CME

Assessing upper gastrointestinal bleeding in adults

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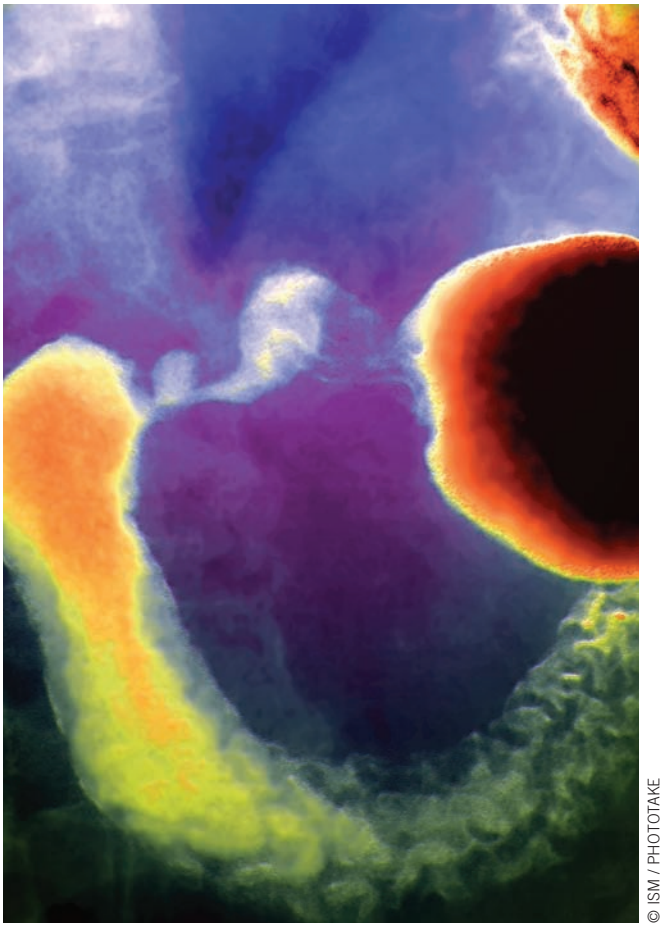
ABSTRACT

Acute upper gastrointestinal (GI) bleeding is a potentially life-threatening condition requiring accurate, prompt, and appropriate patient evaluation and management. Clinicians of all specialties must know the best practices for preventing and managing upper GI bleeding. This article focuses on assessing and managing adults with acute nonvariceal upper GI bleeding.

Keywords: upper gastrointestinal bleeding, hematemesis, melena, gastritis, Mallory-Weiss tears, Blatchford scale

Learning objectives

- Recognize the signs and symptoms of upper GI bleeding.
- Describe an evidence-based assessment strategy for adults with acute non-variceal upper GI bleeding.
- Develop therapeutic management plans for patients with upper GI bleeding.



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Acute upper gastrointestinal (GI) bleeding, defined as hemorrhage in the GI tract proximal to the ligament of Treitz, is a potentially life-threatening condition requiring accurate, prompt, and appropriate patient evaluation and management. Bleeding from the upper GI tract is four times as common as from the lower GI tract and is a major cause of morbidity, particularly among patients with comorbid illnesses (which is the case in up to 50% of patients).[1,2] Mortality for upper GI bleeding is 6% to 10%.[1] Upper GI bleeding causes significant clinical and economic burden in the United States, accounting for 300,000 to 400,000 hospitalizations, 30,000 deaths, and healthcare costs of more than $2 billion annually.[3,4] Given the repercussions of upper GI bleeding and complications of rebleeding or continued bleeding, clinicians in all specialties must know the best practices for preventing and managing upper GI bleeding. This article focuses on assessment of adults with acute nonvariceal upper GI bleeding, reviews the differential diagnosis and pathophysiology of common causes, and describes guidelines for selecting appropriate diagnostic tests and evidence-based therapeutic management.

EVALUATING PATIENTS

The initial assessment of patients with upper GI bleeding focuses on assessing severity and urgency of bleeding and identifying high-risk patients who require rapid intervention to minimize morbidity and mortality.[4] Patients with acute, active, and severe upper GI bleeding, significant

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Key points

- Bleeding from the upper GI tract is more common than lower GI tract bleeding, and is potentially life-threatening.
- Assess the severity of bleeding and patient comorbidities, looking for symptoms such as syncope, confusion, and clammy extremities that point to hemodynamic compromise.
- Peptic ulcer disease is the most common cause of upper GI bleeding, accounting for nearly half of all cases.
- EGD is the method of choice for controlling bleeding.

comorbidities (such as cerebrovascular or cardiovascular disease, diabetes, coronary artery disease, chronic kidney disease, lung disease, or liver disease), history of coagulopathy, and/or unstable vital signs warrant emergent clinical assessment and management. An abbreviated history can be obtained during emergency resuscitation and revisited upon stabilization. Focus on the GI tract, relevant history, and comorbid conditions. Patients may have difficulty quantifying bleeding, so elicit symptoms of blood loss (such as orthostasis; dizziness; syncope; confusion; cold, clammy extremities; dyspnea; angina; and palpitations) that suggest hemodynamic compromise.

Hematemesis and melena are classic upper GI bleeding symptoms found in 50% and 75% of patients with gross upper GI bleeding, respectively.[3] Remember that typical lower GI bleeding symptoms such as hematochezia arise from upper GI bleeding sources 14% of the time.[1] Upper GI bleeding must be excluded when hematochezia is accompanied by signs of hypovolemia or hypoperfusion.[4] In stable, asymptomatic patients, differentiate upper GI bleeding from other causes of darkened stools, such as iron supplements, bismuth, beets, and oropharyngeal bleeding. Assess for previous history of upper GI bleeding, the cause of that bleeding, and past assessment or treatment. About 60% of patients with a history of upper GI bleeding are bleeding from the lesion previously identified.[4]

Assess medication and psychosocial history for agents that promote gastrotoxicity (**Table 1**). Patients taking antacids, histamine$_2$-antagonists, or proton pump inhibitors (PPIs) may have gastritis, esophagitis, or peptic ulcer disease. Assess the patient for GI symptoms. Abdominal pain and dyspepsia are commonly reported in patients with upper GI bleeding. Patients with gastritis or peptic ulcer disease usually report chronic gnawing or burning epigastric pain after meals. Classically, patients with gastric ulcers experience abdominal pain shortly after eating; those with duodenal ulcers experience pain 1 to 2 hours after meals and/or during the night. Patients with peptic ulcer disease also may report belching, bloating, or heartburn. Be sure to review systems for underlying causes or symptoms associated with upper GI bleeding. Fever and chills support infectious or inflammatory causes.

TABLE 1. Gastrotoxic medications and substances that promote upper GI bleeding

- Alcohol
- Tobacco
- Cocaine, which is associated with GI ischemia and infarction and hemorrhage, and may be a cause of upper GI bleeding in young men without significant comorbidities
- Aspirin and NSAIDs
- Glucocorticosteroids
- Anticoagulants and antiplatelet agents
- Tetracyclines, bisphosphonates, quinidine preparations, iron compounds, and potassium chloride, which taken orally may lead to "pill esophagitis"

A history of vomiting, coughing, or retching suggests Mallory-Weiss syndrome (bleeding from a mucosal tear at the gastroesophageal junction). Involuntary weight loss, anemia, weakness, dysphagia, and early satiety are indicative of malignancy. Sore throat, heartburn, regurgitation, and dysphagia or odynophagia may indicate esophagitis.

Physical examination Continuous monitoring of the patient's vital signs and hemodynamics provide serial measurements of hemodynamic stability, blood loss, and compensation. Resting tachycardia suggests mild-to-moderate hypovolemia.[4] Signs of hypovolemic shock include systolic BP less than 90 mm Hg, tachycardia, decreased pulse pressure, and cool, clammy extremities with thready, weak pulses. Orthostatic hypotension signifies a total blood volume deficit of 15% or more, and supine hypotension indicates a volume loss of 40% or more.[4] Signs of chronic liver disease include jaundice, spider angiomata, palmar erythema, gynecomastia, hepatomegaly, coagulopathy and ascites. Skin ecchymosis, petechiae, purpura, gingival bleeding, hematuria, or wound or IV site bleeding suggest coagulopathy. Consider disseminated intravascular coagulation in patients with major trauma or gram-negative sepsis, and ICU patients.

Inspect the patient's abdomen for distension, ascites, caput medusa, abnormal pulsations, and masses. Hyperactive bowel sounds are common as proximal blood irritates the GI tract and induces peristalsis. Hypoactive bowel sounds suggest bowel ischemia, ileus, or obstruction. Abdominal tenderness often is limited to gastritis, peptic ulcer disease, and malignancy, which usually localizes to the epigastric region. Severe tenderness, guarding, rigidity, and rebound tenderness suggest GI perforation. Assess for organomegaly and enlarged lymph nodes, specifically Virchow left supraclavicular, Irish anterior axillary, and Sister Mary Joseph periumbilical nodes characteristic of GI malignancy. Examine the rectum for frank or occult bleeding, hemorrhoids, and anal fissures.

DIFFERENTIAL DIAGNOSIS

Peptic ulcer disease and erosive gastritis The most common cause of upper GI bleeding, peptic ulcer disease accounts for 40% to 50% of cases.[4] A peptic ulcer is a discrete defect in the gastric or duodenal wall extending at least as deeply as the muscularis mucosa. Erosive gastritis is indistinguishable from peptic ulcer disease in clinical presentation, and will be discussed together. Duodenal ulcers are much more common than gastric ulcers, but incidence of bleeding is identical for both.[1] Commonly, bleeding is caused by the erosion of an artery in the base of the ulcer and stops spontaneously in 80% of patients.[1] The patient also may have dyspepsia, anemia, and symptoms of gastric outlet obstruction. Consider the differential diagnosis of esophagitis in patients presenting with dysphagia, heartburn, and upper GI bleeding—causes of esophagitis include peptic stricture, autoimmune, infectious, caustic, trauma, tumor, iatrogenic, medication, or radiation-induced esophageal injury.

Peptic ulcer disease is strongly associated with NSAID use and *Helicobacter pylori* infection, though current incidence patterns reveal that peptic ulcer disease from long-term NSAID therapy is increasing and is the predominant cause of peptic ulcer disease leading to upper GI bleeding. *H. pylori* disrupts the mucous barrier and has a direct inflammatory effect on the gastric and duodenal mucosa (**Figure 1**). *H. pylori* is found in 71% of bleeding duodenal ulcers and 93% of nonbleeding ulcers.[1]

NSAIDs interrupt prostaglandin synthesis by increasing hydrochloric acid and mucin secretion, leading to an impaired mucosal defense and repair. Cyclooxygenase-2 (COX-2) selective NSAIDs possess half the risk of ulcer formation as COX-1 selective NSAIDs. Screen patients for factors that increase their risk of upper GI bleeding, including use of nonselective high-dose NSAIDs, past GI injury from NSAIDs, history of *H. pylori,* alcohol abuse, smoking, chronic renal failure, and concurrent use of NSAIDs with high-dose aspirin, steroids, anticoagulants, bisphosphonates, or selective serotonin reuptake inhibitors.[4]

Stress gastritis is the most common cause of acute upper GI bleeding in critically ill patients, accounting for 1.5% to 8.5% of cases.[4] Enhanced sympathetic activity shunts blood flow from the stomach, altering local mucosal protective barriers and inducing diffuse superficial mucosal inflammation and erosions. Two risk factors for upper GI bleeding in critically ill patients are mechanical ventilation for more than 48 hours and coagulopathy.[4] Other risks include resuscitation from shock, significant surgery or major/multiple trauma, acute respiratory distress syndrome, sepsis, multisystem organ failure, neurologic injury, and severe burns.[4] The bleeding is usually mild and self-limiting and rarely progresses to life-threatening hemorrhage. Stress ulcers associated with head injury and increased intracranial pressure are called Cushing ulcers; those in patients with severe burns are called Curling ulcers.

Mallory-Weiss tears Mallory-Weiss tears account for 5% to 15% of cases of upper GI bleeding.[4] These mucosal lacerations of the esophagus or stomach fundus may be caused by forceful vomiting, retching, straining, lifting, coughing, CPR, or blunt abdominal trauma. They are most common in middle-aged men with history of alcohol abuse, but also are associated with severe gastroenteritis, history of hiatal hernia, hyperemesis gravidarum, and bulimia nervosa. Physical examination findings are nonspecific. Mallory-Weiss tears resolve spontaneously in up to 90% of patients; about 7% require further treatment such as endoscopic sclerotherapy, esophageal clips, band ligation, or surgical oversewing of the tear.[1]

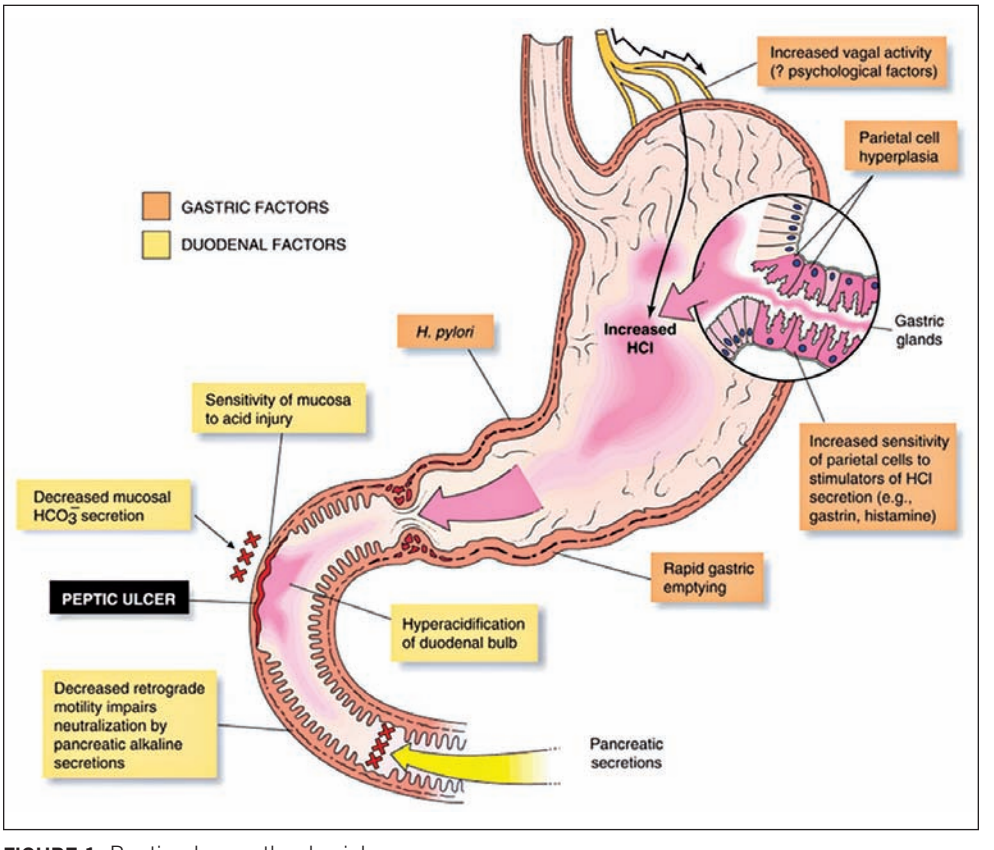

FIGURE 1. Peptic ulcer pathophysiology

GI malignancy Gastric cancer is the 14th most common cancer in the United States and seventh leading cause of cancer deaths, accounting for over 21,000 new cases and more than 10,000 deaths annually.[1] Worldwide, gastric cancer is the second most common cause of cancer death; rates of the disease are highest in Asia and parts of South America. Symptoms of gastric cancer reflect advanced disease: indigestion, nausea, vomiting, early satiety or postprandial fullness, anorexia, dysphagia, hematemesis, iron-deficiency anemia, unintended weight loss, stomach enlargement, and lymphadenopathy. Chronic infection with *H. pylori* is the strongest risk factor for gastric cancer. Other risks include a diet rich in pickled, salted, or smoked foods; smoking; alcohol consumption; a history of gastric ulcers; previous gastric surgery; pernicious anemia; and radiation exposure.

Progressive dysphagia for solids and later liquids over weeks to months is the most common presenting symptom for esophageal carcinoma, and should be strongly suspected in patients who also have weight loss, anorexia, and iron-deficiency anemia. Other manifestations include epigastric or chest pain, odynophagia, bleeding, hoarseness, hepatomegaly, lymphadenopathy, and respiratory symptoms, all of which suggest invasive or metastatic disease. Smoking and chronic alcohol exposure are risk factors for squamous cell esophageal carcinoma; long-standing reflux with Barrett esophagus is the greatest risk factor for distal esophageal adenocarcinoma.

Angiodysplasia This arteriovenous malformation is the most common vascular lesion of the GI tract and accounts for 2% to 4% of upper GI bleeding.[1] These degenerative vascular lesions of previously healthy blood vessels become abnormally dilated thin-walled mucosal and submucosal vessels. More than 90% of these lesions are at or distal to the jejunum, making angiodysplasia a more common cause of lower GI bleeding. Prevalence is increased in patients with end-stage renal disease, von Willebrand disease, and aortic stenosis. Low-grade bleeding with painless melena is the usual clinical presentation, but up to 15% of patients may have massive bleeding.[5] Bleeding stops spontaneously in more than 90% of cases but may be recurrent.

Other less-common differentials are shown in **Table 2**. Further considerations based on clinical scenario may include foreign body ingestion, gastric polyps, submucosal lesions or masses, Kaposi sarcoma, and connective tissue disease.

TABLE 2. Less common causes of upper GI bleeding[3,4]

- **Aortoenteric fistula**—an erosion of an aortic graft into the bowel lumen, usually the duodenum. Suspect this in patients with known aortic aneurysm, previous aortic aneurysm grafts (necrosis and graft rejection are implicated), or severe atherosclerosis.
- **Dieulafoy lesion**—a congenital vascular malformation of submucosal arteries in the proximal stomach, most commonly along lesser curvature of the stomach. Ulceration of these arteries accounts for 2% to 5% of cases of severe upper GI bleeding. Risk factors include male sex, cardiovascular disease, chronic kidney disease, diabetes, chronic gastritis, and alcohol consumption.
- **Iatrogenic, postprocedure, and anastomotic ulcers**—occur after chemotherapy or radiation, or after a procedure due to NG tube erosion, endoscopic biopsy, polypectomy, or sphincterotomy. Anastomic ulcers occur after peptic ulcer or bariatric surgery.
- **Hemobilia**—or bleeding from the gallbladder or biliary tree from a fistula between splanchnic vessels and the biliary system. Consider upper GI bleeding in patients with right upper quadrant pain and/or jaundice, especially if they have a history of liver injury or instrumentation (such as from cholecystectomy). In pseudohematobilia, bleeding originates in the pancreas, pancreatic duct, or splenic artery that drain into the duodenum. Hemobilia is associated with pancreatitis, pseudocysts, cancer, and splenic artery aneurysms.
- **Cameron lesion**—is an ulcer in the hiatal sac of patients with hiatal hernia. Uncommon and usually asymptomatic, Cameron lesion rarely may cause acute or chronic upper GI bleeding.

LABORATORY EVALUATION

Obtain a complete blood cell (CBC) count, chemistry panel including renal and liver function tests, and blood type and crossmatch for pending transfusion. A decline in hematocrit after 24 hours reflects dilution from intracellular fluid autoinfusion; this decline may occur earlier in patients receiving IV isotonic fluid resuscitation. Serial hematocrit levels are helpful in assessing the severity of upper GI bleeding but should be integrated with hemodynamic assessment because fluid volume status can alter hematocrit levels. If the patient is hypotensive and volume status needs to be ascertained, noninvasive methods such as urine output, cognitive status, and echocardiography can provide valuable data.

Expect normocytic erythrocytes in acute upper GI bleeding; microcytic erythrocytes are suggestive of iron-deficiency anemia with chronic blood loss. Leukocytosis may occur as a normal stress response to upper GI bleeding, but carefully exclude underlying infection when appropriate. Hypovolemia may result in prerenal azotemia reflective of a blood urea nitrogen (BUN):creatinine ratio greater than 20:1. A BUN:creatinine ratio greater than 36:1 suggests upper GI bleeding, because the small intestine absorbs blood, leading to higher BUN levels.[6] Additional laboratory assessment should be focused on the patient's history and clinical context (**Table 3**).

Older adults and those with significant comorbid illnesses are at risk for myocardial ischemia and infarction (MI) following even transient hypotension related to upper GI bleeding. Small-scale studies suggest a 12.3% prevalence of MI following severe upper GI bleeding in these populations; only 50% of patients experienced chest pain.[3] In

at-risk patients, exclude MI with serial ECGs and cardiac enzyme levels.

As clinical assessment and laboratory results reveal, immediately begin resuscitating the patient. A hematocrit level of 30% or greater is the goal in high-risk patients and 20% or greater must be maintained in young, healthy patients.[7] Patients with known coagulopathy should be corrected through the infusion of fresh frozen plasma or vitamin K. Infuse crystalloid fluids through two large-bore IVs to maintain BP. Administer supplemental oxygen and consider definitive airway management with intubation.

INVESTIGATIONAL PROCEDURES

Nasogastric (NG) lavage and aspiration NG intubation may help confirm, but cannot discount, suspected upper GI bleeding, as blood may have evacuated the stomach or pylorospasm may prevent access to duodenal blood.[4,8] Up to 18% of patients with upper GI bleeding have nonbloody aspirate.[9] NG intubation is uncomfortable for patients and may cause epistaxis, gastric erosion, pylorospasm, or inflammation. In determining ongoing bleeding, NG intubation benefits a small population, and is best used in hemodynamically stable patients who do not have evidence of hematemesis.[4]

Endoscopy Esophagogastroduodenoscopy (EGD) with intervention is the method of choice for controlling active upper GI bleeding and when early intervention is performed, it reduces rebleeding, mortality, need for surgery, transfusion, and the need for repeat EGD.[4] Patients with no active bleeding, stable vital signs, and low risk for rebleeding can be supportively treated and forego endoscopy. EGD should be performed within 24 hours of admission in patients with suspected upper GI bleeding, or immediately after resuscitation when active upper GI bleeding is suspected. Before EGD, evacuate intragastric blood and contents with NG lavage or IV erythromycin.[8] EGD localizes bleeding in 75% to 95% of cases and offers a variety of treatment modalities, including clips, argon plasma coagulation, injection of epinephrine or sclerosants, electrocoagulation, band ligation, and laser therapy.[4] Obtain biopsies for gastric ulcers, *H. pylori* infection, and malignancy. Optimal efficacy is achieved when a combination of endoscopic techniques are implemented rather than a single therapy. The international consensus guidelines on upper GI bleeding recommend use of endoscopic clips or thermal therapy for high-risk lesions, such as actively bleeding ulcers.[4]

> The use of PPIs in high-risk patients decreases the risk of ulcer rebleeding.

Assess the quality of ulcer from its appearance. Gastric ulcers appear as solitary, well-demarcated, discrete mucosal lesions with a punched-out, smooth ulcer base, often filled with white, fibrinoid exudate. Benign ulcers have smooth, regular, rounded edges with a flat, smooth base; malignant ulcers usually have irregular heaped-up or overhanging margins. Duodenal ulcers are well-demarcated breaks in the mucosa that may extend to the muscularis propia. Angiodysplasia appears as small (5 to 10 mm) flat, cherry-red lesions with a fern-like pattern of arborization, ectatic blood vessels radiating from a central vessel. Angiodysplasia can only be confidently considered the source of upper GI bleeding if active bleeding is seen.

EGD is contraindicated in patients who are hemodynamically unstable or at risk for perforation of the upper GI tract. Although complications are rare, risk of aspiration, adverse reaction to sedation, bleeding at the site of extracted biopsies, and perforation may occur. If endoscopic intervention fails in Forrest Class Ia and Ib (**Table 4**) with severe bleeding, selective arterial embolization by interventional radiology is indicated; if interventional radiology is not available, use surgical hemostasis.[8] Angiography localizes upper GI bleeding lesions in 86% of patients and allows infusion of an intra-arterial vasoconstrictor into mesenteric vessels.[4] The success rate is 90%; however, rebleeding rates of up to 50% have been reported once the infusion is stopped.[4] Transcatheter embolization selectively reduces blood supply to the source of bleeding and has clinical success rates of 90%.[4]

Prognostic scales The Blatchford, Rockall, and Forrest classification validated risk scales make use of clinical, laboratory, and endoscopic criteria to predict patient risk for rebleeding, morbidity, and mortality.[4,8] The Blatchford

TABLE 3. Laboratory assessment for differentials of upper GI bleeding

Cholestatic liver disease
- increased alkaline phosphatase
- increased gamma-glutamyl transpeptidase
- increased conjugated bilirubin
- increased total serum cholesterol
- increased aminotransferases

Zollinger-Ellison syndrome
- increased fasting serum gastrin level
- increased basal acid output

Peptic ulcer perforation
- increased serum amylase
- leukocytosis

Advanced liver disease
- macrocytic anemia
- increased bilirubin
- increased alkaline phosphatase
- decreased albumin
- prolonged prothrombin time
- hypoglycemia
- increased serum triglycerides and cholesterol
- normal or increased aminotransferases

scale uses assessments of systolic BP, BUN, hemoglobin, pulse, presentation of melena or syncope, and presence of hepatic disease or cardiac failure. The score is computed at initial assessment, ranges from 0 to 23, and is most useful in identifying low-risk patients stable for early discharge.[4]

The Rockall scale is calculated following endoscopy (**Table 4**). Scores of 2 or less indicate low risk of rebleeding and death; 3 to 7 indicate moderate risk; and 8 and above indicate high risk.[4] The Forrest classification (**Table 4**) uses endoscopic results to stratify patients into high- and low-risk categories for mortality and rebleeding and also guides endoscopic therapy.

TREATMENT AND FOLLOW-UP

Overall, 80% of upper GI bleeding will resolve spontaneously without treatment and 20% will recur.[4] Stable patients with low risk of rebleeding can be managed with PPI therapy as outpatients. In high-risk patients, administer IV high-dose PPI therapy during the 24-hour period before EGD to maintain neutral gastric pH. Studies reveal that a PPI bolus followed by a continuous infusion for 72 hours is more effective than bolus alone in decreasing the incidence of rebleeding and need for surgery.[4] Overall, the use of PPIs in high-risk patients decreases the risk of ulcer rebleeding, need for urgent surgery, and mortality as compared to histamine$_2$-blockers or placebo.[4]

After EGD, patients determined to be low risk can be discharged on oral PPIs. High-risk patients should be hospitalized for at least 72 hours and then discharged with an oral PPI.[4,10] Repeat EGD is no longer recommended.[4,8]

Patients with NSAID-induced upper GI bleeding should discontinue their NSAID and be administered PPI therapy. In patients who require continued NSAIDs, combination of a COX-2 inhibitor with long-term PPI therapy is recommended after GI and cardiovascular risk factors are considered.[4,10] This combination has a lower risk of GI complications compared with a nonselective NSAID or COX-2 inhibitor alone.[4] Adverse reactions to long-term PPI therapy include *Clostridium difficile* infection, pneumonia, osteoporosis-related fractures, and cardiac dysrhythmias.

Patients with bleeding peptic ulcers should be tested for *H. pylori* and receive eradication therapy and follow-up to confirm eradication. Triple therapy with amoxicillin, clarithromycin, and a PPI for 10 to 14 days is the first-line treatment, and reduces the risk of recurrent peptic ulcers and rebleeding.[4] Additional gastroprotective agents such as misoprostol and carafate can be prescribed to further prevent and treat NSAID-induced ulcers.

Treat the underlying disease process and follow surgical or medical management guidelines for less-common causes of upper GI bleeding. Finally, counsel patients on modifiable risk factors for upper GI bleeding, including smoking, alcohol or cocaine use, and provide education on avoiding aggravating factors such as coffee and other acidic foods.

TABLE 4. Scoring systems for upper GI bleeding[4,11]

Rockall score

- Age: <60 years (0 points), 60-79 years (1 point), >80 years (2 points)
- Shock: Heart rate >100 beats/minute (1 point); systolic BP <100 mm Hg (2 points)
- Coexisting illness: Ischemic heart disease, heart failure, other major illness (2 points); renal failure, hepatic failure, metastatic cancer (3 points)
- Endoscopic diagnosis: No observed lesion, Mallory-Weiss tear (0 points); peptic ulcer, erosive disease, esophagitis (1 point); cancer of the upper GI tract (2 points)
- Endoscopic stigmata of recent hemorrhage: Clean base ulcer, flat pigmented spot (0 points); blood in upper GI tract, active bleeding, visible vessel, clot (2 points)

Scoring: ≤2, low risk of rebleeding and death; 3 to 7, moderate risk; ≥8, high risk.

Forrest classification

- Class I: acute hemorrhage
  - Class Ia: spurting hemorrhage
  - Class Ib: oozing hemorrhage
- Class II: recent hemorrhage
  - Class IIa: nonbleeding visible vessel
  - Class IIb: adherent clot (associated with a 22% risk of rebleeding)
  - Class IIc: flat pigmented spot on ulcer base (associated with a 5% to 10% risk of rebleeding)
- Class III: no active bleeding. Lesions without signs of recent hemorrhage or with fibrin-covered clean ulcer base.

Treatment for Forrest classifications

- Lesions with low risk of rebleeding (Class IIc and III) can be managed on an outpatient basis with PPIs, have an excellent prognosis, and do not warrant EGD.
- Lesions with a high risk of rebleeding (Class I, IIa, and IIb) call for endoscopic hemostasis and high-dose PPI therapy for 72 hours.
- Patients with nonbleeding visible vessels or active bleeding have a 43% to 55% chance of rebleeding and should be admitted to the ICU.

For patients on single or dual antiplatelet therapy, maintain low-dose aspirin therapy and discontinue clopidogrel until consultation with specialists.[8] Discontinuation of aspirin is associated with a threefold increased risk of a major cardiovascular event and significantly higher mortality at 8 weeks; immediately restarting aspirin in combination with a PPI results in a twofold increase in the risk of rebleeding.[4] International consensus guidelines recommend reinitiating aspirin after 7 to 10 days, when the patient's risk of thromboembolic events outweighs the risk of ulcer bleeding.[4] PPIs decrease the antiplatelet effect of clopidogrel; when restarting patients on this medication, concurrent use of pantoprazole, a PPI with the least potential for drug interaction, may be an option.[4]

CONCLUSION

Considerable advances have been made in endoscopic, surgical, and pharmacologic therapy for upper GI bleeding. This common condition presents in a wide variety of settings, and physician assistants play a key role in prevention, diagnosis, and treatment. Familiarity with the practical, current, and evidence-based approach to diagnosis and management of upper GI bleeding is imperative. **JAAPA**

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