropriate for patients with melena and hematemesis and a subset of patients with hematochezia resulting from an upper GI source.

In upper GI bleeding, early endoscopy (within 24 hours of admission) has a greater impact than delayed endoscopy on some clinical outcomes, such as blood transfusion requirements and hospital length of stay (40). Although emergent endoscopy (<6–8 hours) does not seem to further reduce rebleeding, hospital length of stay, transfusion

requirements, rescue surgery, or mortality (41–43), urgent endoscopy (<12 hours) is indicated in patients with suspected variceal bleeding (39, 44, 45). It also provides valuable information for appropriate patient triage (see “Can risk for adverse outcomes be predicted in patients with acute GI bleeding?”).

What is the role of prokinetic medications before upper endoscopy in patients with acute GI bleeding?

Prokinetic medications (such as erythromycin and, to a lesser extent, metoclopramide) administered intravenously 20 to 120 minutes before upper endoscopy has the potential to facilitate clearance of blood and clots from the stomach, thus improving visualization in patients with upper GI bleeding. However, they do not seem to alter important clinical outcomes, including diagnostic rates, need for transfusions or surgery, or hospital length of stay. Routine use before upper endoscopy is not advocated and should be reserved for patients with red blood hematemesis or blood in the nasogastric aspirate (22, 39, 46).

What adjunctive tests can help evaluate (and/or treat) patients with acute GI bleeding without an identified source on EGD or colonoscopy?

Patients without a demonstrable bleeding source on good-quality upper endoscopy or colonoscopy have “obscure” GI bleeding. Small-bowel barium radiography, “push” enterography, technetium-labeled red blood cell scan, and angiography have historically been considered as subsequent diagnostic tests, however with low diagnostic yield. Wireless video capsule endoscopy (VCE) has become most gastroenterologists’ test of choice for patients with obscure GI bleeding. The diagnostic yield ranges from 30–50% (7); however, no VCE systems can provide hemostatic interventions, and many institutions are unable to perform urgent inpatient

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22. Barkun AN, Bardou M, Kuipers EJ, et al. International consensus recommendations on the management of patients with nonvariceal upper gastrointestinal bleeding. *Ann Intern Med.* 2010;152:101-13. [PMID: 20083829]
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26. Strate LL, Orav EJ, Syngal S. Early predictors of severity in acute lower intestinal tract bleeding. *Arch Intern Med.* 2003;163:838-43. [PMID: 12695275]
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VCE. Another option is angiography, which allows for therapeutic intervention (embolization) if a lesion is localized; however, it requires active bleeding to be diagnostic. Computed tomography angiography or computed tomography/magnetic resonance enterography are alternative diagnostic options that identify sources of obscure bleeding in

approximately 50–70% and 25–70% of cases, respectively (16). Some centers offer advanced deep enteroscopic procedures, which are low-risk and enable visualization and therapeutics deep within the small intestine, essentially eliminating the need for high-risk intraoperative enteroscopy for patients with suspected small bowel pathology.

Presentation and Diagnosis... GI bleeding can present with myriad signs and symptoms, ranging from asymptomatic to overt hematemesis or hematochezia, and can be due to a number of causes virtually anywhere along the GI tract. Initial evaluation, including history and physical examination and routine laboratory tests, can help to narrow the differential diagnosis.

CLINICAL BOTTOM LINE

What interventions should be started immediately in patients with acute GI bleeding?

Regardless of the source, initial management of a patient with acute GI bleeding involves rapid diagnostic assessment and aggressive resuscitation with isotonic fluids via 2 large-bore (18-gauge or larger) peripheral IV catheters. Early intensive resuscitation has been associated with decreased mortality from GI bleeding (48). The goal of resuscitation is to maintain tissue perfusion until bleeding resolves spontaneously or is controlled by more definitive therapy. Smaller catheters (e.g., 20- or 22-gauge) do not allow rapid fluid administration. Longer central venous catheters increase resistance to fluid flow, thereby slowing the rate at which fluids may be given. Patients with emesis who are unable to protect the airway from aspiration should be intubated. Isotonic IV fluids, such as normal saline or lactated Ringer solution, should be given immediately, with the goal of replenishing intravascular volume as quickly as blood has been lost and as rapidly as is tolerated by the patient's underlying

cardiovascular function, with the goal of normal, stable vital signs.

Observational studies and small controlled trials have suggested that blood transfusion to a target hemoglobin level of 9–10 mg/dL might actually be detrimental for some patients with hypovolemic anemia, including those with GI bleeding.

A recent randomized controlled trial (49) compared a "restrictive" vs. a "liberal" transfusion strategy in patients with acute upper GI bleeding. The threshold for transfusion of red blood cells was a hemoglobin value <7 g/dL in the restrictive transfusion group (target range, 7–9 g/dL) and 9 g/dL in the liberal transfusion group (target range, 9–11 g/dL). Overall, the restrictive transfusion strategy was associated with a significantly greater probability of survival at 6 weeks, lower incidence of further bleeding, fewer adverse events, fewer total units of blood transfused, and less need for rescue therapy. Subgroup analysis revealed that the survival advantage of the restrictive strategy was limited to Child–Pugh class A and B cirrhosis (not class C). Patients with "massive exsanguinating bleeding," the acute coronary syndrome, symptomatic peripheral vasculopathy, stroke, transient ischemic attack, and lower GI bleeding were excluded from the trial, and

Treatment

29. Chen IC, Hung MS, Chiu TF, Chen JC, Hsiao CT. Risk scoring systems to predict need for clinical intervention for patients with non-variceal upper gastrointestinal tract bleeding. *Am J Emerg Med.* 2007;25:774-9. [PMID: 17870480]
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35. Cappel MS, Friedel D. Initial management of acute upper gastrointestinal bleeding: from initial evaluation up to gastrointestinal endoscopy. *Med Clin North Am.* 2008;92:491-509. [PMID: 18387374]

a restrictive transfusion strategy may not be applicable to every patient.

Although coagulopathy has been shown to be a risk factor for nonvariceal upper GI bleeding, data are limited and conflicting (48, 50, 51). Acknowledging the paucity of published data on the topic, the recently published International Consensus Recommendations advise that coagulopathy in patients receiving anticoagulants be treated, but that management should not delay therapeutic endoscopy unless the INR is supratherapeutic (>2.5). Furthermore, this approach should not be generalized to patients with cirrhosis because the INR, at any threshold, cannot predict bleeding risk in these patients (22). In addition, a systematic review of published studies reported a lack of data on which to recommend optimal platelet counts for patients with GI bleeding. Based on expert opinion, they recommended targeting values of 50 000/ μL in the absence of platelet dysfunction, or 100 000/ μL if functional platelet dysfunction is suspected (52).

How should acute upper GI bleeding due to peptic ulcer disease be managed?

The initial diagnostic approach to bleeding peptic ulcers is the same as for other causes of GI bleeding. Endoscopy is nearly 100% specific and $>90\%$ sensitive; can be done at the bedside in the emergency department, endoscopy unit, or ICU; and allows biopsy to assess the cause of the ulcer (e.g., *H. pylori* infection or an ulcer within an adenocarcinoma). Endoscopists use the Forrest classification to describe peptic ulcers and to predict rebleeding risk associated with different ulcer appearances. Approximately 50% of ulcers have an appearance associated with a low probability of rebleeding (i.e., clean ulcer base or flat pigmented spot in the ulcer base). Patients with these ulcers require only pharmacologic treatment. By contrast, ulcers with adherent clots, nonbleeding visible

vessels, or active bleeding are associated with a high-risk for continued or recurrent bleeding and warrant pharmacologic treatment and such endoscopic interventions as thermal methods; injection with vasoconstrictive agents, such as epinephrine; and mechanical hemostasis with various clips or sealants; or some combination (53, 54). Strong evidence indicates that monotherapy with epinephrine injection alone is less effective than combination therapy (injection plus thermal therapy, or clips followed by injection) (53, 54).

In patients with peptic ulcer bleeding, PPI therapy (bolus followed by infusion) before endoscopy has been shown to decrease the likelihood of high-risk stigmata on subsequent endoscopy and to reduce the likelihood of requiring an intervention during endoscopy. It may also be associated with shorter length of stay in the hospital. However, preendoscopic PPI therapy has not been shown to reduce mortality or the need for rescue surgery or rebleeding rates when endoscopic therapy is consistently delivered (55, 56) (Appendix Figure, available at www.annals.org). Thus, preendoscopic PPIs should be considered but should not delay early endoscopy (within the first 24 h) or replace resuscitation because its effect on outcomes is minor at best (57). After endoscopy, hemodynamically stable patients without serious comorbid conditions who are found to have low-risk ulcers (e.g., clean-based, flat pigmented spot) can generally be discharged early on once-daily oral PPIs (58).

A meta-analysis of randomized trials of high-dose IV PPI therapy (80 mg bolus then 8 mg/h infusion for 72 h after endoscopic therapy) after successful endoscopic therapy for ulcers in patients with high-risk stigmata (i.e., active bleeding, nonbleeding visible vessel or adherent clot, or Forrest classification IA, IB, IIa, or IIb) (54, 59) concluded that mortality, need for surgery, and rebleeding is reduced (relative risk 0.41, 0.43, and 0.40, respectively) compared with endoscopy alone. Another meta-analysis also concluded that for

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patients with ulcer bleeding, PPIs reduce re-bleeding and need for surgery or repeated endoscopic procedures and improve mortality in the highest-risk patients (59).

The optimal dose and route of acute PPI administration remain unclear, but patients with high-risk lesions having endoscopic therapy should receive in-hospital therapy (IV, high-dose therapy remains the regimen with the best evidence [22]) for 3 days, mirroring the period during which risk for rebleeding is greatest (60, 61). In the absence of comparative data, a once-daily oral PPI is recommended after completion of 72 hours of IV therapy (22, 30, 39). H₂-receptor blockers are not as effective and should not replace PPIs for acute management of bleeding ulcers.

How should acute esophageal variceal bleeding be treated?

Acute bleeding from esophageal varices is frequently life-threatening because it can be severe, difficult to control, and rarely resolves spontaneously. It usually occurs in patients with end-stage liver disease. Esophageal varices are caused by significant portal hypertension; therefore, bleeding occurs under high pressure and is often brisk. In addition, patients have synthetic liver dysfunction and coagulopathy. Under these circumstances, acute management requires rapid and aggressive interventions, including fluid resuscitation, bleeding control, and efforts aimed at reducing portal pressures.

Intravascular volume replacement should occur as with any patient with acute GI hemorrhage. Resuscitation of patients with end-stage liver disease is often difficult due to hypoalbuminemia and extravasation of fluid from the intravascular space. Some experts advocate preferential use of blood products and albumin for intravascular volume resuscitation, because crystalloid fluids deliver a sodium load to patients who are typically already total-body sodium overloaded and tend to eventually exacerbate ascites. Patients should be

monitored closely for adverse effects of volume replacement, including pulmonary edema. Targeting a hemoglobin level between 7–8 g/dL might prevent excessive restitution of blood volume and subsequent increases in portal pressure (62).

There are no definitive guidelines for management of coagulopathy and thrombocytopenia in patients with acute variceal hemorrhage. Reversal of coagulopathy requires administration of fresh frozen plasma, although the volume necessary may be prohibitive. Recombinant factor VIIa as an alternative means of normalizing the prothrombin time has been proposed, but randomized, controlled trials have not shown a consistent advantage (62). Because prothrombin time–INR is not a reliable indicator of the coagulation status in patients with cirrhosis, recommendations regarding management of coagulopathy/thrombocytopenia cannot be made on the basis of available data (63).

Antibiotic prophylaxis reduces infectious complications and decreases rebleeding with acute variceal bleeding. A short-term quinolone course (e.g., up to 7 days of norfloxacin or ciprofloxacin) is recommended. Data from a few studies suggest that ceftriaxone may prevent bacterial infections better than norfloxacin, and IV ceftriaxone (1 g/day) may be preferable in patients with advanced cirrhosis and/or in regions with high prevalence of quinolone-resistant organisms (62, 64).

Variceal bleeding can often be controlled by both medical and endoscopic means. Medical therapy primarily involves infusion of octreotide, a somatostatin analogue that causes splanchnic vasoconstriction and reduced portal pressure. Numerous trials of octreotide have yielded equivocal results, but it is recommended in combination with endoscopic therapies to control variceal bleeding and reduce risk for recurrence (18). Octreotide should

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be given as a bolus dose of 50 µg followed by continuous IV infusion of 50 µg/hour for a total of 3 to 5 days, although many experts currently prefer a 5-day regimen (63).

Endoscopy should be done within 12 hours in patients with known or suspected varices who present with upper GI bleeding. There are 2 primary endoscopic interventions: sclerotherapy and band ligation. Sclerotherapy involves injection of a sclerosing agent (e.g., sodium morrhuate or ethanolamine) into varices. Band ligation involves wrapping elastic bands over the varices in the distal esophagus. Clinical trials have shown that compared with sclerotherapy, banding has more prolonged benefit; lower rates of rebleeding, mortality, and complications (such as esophageal stricture formation, perforation, and mediastinitis); and reduced need for endoscopic treatments (65). Thus, band ligation has become the acute treatment of choice in most institutions.

For patients with variceal bleeding refractory to medical and endoscopic therapy, balloon tamponade (e.g., with a Sengstaken–Blakemore tube) may be used as a temporizing measure. The balloons should be inflated for no more than 12 hours. More definitive treatments include transjugular intrahepatic portosystemic shunt (TIPS) placement with a PTFE stent and surgical interventions. In patients at high risk for treatment failure (e.g., Child–Pugh class C score < 14 or class B with active bleeding), TIPS should be placed within 72 hours (63). Salvage (rescue) TIPS placement is associated with a high mortality rate. These therapies are effective and often render surgical interventions unnecessary. Surgical options include portosystemic shunting, esophageal transection, and liver transplantation. Mortality approaches 80% for patients with continued bleeding from esophageal varices.

For patients with bleeding gastric varices, initial clinical and pharmacologic management is similar to that for esophageal varices. However, decision making regarding definitive treatment options, particularly for isolated gastric varices, is more complex. Band ligation, injection sclerotherapy, and TIPS have been reported and are sometimes used in clinical practice. More recent therapeutic developments for treatment of gastric varices include endoscopic injection of biological glues and a radiologic procedure called balloon-occlusion retrograde transvenous obliteration. Recently, self-expanding esophageal stents have been used to staunch bleeding, particularly in refractory cases; however, further studies are needed (63).

How should patients with acute lower GI bleeding from colonic diverticulosis be treated?

Initial management of colonic diverticular hemorrhage is the same as that for other causes of GI bleeding, including fluid resuscitation, blood transfusion, and diagnostic testing. Colonoscopy should be done to try to localize the bleeding, although with brisk hemorrhage, localization can be difficult due to poor visualization. The timing of the colonoscopy remains controversial; however, it is usually done within 12–24 hours of presentation with a rapid colonic preparation that seems to be safe in this setting (66). The most important contribution of colonoscopy is usually exclusion of other causes. In rare cases, it can also be therapeutic if a visible vessel or adherent clot is noted. More typically, however, bleeding occurs from multiple sites and may be intermittent, so the opportunity for intervention is limited. Other studies that may localize the bleeding include nuclear imaging and angiography (19).

Bleeding resolves spontaneously in approximately 75% of patients with

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diverticular hemorrhage; recurrent bleeding occurs in 25–35% of patients. If bleeding does not resolve spontaneously or with angiographic intervention, surgical resection is the remaining therapeutic option. Approximately 20% of patients hospitalized for diverticular bleeding require surgery. Segmental/subtotal colectomy may be required if bleeding can be localized and endoscopic therapy is not feasible. It may also be necessary if lower GI bleeding remains nonlocalizable, despite the risk for ongoing bleeding (due to failure to resect the actual bleeding site) and may be associated with significant morbidity and mortality (67).

What is the role of angiography?

Angiographic interventions include local administration of vasopressin and embolization of the source. Vasopressin is a potent vasoconstrictive agent, and local instillation can control bleeding in up to 80% of patients; however, rebleeding occurs on cessation of the infusion in up to 50% of patients. Therefore, vasopressin may be most useful as a temporizing measure. Although there are no absolute contraindications, vasopressin should be used with caution in patients with coronary artery disease or peripheral vascular disease because of the risk for vasoconstriction at sites other than the target lesion. Vasopressin can also cause cardiac arrhythmia and hyponatremia.

Embolization is a common intervention for control of active hemorrhaging. It involves injection of sealant materials, such as gel foam or polyvinyl alcohol, or mechanical devices, such as coils and temporary balloons. Embolization is effective in up to 80% of cases. The primary contraindication is poor collateral blood supply. The major complications are intestinal ischemia and mechanical complications of the arterial cannulation (such as local hematoma, arterial dissection, or pseudoaneurysm).

How should therapy for acute GI bleeding be monitored?

Continued monitoring is required to assess whether bleeding has stopped. Tachycardia may be an early warning of recurrent bleeding, followed soon by hypotension. Hemoglobin levels should be checked on a serial basis at least every several hours initially to assess for stability after blood transfusions; levels that do not increase by approximately 1 g/unit of transfused packed red blood cells may indicate ongoing blood loss. Additional blood transfusions and diagnostic testing (e.g., repeated endoscopy) should be considered if the patient has evidence of ongoing blood loss. Similarly, platelet count and coagulation should be measured serially to assess the need for repeated transfusions. Patients requiring multiple transfusions of red blood cells should be monitored for hypocalcemia.

When should a surgeon be consulted for the management of a patient with acute GI bleeding?

Advances in medical and endoscopic therapies have resulted in fewer patients requiring surgery for acute GI bleeding. However, surgical consultation should be considered early in the evaluation and management of patients with severe bleeding. Surgery is indicated when life-threatening bleeding continues, hemodynamic compromise continues despite initial aggressive resuscitation, or bleeding cannot be stopped by endoscopic or angiographic means. The choice of surgery depends on bleeding location and comorbid conditions. Localization of the bleeding site is critical for surgical planning.

What follow-up outpatient evaluations are required in patients who have experienced acute GI bleeding?

Specific guidelines for management of patients after discharge other than those addressing secondary prophylaxis are lacking. In general, decisions regarding timing of postdischarge outpatient visits and repeated blood

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work are based on risk-stratification. Early outpatient evaluation is appropriate for patients with high-risk endoscopic findings, significant comorbid conditions, recent severe bleeding episode, and those in need of decision-making pertaining to antiplatelet or anticoagulant medication use. Conversely, the remaining low-risk patients usually do not require early outpatient evaluation. Patients should be advised to report symptoms or signs of recurrent bleeding. With the exception of select clinical scenarios (see below), empirical repeated or “surveillance” endoscopy is not required for most patients particularly if the bleeding source is associated with a low-risk for recurrent bleeding (e.g., Mallory–Weiss tears, postpolypectomy bleeding).

Patients with cirrhosis who survive variceal hemorrhage but do not have further therapy are at significant risk for rebleeding (60%) and death (approximately 33%) within 1–2 years of the initial bleeding episode. These outcomes are improved using combined endoscopic variceal band ligation and beta-blockers (which are often given once there has been no evidence of hemorrhage for at least 24 hours, barring any contraindications). Patients who have shunt surgery or TIPS do not require further preventive measures. The American Association for the Study of Liver Disease suggests repeating endoscopy at 7- to 14-day intervals until eradication of esophageal varices has been achieved (usually requires 2–4 sessions); and once varices are eradicated, repeating endoscopy every 3–6 months initially to evaluate for recurrence and need for repeated band ligation (18). Eventually, the interval can be increased to 6–12 months.

For patients that recover from gastric ulcer bleeding, surveillance endoscopy 6 to 12 weeks after the last bleeding episode to exclude a non-healing ulcer (which raises concern for cancer) is common in clinical practice. However, the literature

diverges on this topic, reporting inadequate outcomes and cost-effectiveness to support use of this strategy for every patient. Society guidelines advocate an individualized approach (68). Surveillance endoscopy is most appropriate for patients with suspicious ulcers on initial endoscopy in whom initial biopsies were negative or were not done, persistent symptoms despite a course of antisecretory medications, absence of a defined cause, or patients from demographic regions with high incidence of gastric carcinoma. If *H. pylori* infection has not been assessed via prior gastric mucosal biopsies, serology, or stool antigen evaluation, gastric mucosal biopsies for *H. pylori* testing should be considered. Literature pertaining to detailed management of patients with *H. pylori* is available (69). Bleeding sources that may require additional endoscopic therapy include gastric antral vascular ectasia (GAVE, or “watermelon stomach”) and radiation proctopathy.

How long should antisecretory therapy be continued?

Antisecretory therapy for patients with small (<1 cm), uncomplicated ulcers can usually be discontinued after 4–6 weeks in the absence of ongoing ulcer-related symptoms. Maintenance therapy should be considered for higher-risk patients (complicated, recurrent, or large ulcers). In patients with *H. pylori*-associated ulcers, therapy is often continued until eradication of infection is confirmed, or indefinitely if attempts at *H. pylori* eradication have failed. Instructions for patients with alternative causes of bleeding are made on a case-by-case basis.

What instructions do patients require after acute GI bleeding?

Specific guidelines for management of patients after discharge are lacking and should be individualized. Patients should be educated regarding the signs and symptoms of recurrent bleeding and the anticipated benefit and duration of targeted therapies.

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Patients with bleeding ulcers associated with *H. pylori* infection should be encouraged to complete a full course of *H. pylori* therapy and to have testing, such as a urea breath test or *H. pylori* stool antigen assay, to confirm successful eradication of the bacteria. For patients suspected of having NSAID-related ulcer bleeding, discontinuation is preferable but sometimes not feasible. If a patient must resume NSAIDs, a cyclooxygenase-2-selective NSAID at the lowest effective dose plus a daily PPI is

recommended (70). Similarly, patients with a bleeding ulcer while receiving low-dose aspirin therapy for secondary prevention of established cardiovascular disease should resume therapy as soon as possible after cessation of bleeding (within 1 week, ideally as early as 3–5 days). Data are less robust for management of aspirin therapy after acute bleeding when aspirin is being used for primary prevention of cardiovascular events, and the risk and benefits must be weighed on an individual basis.

Treatment... Treatment of GI bleeding is highly dependent on the cause and severity. Initial evaluation and management in all cases should include history and physical examination, with simultaneous stabilization interventions, such as placement of IV access and IV fluid resuscitation. Emergent endoscopy (e.g., within 6 hours) is rarely indicated, but urgent endoscopy (e.g., within 12 h) in selected circumstances can be considered, and is mandatory if variceal bleeding is suspected. Administering a PPI is warranted in patients with suspected peptic ulcer disease but should not delay endoscopy. Transfusion practices should target a hemoglobin threshold of 7–8 g/dL. Out-patient follow-up should be individualized based on the cause of bleeding and the estimated risk for rebleeding.

CLINICAL BOTTOM LINE

What do professional organizations recommend with regard to the prevention, diagnosis and treatment of acute GI bleeding?

The International Consensus Upper Gastrointestinal Bleeding Conference Group recommends early risk stratification and early diagnostic endoscopy in most patients with upper GI bleeding.

The American Society of Gastroenterology recommends early colonoscopy for diagnosis of acute lower GI bleeding, with angiography and tagged red blood cell scanning in patients with active bleeding and nondiagnostic colonoscopies. If surgical intervention is contemplated, preoperative localization of bleeding is desirable.

In patients with obscure acute GI bleeding in whom EGD and

colonoscopy have been unrevealing, the American Society of Gastroenterology recommends early evaluation of the small bowel by VCE or angiography, with CT angiography, CT enteroscopy, and deep enteroscopy as secondary considerations, depending on availability and expertise at the institution.

What measures do stakeholders use to evaluate the quality of care for patients with acute GI bleeding?

The Agency for Healthcare Research and Quality has identified mortality rate as the primary quality indicator for care of patients with GI bleeding. An expert panel independently developed a list of 26 quality indicators for non-variceal upper GI bleeding, categorized as preendoscopic, endoscopic, and postendoscopic factors (70).

Practice Improvement

70. Kanwal F, Barkun A, Gralnek IM, et al. Measuring quality of care in patients with nonvariceal upper gastrointestinal hemorrhage: development of an explicit quality indicator set. *Am J Gastroenterol.* 2010;105:1710-8. [PMID: 20686458]

In the Clinic Tool Kit

Acute Gastrointestinal Bleeding

PIER Module

<http://pier.acponline.org/physicians/diseases/d184/d184.html>

PIER module on gastrointestinal (GI) bleeding from the American College of Physicians.

Patient Information

<http://pier.acponline.org/physicians/diseases/d184/d184-pi.html>

Patient information that appears on the next page for duplication and distribution to patients.

www.nlm.nih.gov/medlineplus/gastrointestinalbleeding.html

www.nlm.nih.gov/medlineplus/tutorials/uppergiendoscopy/btm/index.htm

www.nlm.nih.gov/medlineplus/spanish/tutorials/uppergiendoscopy/btm/index.htm

Resources related to GI bleeding from the National Institutes of Health's MedlinePLUS, including an interactive tutorial on upper GI endoscopy, in English and Spanish.

www.gi.org/physician-resources/brochures/

Patient brochure from the American College of Gastroenterology (ACG) on understanding ulcers, common pain medications, and GI Bleeding.

Clinical Guidelines

<http://annals.org/article.aspx?articleid=745521>

International consensus recommendations for managing patients with nonvariceal upper gastrointestinal bleeding, published in *Annals of Internal Medicine* in 2010.

www.sign.ac.uk/guidelines/fulltext/105/index.html

Clinical guideline on the management of acute upper and lower GI bleeding from the Scottish Intercollegiate Guidelines Network in 2008.

<http://circ.ahajournals.org/content/118/18/1894.full>

Consensus document on reducing the GI risks of antiplatelet therapy and NSAID use from the American College of Cardiology Foundation (ACCF), ACG, and American Heart Association (AHA) in 2008.

<http://content.onlinejacc.org/article.aspx?articleid=1143980>

Consensus document on concomitant use of proton pump inhibitors and thienopyridines, an update from the ACCF, ACG, and AHA in 2010.

Diagnostic Tests and Criteria

<http://pier.acponline.org/physicians/diseases/d184/tables/d184-tlab.html>

List of laboratory and other studies for acute upper GI bleeding from PIER.

<http://pier.acponline.org/physicians/diseases/d184/tables/d184-t1.html>

Glasgow–Blatchford screening tool to assess the likelihood that a patient with an acute upper GI bleeding will need medical intervention.

<http://pier.acponline.org/physicians/diseases/d184/tables/d184-t2.html>

Rockall scoring system for identifying patients at risk for adverse outcome after acute upper GI bleeding.

In the Clinic

THINGS YOU SHOULD KNOW ABOUT GASTROINTESTINAL BLEEDING

In the Clinic
Annals of Internal Medicine

What is gastrointestinal (GI) bleeding?

- Bleeding in the digestive tract.
- The upper digestive tract includes the esophagus, stomach, and upper portion of the small intestine.
- The lower digestive tract includes lower portion of the small intestine, large intestine (also called the colon), and anus.
- Most causes of bleeding are curable or controllable, but some may be life-threatening if left untreated.

What causes bleeding in the digestive tract?

Causes of bleeding in the upper digestive tract include:

- Peptic ulcers from *Helicobacter pylori* infections or long-term use of nonsteroidal anti-inflammatory drugs, such as aspirin and ibuprofen.
- Varices (enlarged veins) in the lower esophagus that rupture and bleed.
- Tears in the lining of the esophagus or inflammation in the lining of the stomach (gastritis) or esophagus (esophagitis).
- Cancerous or noncancerous (benign) growths.

Causes of bleeding in the lower digestive tract include:

- Diverticular disease (diverticula are irregular pouches that develop in the colon wall).
- Colitis (inflammation of the colon) or angiodysplasia (abnormalities in blood vessels of the intestine).
- Hemorrhoids (ruptured veins in the anus or rectum) or fissures (anal cuts or tears).
- Cancerous or noncancerous (benign) growths.

What are the signs and symptoms?

- Vomiting bright-red blood or vomit that looks like coffee grounds indicates bleeding in upper digestive tract.
- Black or tarry stool or stool that contains dark or bright red blood indicates bleeding in upper or lower digestive tract.



- Chronic bleeding (light bleeding that continues for a long time or starts and stops) may lead to fatigue, lethargy, and shortness of breath over time.
- Acute bleeding (heavy bleeding) may lead to dizziness or faintness, shortness of breath, abdominal pain, and shock.

How is it treated?

- Medical imaging techniques, such as endoscopy or angiography, may be used to locate the source of the bleeding inside of the digestive tract and to stop the bleeding.
- Surgery may be needed if these interventions do not work.
- Your doctor will try to prevent future bleeding by treating the condition that is causing the bleeding.

For More Information

<http://digestive.niddk.nih.gov/ddiseases/pubs/bleeding/>
<http://digestive.niddk.nih.gov/Spanish/pubs/bleeding/index.aspx>
Information on bleeding in the digestive tract from the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), in English and Spanish.

http://digestive.niddk.nih.gov/ddiseases/pubs/lowergi/Lower_GI_Series_T_508.pdf
Information on the lower GI x-rays ordered to help diagnose problems of the large intestine from the NIDDK.

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INTERNAL MEDICINE | Doctors for Adults

1. A 58-year-old man is evaluated in the emergency department for painless bright-red blood per rectum that began 3 hours ago. The bleeding was accompanied by syncope. He has a history of rheumatoid arthritis. His current medications are adalimumab, methotrexate, and ibuprofen.

On physical examination, temperature is 37.2°C (99.0°F), blood pressure is 88/58 mm Hg, pulse rate is 132/min, and respiration rate is 24/min.

Abdominal examination is normal. Rectal examination discloses bright-red blood in the rectal vault. Nasogastric tube aspirate shows no evidence of blood or coffee-ground material.

Laboratory studies reveal a hemoglobin level of 7.3 g/dL (73 g/L).

Emergency intravenous fluid resuscitation is begun.

Which of the following is the most appropriate diagnostic test to perform next?

- A. Colonoscopy
- B. Tagged red blood cell scan
- C. Upper endoscopy
- D. Video capsule endoscopy

2. A 46-year-old man is evaluated for a 3-week history of painless occasional bright-red rectal bleeding. He has no fatigue, lightheadedness, weight loss, or abdominal pain. His stools are frequently firm, occasionally hard, and there is no change in the frequency or consistency of bowel movements. He has never been screened for colorectal cancer.

On physical examination, temperature is 37.2°C (98.9°F), blood pressure is 132/78 mm Hg, and pulse rate is 84/min. Digital rectal examination yields a stool sample that is positive for occult blood; the examination is otherwise normal.

Anoscopy reveals a few internal hemorrhoids without active bleeding.

Laboratory studies show a blood hemoglobin level of 14 g/dL (140 g/L).

Which of the following is the most appropriate management of this patient?

- A. Banding of hemorrhoids
- B. Colonoscopy
- C. Fiber supplementation without further evaluation
- D. Home fecal occult blood testing

3. A 60-year-old man hospitalized for advanced cirrhosis complicated by ascites and encephalopathy is evaluated for massive hematemesis and hypotension. The patient's medications are spironolactone, furosemide, and lactulose.

On physical examination, temperature is 35.6°C (96°F), blood pressure is 80/50 mm Hg, pulse rate is 146/min, and respiration rate is 20/min. The patient has just vomited red blood and has large-volume ascites; the stool is brown and positive for occult blood. Laboratory studies show hemoglobin of 9 g/dL (90 g/L), platelet count of 60 000/μL ($60 \times 10^9/L$), and INR of 3.

In addition to rapid volume resuscitation, which of the following is the most appropriate management of this patient?

- A. Arteriography
- B. Esophagogastroduodenoscopy
- C. Intravenous nadolol
- D. Mesocaval shunt
- E. Transjugular intrahepatic portosystemic shunt

4. A 78-year-old woman is evaluated in the hospital after being admitted 5 days ago for a 2-week history of abdominal pain and nausea. She has also had black, tarry stools for the past 36 hours. On day 1, esophagogastroduodenoscopy showed a clean-based bleeding gastric ulcer that was positive for *Helicobacter pylori* infection; the ulcer was treated with injection therapy and coagulation therapy with probe cautery, and proton-pump inhibitor therapy was initiated. The bleeding did not stop, and esophagogastroduodenoscopy was repeated on day 3 with endoclip therapy. The bleeding continued, and the patient has received eight 8 of packed erythrocytes.

On physical examination on day 5, temperature is 37.2°C (99.0°F), blood pressure is 95/50 mm Hg, pulse rate is 103/min, and respiratory rate is 16/min. Rectal examination reveals melanic stool. Laboratory studies reveal hemoglobin of 10.8 g/dL (108 g/L); all other tests, including coagulation parameters, are normal.

Which of the following is the most appropriate next step in the management of this patient?

- A. Bleeding scan
- B. *Helicobacter pylori* eradication therapy
- C. Intravenous octreotide
- D. Surgery

Questions are largely from the ACP's Medical Knowledge Self-Assessment Program (MKSAP, accessed at http://www.acponline.org/products_services/mksap/15/?pr31). Go to www.annals.org/intheclinic/ to complete the quiz and earn up to 1.5 CME credits, or to purchase the complete MKSAP program.