

Chapter 156: Upper Gastrointestinal Bleeding

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INTRODUCTION

Key Clinical Questions

- 1 What is the timing and treatment of peptic ulcer disease?
- 2 What are the factors in diagnosis and treatment of aortoenteric fistula?
- 3 What treatments are available for each etiology of upper GI bleeding?
- 4 What is the appropriate management and follow-up of variceal bleeding?
- 5 How do you estimate the severity of bleeding so that you can triage appropriate patients to the ICU, medical floor, or observation unit?
- 6 Which patients are more likely to rebleed and hence require continued observation in the hospital after their bleeding has apparently stopped, and for how long?

Upper gastrointestinal (GI) bleeding is responsible for over 300,000 hospitalizations per year in the United States. An additional 100,000 to 150,000 patients develop upper GI bleeding during hospitalizations. The annual cost of treating nonvariceal acute upper GI bleeding in the United States exceeds \$7 billion.

Upper GI bleeding is defined as a bleeding source in the GI tract proximal to the ligament of Treitz. The presentation varies depending on the nature and severity of bleeding and includes hematemesis, melena, hematochezia (in rapid upper GI bleeding), and anemia with heme-positive stools. Bleeding can be associated with changes in vital signs, including tachycardia and hypotension including orthostatic hypotension. Given the range of presentations, pinpointing the nature and severity of GI bleeding may be a challenging task.

The natural history of nonvariceal upper GI bleeding is that 80% of patients will stop bleeding spontaneously and no further urgent intervention will be needed. In contrast, only 50% of patients with a variceal hemorrhage stop bleeding spontaneously. Following cessation of active variceal bleeding, there is a high risk of recurrent bleeding within 6 weeks.

The mortality rate for nonvariceal upper GI bleeding is 2% to 14%. This mortality rate has improved because of the development of new medications, endoscopy (both diagnostic and therapeutic), intensive care units (ICUs), and advances in surgical management. The mortality remains high since patients with GI bleeding now are older, have more comorbidities, and are taking more medications, including nonsteroidal

anti-inflammatory drugs (NSAIDs), anticoagulants, and antiplatelet agents. For variceal bleeding, mortality is between 15% and 50% for each bleeding episode, and 70% to 80% in those with continuous bleeding. Variceal hemorrhage is responsible for one-third of all deaths due to cirrhosis.

DIFFERENTIAL DIAGNOSIS

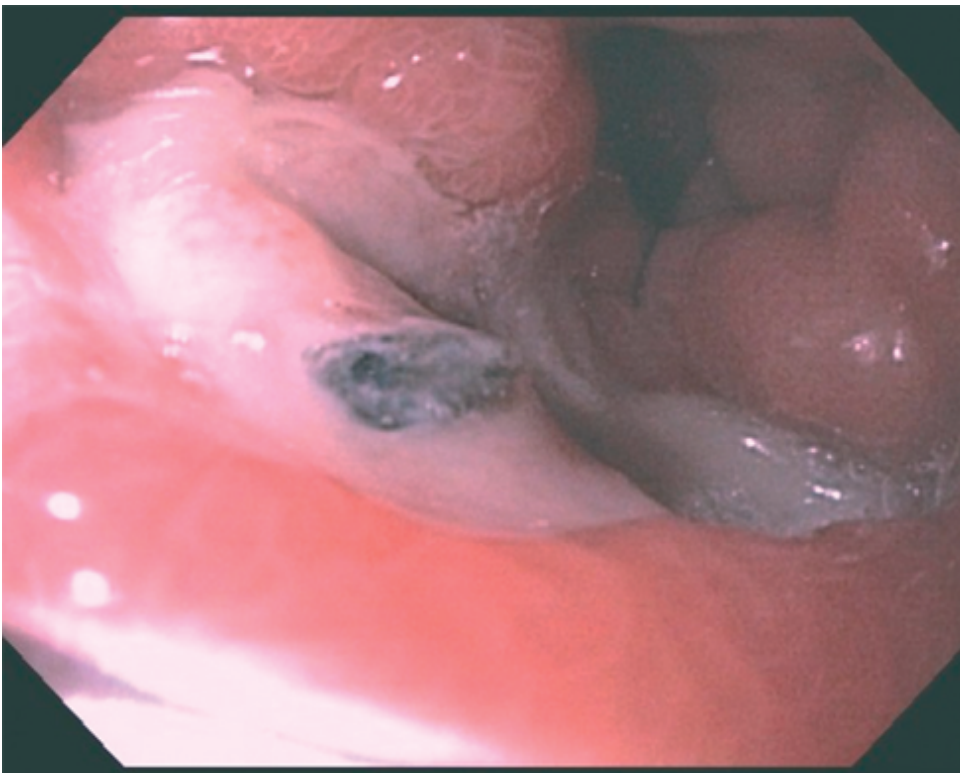
NONVARICEAL BLEEDING

Peptic ulcer disease

Diagnosis: Peptic ulcer disease is a common condition stemming from an imbalance of protective and disruptive factors of the GI mucosa. Ulcers are most commonly found in the stomach and proximal duodenum (**Figures 156-1** and **156-2**). Peptic ulcer disease is the most common cause of upper GI bleeding and accounts for up to 50% of total cases and over 100,000 hospital admissions per year in the United States. The annual incidence of peptic ulcer disease in patients infected with *Helicobacter pylori* is about 1% per year, which is six to ten times higher than patients who are uninfected. Diagnosis is made by endoscopy performed when symptoms prompt endoscopic investigation. An initial step in management is to identify *H. pylori* infection and users of NSAIDs. These two risk factors account for the vast majority of ulcers in the upper GI tract.

Figure 156-1

Duodenal ulcer.



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Figure 156-2

Gastric ulcer.



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Disruptive factors that can damage the mucosa of the GI tract include acid, pepsin, bile salts, ischemia, and *H. pylori*. Exogenous causes are predominately medications (NSAIDs, aspirin, and SSRIs). Stress-induced ulcers are a common cause of bleeding in patients hospitalized for other severe illnesses. Risk factors for stress-induced ulcers include respiratory failure (especially intubated patients), coagulopathy (international normalized ratio [INR] > 1.5 or platelets <50,000/ μ L), trauma, sepsis, renal failure, burns, ICU admission, and surgery.

Pathophysiology: The defensive forces of the esophagus include esophageal motility with clearance of refluxed materials, the lower esophageal sphincter to prevent reflux, and salivary secretions that contain bicarbonate. Gastric protective factors include the mucous layer as well as tissue mediators. *H. pylori* is a flagellated bacterium with several unique attributes that aid in its ability to break down gastric mucosa. These factors include its production of urease, which neutralizes the acidic gastric environment. NSAIDs inhibit cyclooxygenase, thus decreasing mucosal protective prostaglandin levels in the stomach. They also increase gastric vascular endothelial adhesion molecules, thus leading to neutrophil adherence and mucosal injury.

Clinical findings: Peptic ulcer disease presents in a variety of ways. Patients can experience epigastric pain, melena, hematochezia, dyspepsia, bloating, or can be asymptomatic with or without anemia. On examination, pain may be epigastric and reproducible, but many patients have right-sided pain or no pain at all.

Prognosis: Response to treatment and outcomes vary for peptic ulcer disease. Treatment is with proton-pump inhibitor (PPI) therapy until asymptomatic and/or mucosal healing is demonstrated via an esophagogastroduodenoscopy (EGD) and/or antibiotics where indicated and removal of offending agents (eg, NSAIDs) leads to mucosal healing ([Table 156-1](#)). Chronic, nonhealing gastric ulcers should prompt

consideration of underlying illness or an alternative diagnosis, such as malignancy, and thus should be biopsied.

TABLE 156-1

Recommended Treatment Regimens for *H. Pylori* Infection

- **Three-drug regimens**

- Proton-pump inhibitor (PPI) orally twice daily + clarithromycin 500 mg orally twice daily + amoxicillin 1 g orally twice daily. Eradication 85%-90%. Duration 10-14 days.
- Proton-pump inhibitor orally twice daily + clarithromycin 500 mg orally twice daily + metronidazole 500 mg orally twice daily. Eradication 75%-85%. Duration 10-14 days. This regimen is generally used for PCN allergic patients.

- **Four-drug regimen**

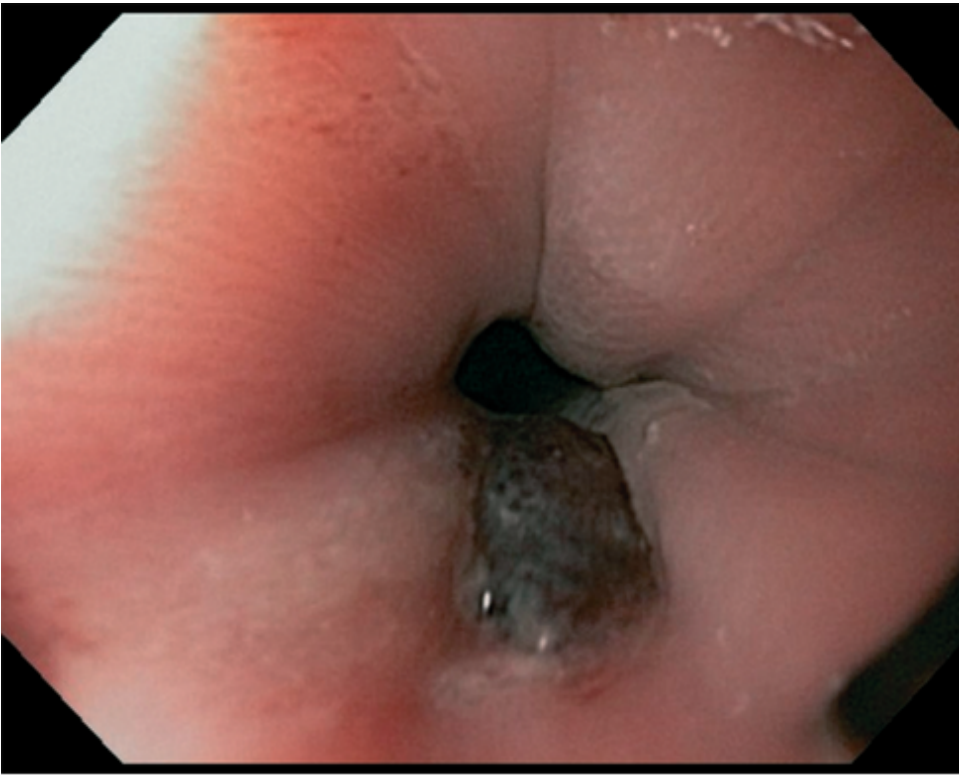
- Proton-pump inhibitor orally twice daily + bismuth subsalicylate 525 mg 4 times daily + Tetracycline 500 mg 4 times daily + metronidazole 500 mg orally 3-4 times daily. Eradication 75%-90%. Duration 2 weeks.

Mallory-Weiss tears

Diagnosis: Mallory-Weiss tears occur at the gastroesophageal junction, in the distal esophagus, or proximal stomach (**Figure 156-3**). Typically, these tears form after repeated and severe vomiting or retching. The tears may extend into the underlying blood vessels. Acute upper GI bleeding is the major clinical finding, which may be associated with epigastric pain, which may radiate to the back. Endoscopy is used to document the presence of a gastroesophageal tear and allows the possibility of therapeutic intervention.

Figure 156-3

Mallory-Weiss tear.



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Bleeding occurs when the tear involves the underlying esophageal venous or arterial plexus. Patients often have a history of nonbloody vomiting or retching before the onset of hematemesis. The tears are located at the esophagogastric junction, and can be within a hiatal hernia. Tears can extend downward into the cardia or, less commonly, upward into the esophagus.

Pathophysiology: Mallory-Weiss syndrome is characterized by longitudinal mucosal lacerations in the distal esophagus and proximal stomach, which are usually associated with forceful retching.

Prognosis: Although 40% to 70% of patients with bleeding Mallory-Weiss tears require blood transfusions, most tears heal spontaneously within 24 to 48 hours of presentation. Risk factors for rebleeding include portal hypertension and coagulopathy. Patients with active or ongoing bleeding can be treated with endoscopic hemostatic methods. For the rare patient with ongoing bleeding unresponsive to endoscopic control, intravenous (IV) infusions of octreotide, arterial embolization via angiography, and esophageal balloon tamponade have been used. In refractory bleeding, surgery with over-sewing of the bleeding vessel may be required.

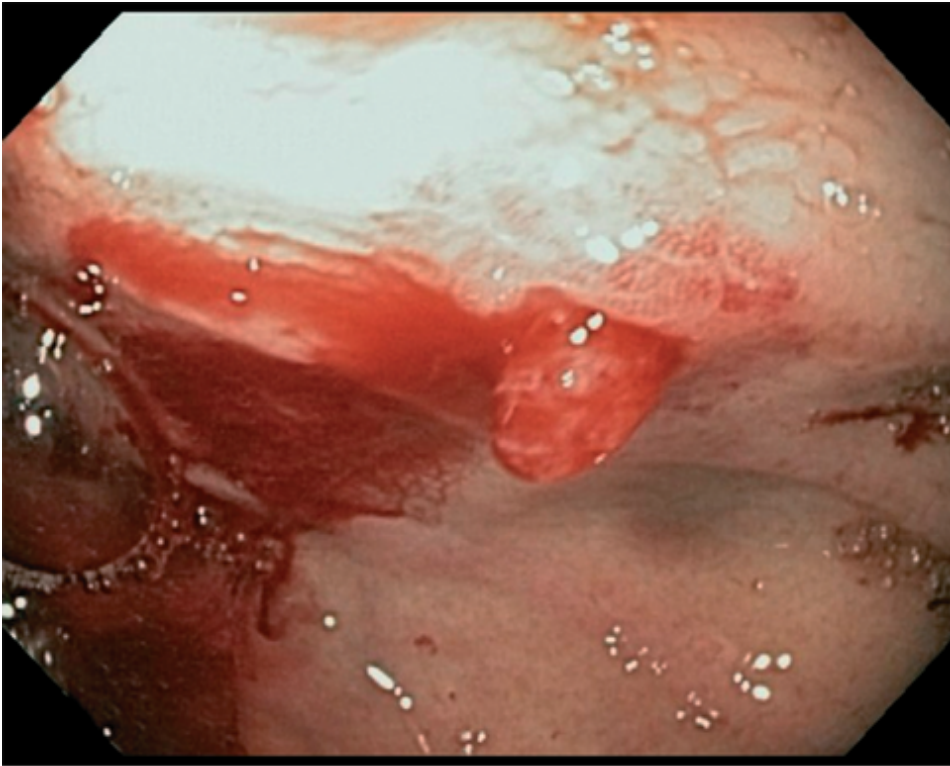
Dieulafoy lesion

Essentials of diagnosis: A Dieulafoy lesion is a dilated, aberrant submucosal vessel that erodes through normal epithelium and is not associated with an ulcer ([Figure 156-4](#)). Dieulafoy lesions are typically located just below the gastroesophageal junction on the lesser curvature of the stomach but can be found in areas throughout the GI tract, including the esophagus, duodenum, and colon. When a Dieulafoy lesion bleeds, massive arterial bleeding can occur. In the absence of active bleeding, the lesion is difficult to see as it may appear as a small and subtle raised area, such as a nipple or visible vessel without an associated ulcer. In patients with massive bleeding and no obvious cause, the lesser curvature of the stomach within 6 cms of

the gastroesophageal junction should be carefully inspected endoscopically for evidence of a Dieulafoy lesion.

Figure 156-4

Dieulafoy lesion.



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The typical patient with a Dieulafoy lesion is a man, with multiple comorbidities, already hospitalized for other problems. Diagnosis is confirmed with finding a visible vessel or active arterial pumping, without an associated ulcer or mass. The diagnosis can be difficult, as bleeding may be massive such that the area may be covered with blood and the source may not be visualized. It is especially challenging to diagnose Dieulafoy lesions that are not bleeding as the absence of an associated ulcer makes detection of the vessel difficult.

Pathophysiology: The cause of a Dieulafoy lesion is unknown. The arteries are 1 to 3 mm in size, which is approximately 10 times the caliber of mucosal capillaries. The typical location is in the upper stomach along the high lesser curvature, within 6 cm of the gastroesophageal junction.

Clinical findings: A Dieulafoy lesion often presents with a massive acute upper GI bleed, although the bleeding may be self-limited in nature. The diagnosis is confirmed with finding a visible vessel or with active arterial pumping without an associated ulcer or mass.

Prognosis: Endoscopic treatment is the mainstay for bleeding control of a Dieulafoy lesion via cauterity, clips or the combination of [epinephrine](#) injection and one of these two treatment modalities. The risk of recurrent bleeding following endoscopic treatment is relatively high due to the size of the underlying artery. In patients with recurrent bleeding, repeat endoscopic therapy is often the next step; however, IR angiography or surgical therapy may be necessary and consultation by a surgeon and interventional radiologist early on is recommended.

VASCULAR MALFORMATIONS

Angiodysplasia

Diagnosis: Angiodysplasias may occur throughout the GI tract, may be multiple in one gastrointestinal region, or coexist in several different gastrointestinal locations. Although angiodysplasias are a relatively common source of small bowel bleeding, they are a rare source of massive bleeding from the stomach. Bleeding from an upper GI vascular malformation is typically intermittent and low grade in nature, although it can be acute and overt.

Pathophysiology: The cause of most vascular malformations is unknown. Vascular malformations can be present at birth, although they do not appear to be hereditary. They occur equally in both sexes and in different races. Vascular malformations may be caused by a rupture or clotting of a blood vessel during fetal development, although not associated with other problems at birth.

Clinical findings: Bleeding from upper GI vascular malformations is typically intermittent and low grade in nature, although it can be acute and overt. The endoscopic appearance may be variable, but includes a superficial collection or tuft of blood vessels.

Prognosis: Endoscopic therapy with thermal coagulation is the treatment of choice with cautery applied first to the periphery and then to the center of the lesion. Argon plasma coagulation is most commonly used to treat vascular malformations and is an effective therapy. Certain medications have been utilized in an attempt to treat vascular malformations in the upper gastrointestinal tract. Combination estrogen/progesterone therapy has been widely used, as has octreotide. Angiography or surgery may be offered for failures of endoscopic and medical therapy, but is rarely necessary.

Gastric antral vascular ectasia

Diagnosis: Gastric antral vascular ectasia (GAVE), also known as “watermelon stomach,” is an idiopathic condition often associated with portal hypertension. Gastric antral vascular ectasia is a relatively unusual cause of upper GI bleeding. The bleeding that occurs is typically chronic and low grade, often associated with iron deficiency anemia and occult bleeding. It is unusual to have acute or massive upper GI bleeding from GAVE.

Pathophysiology: The etiology of watermelon stomach is not known; however, it has been theorized to be due to gastroduodenal prolapse and occurs in patients with cirrhosis, portal hypertension, and systemic sclerosis. Patients with concomitant portal hypertension have bleeding that is more difficult to control, requiring treatment of the underlying portal hypertension.

Clinical findings: During endoscopy, vascular malformations occur in rows of reddish stripes that radiate outward from the pylorus, in a pattern that resembles the stripes on a watermelon; hence, this entity has been referred to as “watermelon stomach.” These stripes represent rows of vascular malformations and the entity is easy to recognize in the classic form. However, GAVE may have a less organized appearance especially in patients with portal hypertension. Although the diagnosis is by endoscopy, it can be confirmed by biopsies obtained during endoscopy. GAVE histology shows areas of vascular ectasia associated with spindle cell proliferation and fibrohyalinosis.

Prognosis: Most patients will respond to endoscopic therapy most commonly with argon plasma coagulation. Several such endoscopic treatment sessions may be required. Radiofrequency ablation may be used to treat refractory cases. However, surgery with an antrectomy may be required for patients who have persistent bleeding despite endoscopic management.

Aortoenteric fistula

Diagnosis: Aortoenteric fistulas occur when there is a direct communication between the aorta and the gastrointestinal tract and can lead to massive bleeding. Patients may have a “herald” bleed with initial hematemesis or hematochezia, which may spontaneously remit. This might then be followed by torrential bleeding, including exsanguination, making it important to quickly diagnose this condition. Intermittent bleeding can occur if a blood clot overlies a fistulous connection. There may be abdominal or back pain (which occurs in about half of patients), or signs of fever and infection. On physical examination, a bruit may be heard and a pulsatile mass may be palpable at any point along the midabdomen representing the course of the abdominal aorta.

A high index of suspicion is needed to promptly diagnose a patient with an aortoenteric fistula for successful management. In a patient who has a known risk factor such as previous aortic grafting, this diagnosis should be considered early in the course. In fact, it should always be suspected in patients with massive bleeding from the second to third portion of the duodenum. The diagnosis can be confirmed by computed tomography with angiography or with traditional angiography. Diagnostic tests need to be done in an expeditious manner along with prompt vascular surgical evaluation as the management of an aortoenteric fistula is surgical. The mortality rate of an unrecognized fistula is 100%, whereas the mortality for a recognized fistula is 30%.

Hemobilia

Diagnosis: Hemobilia is a rare cause of acute upper GI bleeding that occurs from the hepatobiliary tract in less than 1% of patients who undergo a procedure to the biliary tree (endoscopic retrograde cholangiopancreatography [ERCP], surgery, or CT-guided procedures) and almost never spontaneously with the rare exception of invasive tumors. **The classic triad of hemobilia is biliary colic, obstructive jaundice, and gastrointestinal bleeding.** The diagnosis of hemobilia can be difficult by upper endoscopy, which may not clearly visualize the ampulla. A side-viewing duodenoscope is often utilized to visualize and access the ampulla directly or to perform ERCP.

Hemobilia should be considered in patients who have had a recent history of hepatic or biliary tract injury. This includes trauma to the area, percutaneous (or transjugular) liver biopsies, percutaneous transhepatic cholangiograms, ablations of liver tumors or biopsies. Hemobilia can also occur secondary to gallstones, cholecystitis, hepatobiliary tumors, hepatic abscesses, and aneurysms. Obstructive jaundice may be associated with biliary sepsis. Patients with these findings should be considered as potentially having hemobilia. Treatment is directed at the primary cause of bleeding, although ERCP may also be needed to remove clot from the bile duct causing obstruction. While treatment may occasionally be performed endoscopically, it usually needs to be done angiographically (such as by arterial embolization) or surgically.

Hemosuccus pancreaticus

Diagnosis: Hemosuccus pancreaticus is another rare cause of upper GI bleeding that occurs due to bleeding from the pancreatic duct. This can be secondary to chronic pancreatitis, pancreatic pseudocysts, and pancreatic tumors. This can also occur after a therapeutic endoscopy of the pancreas, including pancreatic sphincterotomy or stone removal. Hemosuccus pancreaticus can be difficult to detect by routine endoscopy; thus, a side-viewing duodenoscope and ERCP may be needed to reveal the source of the bleeding. The diagnosis may be confirmed by abdominal CT scan, angiography, or intra-abdominal exploration.

Bleeding occurs when there is erosion into a vessel, forming a direct communication with the pancreatic duct. Angiography is important for diagnosis and treatment, often with coil embolization to control the acute bleeding. For persistent or massive bleeding, surgery with resection of the bleeding area or ligation of a bleeding vessel may be required.

Cameron lesion

Diagnosis: Cameron lesions are erosions or ulcers that occur in the distal aspect of a hiatal hernia. Although this may be an incidental finding on upper endoscopy, these lesions can be responsible for iron deficiency anemia or acute or chronic upper GI bleeding.

The mechanism of formation of a Cameron lesion is not well understood. Potential etiologies include gastroesophageal reflux and mechanical trauma of the area. Management of a Cameron lesion depends on the clinical situation. Patients with acute bleeding can be treated by endoscopic methods. Patients who do not have acute bleeding, but have chronic blood loss may be treated with medical therapy, such as a proton-pump inhibitor and iron repletion. Surgical repair of the hiatal hernia is curative, but rarely necessary.

Upper gastrointestinal tumors

Diagnosis: Neoplasms of the upper GI tract are a rare cause of upper GI bleeding. These tumors may be esophageal squamous carcinomas, malignant lymphomas or adenocarcinomas (primary or metastatic often from lung or breast), or benign lesions such as leiomyomas or gastrointestinal stromal tumors. The symptoms that suggest bleeding may be from a gastrointestinal tumor include symptoms that may be attributable to the primary tumor such as dysphagia from esophageal cancer, but symptoms may be nonspecific including cachexia, weight loss, and early satiety. If the diagnosis of a tumor is not previously known, it may be detected at the time of endoscopy and confirmed by brushings or biopsies. Endoscopic ultrasound may be needed to evaluate submucosal masses.

Bleeding from an upper gastrointestinal tumor may be from mucosal erosions or ulcerations, or from invasion of the tumor into an underlying vessel. Typically, endoscopic treatments are a temporizing measure prior to more definitive measures such as surgery because rebleeding will frequently occur after endoscopy. Medical therapy is often ineffective in this setting, although palliative measures may be provided including chemotherapy and radiation of the primary tumor. Patients who have bleeding due to an upper GI malignancy have a very poor prognosis. The majority of patients (>60%) will die within 3 months. However, patients who have a benign upper gastrointestinal tumor that bleeds will be cured by surgical resection.

VARICEAL BLEEDING

Gastroesophageal varices

Diagnosis: Varices are dilated venous collaterals that have a tendency to rupture and can bleed massively. Esophageal and gastric varices develop as a result of portal hypertension usually due to advanced liver disease and cirrhosis (**Figures 156-5** and **156-6**). Varices may be isolated to the stomach if they are due to splenic vein thrombosis, acute pancreatitis, or a pancreatic tumor. Esophageal and gastric varices may occur without any clinical symptoms.

Figure 156-5

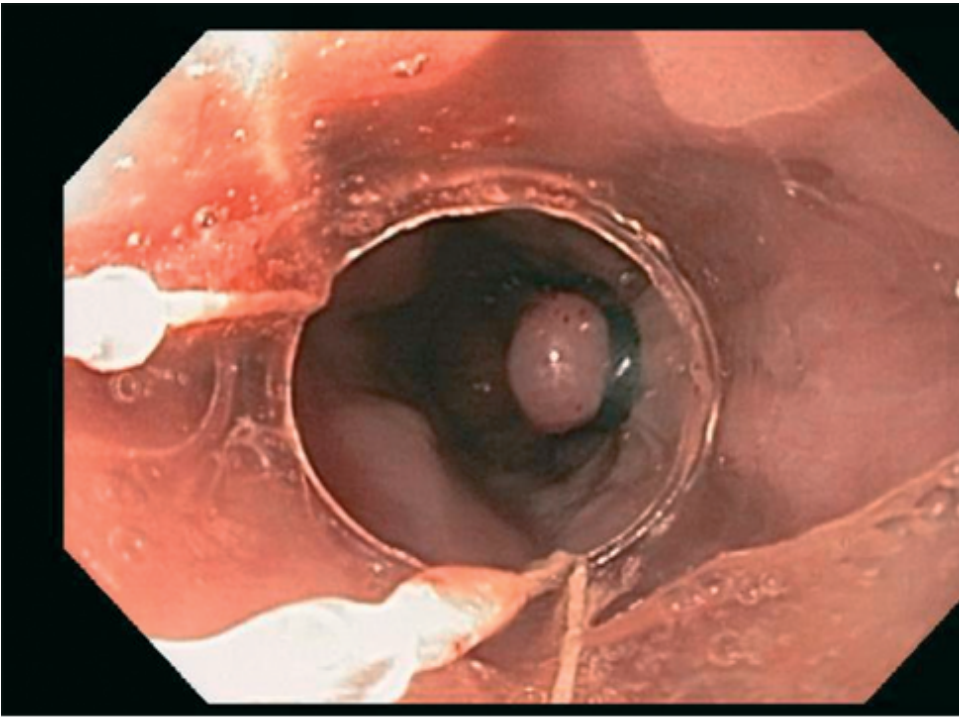
Esophageal varices.



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Figure 156-6

Banded esophageal varix.



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Acute variceal bleeding is different from nonvariceal bleeding in many respects. Upper endoscopy is the primary diagnostic modality and allows for direct visualization of columns of esophageal varices. Endoscopy should be performed quickly (within 24 hours) for potential intervention if varices are suspected as the source of upper GI bleeding. Abdominal CT and ultrasound may show the presence of collateral veins. Portal vein angiography may also demonstrate the presence of collaterals or recanalization of the umbilical vein.

Variceal hemorrhage is responsible for one-third of all deaths due to cirrhosis. Mortality is between 15% and 50% for each bleeding episode, and 70% and 80% in those with continuous bleeding. Following cessation of active variceal bleeding, there is a high risk of recurrent bleeding within 6 weeks (>50%). The time of greatest risk is within the first 48 to 72 hours (90% of rebleeds will occur in this time period), and over half of all subsequent rebleeding episodes occur within the first 10 days. Recurrent variceal bleeding may occur in up to 70% of patients within 6 weeks of the index bleed if preventive measures are not performed.

Survival during the 6 weeks following the index bleed is directly related to rebleeding. Risk factors include age greater than 60 years, large varices, severe initial bleed (hemoglobin <8 g/dL on admission) and renal failure. Patients with acute variceal bleeding are typically treated with multiple modalities simultaneously, including medical therapies and endoscopic management. Over-resuscitation should be avoided as overly vigorous volume repletion has been associated with precipitation of further bleeding. The target hemoglobin goal during resuscitation should be 7 g/dL. While early intervention in the form of transfusions (for low hemoglobin) and intravenous fluids may be warranted in the setting of hypotension, patients who are suspected to have variceal bleeding should undergo endoscopy within 12 hours of presentation and after resuscitation. In addition, radiologic and surgical treatments may be needed. Endoscopic variceal band ligation is a commonly applied therapy. This is a method of placing elastic bands over varices, similar to the technique of banding hemorrhoids. Varices are suctioned into a banding device and the bands are released around the base of the varices. It is typically performed in the distal 5 cm of the esophagus.

Pathophysiology: Normal pressure in the portal vein is 5 to 10 mm Hg. An elevated portal venous pressure of greater than 10 mm Hg (due to obstruction) increases capillary pressure. The connection between the portal and systemic circulation may enlarge to allow blood to bypass the obstruction and pass directly into the systemic circulation. One of the known sites of such confluence is the distal esophagus. Studies have demonstrated the role of endothelin-1 and nitric oxide in the pathogenesis of portal hypertension and esophageal varices. Endothelin-1 is a powerful vasoconstrictor synthesized by sinusoidal endothelial cells, that has been implicated in the increased hepatic vascular resistance of cirrhosis and in the development of liver fibrosis. Nitric oxide is a vasodilator substance that is synthesized by sinusoidal endothelial cells. In the cirrhotic liver, the production of nitric oxide is decreased, and endothelial nitric oxide synthase activity and nitrite production by sinusoidal endothelial cells are reduced.

Clinical findings: Symptoms of variceal bleeding are nonspecific and include hematemesis, melena, and hematochezia. Patients may feel light-headed and dizzy; and those with severe liver disease may have hepatic encephalopathy, which may be the sole presenting feature in a cirrhotic patient who is bleeding from the upper GI tract. Other associated signs and symptoms are the clinical manifestations of cirrhosis including the laboratory abnormalities associated with cirrhosis (see [Chapter 160 \[Cirrhosis and its Complications\]](#)). The objective of upper endoscopy is to find and define the source of bleeding and to treat it. Varices are evaluated for signs of either active bleeding or markers of recent bleeding. These markers include large tortuous varices with red wale marks (longitudinal red streaks on varices that resemble red, corduroy wales), cherry-red spots (discrete cherry-red spots that are flat on a varix) and hemocystic spots (raised discrete red spots that overlie varices that appear as “blood blisters”). Platelet or fibrin plugs are white nipple-like projections that project from a varix indicative of a site with recent bleeding and with a very high risk of bleeding.

Prognosis: Variceal bleeding is a significant cause of rebleeding and mortality. Variceal bleeding will stop in approximately 50% of patients, but those who have continued bleeding have a mortality that approaches 80%. Patients have a high risk of rebleeding (up to 70%) until the gastroesophageal varices are obliterated. If a patient survives the initial bleeding episode, repeated courses of band ligation are performed approximately every 2 weeks until the varices are obliterated. This is combined with medical therapy using nonselective β -blockade to reduce portal pressures. Current data support the use of nonselective β -blockers for medium and large varices prophylactically and for any grade of varix found to bleed. The prognosis for patients with bleeding gastroesophageal varices is poor even with control of bleeding varices as this is indicative of progressive liver disease. Patients die from hepatic decompensation, rebleeding, infections, renal failure, and other complications. Bacterial infections occur in up to 20% of cirrhotics with GI bleeding and subsequently develop in an additional 50% of patients during hospitalization for variceal bleeds. These include spontaneous bacterial peritonitis, pneumonia, sepsis, and skin infections. The use of prophylactic antibiotics in patients with acute variceal bleeding has been shown to decrease the rates of subsequent infection, spontaneous bacterial peritonitis, bacteremia, and death. Data for the use of fluoroquinolones as the prophylactic antibiotic of choice is supported by frequent clinical use, but a growing resistance of organisms to this class must be considered in choosing an antibiotic and alternatives include third-generation cephalosporins.

PRACTICE POINT

Management of acute variceal bleeding:

Volume repletion with a target hemoglobin goal during resuscitation of 7 g/dL

Upper endoscopy following resuscitation within 12 h of presentation

Prophylactic nonselective β -blockers for large varices and for any grade of varix identified as the source of bleeding

Prophylactic antibiotics to decrease the rates of subsequent infection, spontaneous bacterial peritonitis, bacteremia, and death

Portal hypertensive gastropathy

Diagnosis: In patients with cirrhosis and portal hypertension, gastric mucosal blood flow is increased, leading to congestion and hyperemia of the stomach. Portal hypertensive gastropathy occurs when edema and capillary venous dilatation in the stomach causes friability. This may subsequently result in bleeding with rupture of ectatic vessels. The endoscopic appearance is a mosaic-like pattern of pink mucosa, with a characteristic “snakeskin” appearance. Overall, this is a rare cause of acute upper GI bleeding, although it may be a source of chronic blood loss in patients with cirrhosis.

Portal hypertensive gastropathy may develop after treatment of esophageal varices. A proposed mechanism following treatment of varices is increased backpressure into the stomach vasculature, leading to the development of gastric congestion. Thus, the goal of treatment of portal hypertensive gastropathy is to decrease portal pressures. The affected area is diffuse and pharmacologic agents, such as octreotide, may be used acutely to decrease blood flow. Nonselective β -blockers (nadolol and propranolol) are often given, with the dose increased and adjusted if side effects occur. Endoscopic treatments are ineffective in this disorder. Patients with uncontrolled bleeding may need transjugular intrahepatic portal systemic shunt therapy or a surgical shunt in order to reduce portal pressure. In patients with decompensated liver disease, liver transplantation is indicated.

DIAGNOSIS

Upper GI bleeding may be life threatening. Factors that portend a favorable or a poor prognosis in patients with nonvariceal upper GI bleeding may be used to appropriately triage and manage patients. The role of nasogastric (NG) tube aspiration is controversial, and we do not routinely support its use. NG tube aspirates may be falsely negative; false-negatives most commonly occur in patients with bleeding from duodenal ulcers due to spasm of the pylorus, and may occur in other conditions, including gastric ulcers and rarely esophageal varices (if the tube is positioned in a nondependent area of the stomach).

HISTORY TO AID DIAGNOSIS

Similar to utilizing laboratory values to aid in diagnosis, a comprehensive history may help to predict a source of bleeding and prognosticate outcomes. An alcoholic with cirrhosis who has not recently undergone variceal screening endoscopy and presents with a large volume upper GI bleed should be managed as a variceal bleed until proven otherwise, including administration of octreotide and prophylactic antibiotics, and undergo urgent endoscopy. A patient with no history of liver disease or

physical stigmata of cirrhosis probably presents with a nonvariceal source of bleeding. A patient with an extensive NSAID use history most likely has peptic ulcer disease.

LABORATORY TESTING TO ESTABLISH GI BLEEDING SOURCE

Although laboratory values cannot predict a source of bleed, they help to define the clinical status of the bleeding patient. The BUN will be elevated out of proportion to the creatinine due to digestion of blood in most patients with upper GI bleeding. In variceal bleeding, abnormalities of the liver enzymes (ALT, AST) are seen in patients with active hepatocellular damage. Patients may have hyperbilirubinemia and poor synthetic function, with hypoalbuminemia and an elevated INR indicating impaired liver function. Patients with bone marrow suppression from alcohol may have pancytopenia or those with hypersplenism may have low platelet counts. In patients with liver failure, hypoglycemia may be detected. Low albumin states may predict worse outcomes from bleeding.

TRIAGE/HOSPITAL ADMISSION

RISK ASSESSMENT: CLINICAL PREDICTORS AND GUIDELINES

Clinical guidelines have been developed to help optimize the management of patients with nonvariceal upper GI bleeding. The aim of guidelines is to identify low-risk patients who can be discharged either directly from the emergency room or at an early stage of hospitalization, and to identify high-risk patients who will need more resources. Several such guidelines include both clinical data and information obtained at the time of endoscopy. Newer guidelines focus on clinical data to determine which patients bear increased mortality risk and therefore merit urgent endoscopy and/or ICU level care.

Three main prognostic scores for upper GI bleeding have been used clinically. The Rockall score ([Table 156-2](#)) is a postendoscopy scoring system used to predict rebleeding and mortality in patients with nonvariceal upper GI bleeding. In this scoring system, scores of zero to three are assigned to the factors of age, the presence of shock, comorbidity, diagnosis, and endoscopic stigmata. A low-risk patient has a score of two or less, and about 30% of patients will belong to this category. In this low-risk group, there was a 4.3% risk of rebleeding and 0.1% mortality. Patients with Rockall scores of three to five have intermediate rates of rebleeding and mortality (2.0%-7.9%), whereas patients with a score of six or greater have a high rebleeding and mortality rate (15.1%-39.1%). Limitations of this scoring system include that it was derived from a relatively small number of patients and the full score requires both clinical and endoscopic information to calculate.

TABLE 156-2

The Components and Score Assignments of the Rockall Risk Assessment Score for Upper GI Bleeding

Variable	Score			
	0	1	2	3
Age (y)	<60	60-79	>79	
Shock	<ul style="list-style-type: none"> • BP >100 mg Hg • Pulse <100 bpm 	<ul style="list-style-type: none"> • BP >100 mg Hg • Pulse >100 bpm 	<ul style="list-style-type: none"> • BP <100 mg Hg • Pulse >100 bpm 	
Comorbidity	None		Cardiac disease, any other major comorbidity not listed in next column	<ul style="list-style-type: none"> • Renal failure, liver failure • Disseminated malignancy
Endoscopic diagnosis	Mallory-Weiss tear, no lesion	All other diagnoses	Upper GI tract malignancy	
Major stigmata of recent hemorrhage	None, or dark spots only		Blood in upper GI tract, adherent clot or spurting vessels	

BP, blood pressure; GI, gastrointestinal. Max score = 11; low risk ≤ 2 ; high risk ≥ 5 (although this varies in the literature).

A scoring system that only uses clinical information (and not endoscopic data) obtained at the time of presentation prior to endoscopy, developed by Blatchford and colleagues ([Table 156-3](#)), helps to determine urgency of endoscopy and need for intervention. The clinical information incorporated into this scoring system includes hemoglobin, BUN, heart rate, systolic blood pressure, the presence of syncope, melena, liver disease, and heart failure. A new scoring system that also only uses information available at the time of initial presentation is the AIMS65 score. This score includes five factors: albumin <3.0 g/dL, INR >1.5, altered mental status, systolic blood pressure <90 mm Hg, and age >65 years. In this score, each factor is assigned a score of 1 and high-risk patients have an AIMS65 score >1.

TABLE 156-3

The Components and Interpretation of the Blatchford Scoring System

Variables	Points
Systolic blood pressure (mm Hg)	
100-109	1
90-99	2
<90	3
Blood urea nitrogen (mmol/L)	
6.5-7.9	2
8.0-9.9	3
10.0-24.9	4
>25	6
Hemoglobin: Men (g/dL)	
12.0-12.9	1
10.0-11.9	3
<10	6
Hemoglobin: Women (g/dL)	
10.0-11.9	1
<10	2
Other variables	
Pulse >100	1
Presentation with melena	1
Hepatic disease	2

Variables	Points
Cardiac failure	2
Total	—

A score of >1 is considered “high risk” while ≤ 1 is “low risk” in terms of predicting need for intervention.

MANAGEMENT

INITIAL HOSPITAL TREATMENT

While the early goals of treatment for GI bleeding include identification of the bleeding source and facilitation of bleeding cessation, the *primary task is stabilization of the patient*. Patients need to have at least two large bore peripheral intravenous access catheters (18 gauge or larger). Supplemental oxygen should be administered routinely. Crystalloid fluids should be initially administered to maintain an adequate blood pressure. Patients who are not able to adequately protect their airways (including patients with ongoing, severe hematemesis) should be considered for elective endotracheal intubation.

Patients who are high risk, such as the elderly or those with active coronary artery disease, should be transfused to maintain hemoglobin above 10 g/dL. Young and healthy patients should be transfused to maintain hemoglobin of 7 g/dL. Any coagulopathy should be corrected if possible with transfusion of fresh frozen plasma (initially 1 unit, followed by additional units based on subsequent INR) or administration of vitamin K (preferably 10 mg orally if at all possible). In patients with a low platelet count (<50,000/mL), it is recommended to transfuse platelets with a target platelet count goal >50,000/mL, initially with one or two units of platelets, followed by additional units based on subsequent platelet count.

Medications

Medications useful for management of acute upper GI bleeding include proton-pump inhibitors and octreotide. Other medications including H₂ blockers have not been demonstrated to favorably influence the natural history of acute GI bleeding.

Proton-pump inhibitor therapy

Proton-pump inhibitors help stop bleeding and prevent rebleeding in patients with significant nonvariceal GI bleeding. Endoscopic therapy is considered the standard of care for patients with active bleeding and nonbleeding visible vessels. Patients with peptic ulcer and successful endoscopic therapy have less rebleeding with the addition of intravenous PPI therapy ([Table 156-4](#)). In patients initially treated in the emergency room with a bolus infusion of omeprazole followed by a continuous infusion, active bleeding lesions found at endoscopy decreased and clean-based ulcers at endoscopy increased. A recent review and meta-analysis suggests that outcomes with an intermittent PPI dosing strategy is comparable to the previously recommended bolus and continuous infusion PPI dosing for patients with high-risk bleeding

ulcers that are treated endoscopically. Patients who remain stable for 72 hours may then transition to standard dose oral PPIs.

TABLE 156-4

Commonly Used Oral Proton Pump Inhibitors (PPIs) and Their Recommended Dose Regimen for Acute Upper Gastrointestinal (GI) Bleeding

PPI Name	Initial Bolus Dose	Continuous IV Infusion Dose	Duration
Lansoprazole (Prevacid)	60 mg	6 mg/h	Up to 72 h
Pantoprazole (Protonix)	80 mg	8 mg/h	Up to 72 h
Esomeprazole (Nexium)	80 mg	8 mg/h	Up to 72 h

Octreotide

The somatostatin analogue octreotide can be used to treat patients with both variceal and nonvariceal upper GI bleeding. This medication is indicated in patients with variceal bleeding or acute upper GI bleeding thought secondary to varices, using a loading dose of 25 to 50 µg followed by an intravenous infusion of 25 to 50 µg/h. Octreotide may also be helpful in patients with nonvariceal bleeding. While a useful adjunctive treatment in patients with non-variceal upper GI bleeding, octreotide should not be prescribed routinely to patients with nonvariceal upper GI bleeding. For all suspected etiologies of upper GI bleeding, the use of octreotide should be considered in patients who have persistent bleeding on optimal medical management, including PPIs, who are poor surgical risks (such as those with multiple comorbidities already in the hospital).

Endoscopic treatments

Endoscopic therapy for upper GI bleeding has been demonstrated to yield significant improvement in hemostasis, number of units of blood transfused, number of emergency interventions, hospital length of stay, and hospital costs. Endoscopic therapy is the standard of care for patients with the high-risk stigmata of recent hemorrhage of active bleeding or a nonbleeding visible vessel. The management of adherent clots has been controversial with recent data suggesting a possible role for endoscopic therapy.

For many conditions including active bleeding, visible vessels, and esophageal varices, endoscopic techniques are the mainstay of treatment. The timing and occasion for endoscopic treatment often depends on the clinical presentation and/or findings at time of endoscopy. An upper endoscopy examination is indicated within 24 hours of presentation for patients with suspected nonvariceal upper GI bleeding. Conditions that warrant rapid triage and planned endoscopy include bleeding in the setting of known varices, persistent hypotension requiring pressors, and increasing transfusion requirement. However, hemodynamic stability, protection of the airway (if necessary), and platelets, FFP, or vitamin K administration when applicable may be necessary steps prior to endoscopic evaluation regardless of the

urgency. In addition, data support endoscopic evaluation within 24 hours and do not support a need for more urgent endoscopy in most patients with nonvariceal upper GI bleeding.

PRACTICE POINT

Upper endoscopy is indicated within 24 hours of presentation for patients with suspected upper GI bleeding but most patients do not require more urgent endoscopy. Hemodynamic stability is necessary prior to endoscopy.

Optimization of visualization

One of the challenges of managing patients with GI bleeding is visualization during endoscopy due to blood within the gastrointestinal tract. This problem may be overcome by the use of a variety of techniques or a combination of endoscopic techniques including the use of double-channel or large-channel endoscopes and vigorous irrigation.

In addition to endoscopic techniques, intravenous **erythromycin** (250 mg IV bolus or 3 mg/kg over 30 minutes) can be used for its prokinetic properties to increase gastric emptying and clear the stomach of blood. The **erythromycin** is given intravenously 30 to 120 minutes prior to endoscopy as a useful adjunctive treatment in patients with large gastrointestinal bleeds. It can be used either initially or after an endoscopy shows large amounts of blood remaining in the stomach with withdrawal of the endoscope and the use of **erythromycin** before proceeding again with endoscopy. Metoclopramide may also be used similarly, although there are less available data about its effectiveness in patients with upper GI bleeding.

Methods to control bleeding

The current endoscopic modalities to treat nonvariceal GI bleeding include the use of injection therapies (primarily with dilute **epinephrine**), contact thermal therapies including heater and bipolar probes, noncontact thermal methods (predominantly argon plasma coagulation), mechanical treatments including a variety of clips including larger over-the-scope clips and band-ligation techniques, and a combination of the above treatment modalities (typically, injection therapies combined with one of the other modalities) (**Table 156-5**). For nonvariceal upper GI bleeding, combination therapy or use of hemoclips improves outcomes and decreases recurrent bleeding. For esophageal variceal bleeding, band ligation to tamponade blood flow offers effective treatment and outcomes. Success with injection of glues in the treatment of gastric varices has been demonstrated. Decompression of portal pressures when possible is the optimal therapy.

TABLE 156-5

Available Modalities for Endoscopic Therapy

- **Injection therapy**
 - a. Epinephrine
 - b. Hypertonic saline
 - c. Sclerosant (absolute alcohol, polidoconol)
 - d. Tissue adhesives: cyanoacrylate, thrombin/fibrin glue
- **Thermal therapy**
 - a. Contact: HP, bipolar (gold probe, BICAP)
 - b. Noncontact: APC, Nd: YAG laser
- **Mechanical therapy**
 - a. Endoclips
 - b. Endoscopic band ligation
- **Dual therapy (combination of above modalities)**

APC, argon plasma cautery; BICAP, bipolar probe; HP, heater probe.

While endoscopic injection sclerotherapy and banding ligation in conjunction with pharmacologic treatment are the primary methods for controlling acute variceal bleeding, tamponade with a Sengstaken-Blakemore tube may be indicated when these methods fail or before endoscopy if a rapid bleed occurs. The use of these tubes is indicated for acute bleeding from esophageal or gastric varices unresponsive to medical therapy, tears at the gastroesophageal junction, Mallory-Weiss tears unresponsive to medical therapy, or esophageal exclusion to manage distal esophageal perforation. The Sengstaken-Blakemore tube is absolutely contraindicated for patients in whom bleeding has stopped and in those with recent surgery of the gastroesophageal junction or known esophageal stricture.

CASE 156-3

A 62-year-old man with a history of NSAID use and tobacco abuse sought medical attention for intermittent hematemesis for 1 day. His vital signs revealed a heart rate of 114 beats/min and a low-normal BP (100/60 mm Hg). Laboratory studies reported anemia with a hematocrit of 21% (baseline 45%). His physicians administered erythromycin intravenously and initiated an IV PPI. After stabilization with two large bore IVs, and initiation of blood transfusions, a gastroenterologist performed an upper endoscopy, which identified a visible bleeding vessel in a deep duodenal ulcer. The ulcer was injected with dilute epinephrine, and clips were placed to achieve hemostasis. The patient was monitored in an intensive care unit and had an uneventful postprocedure course.

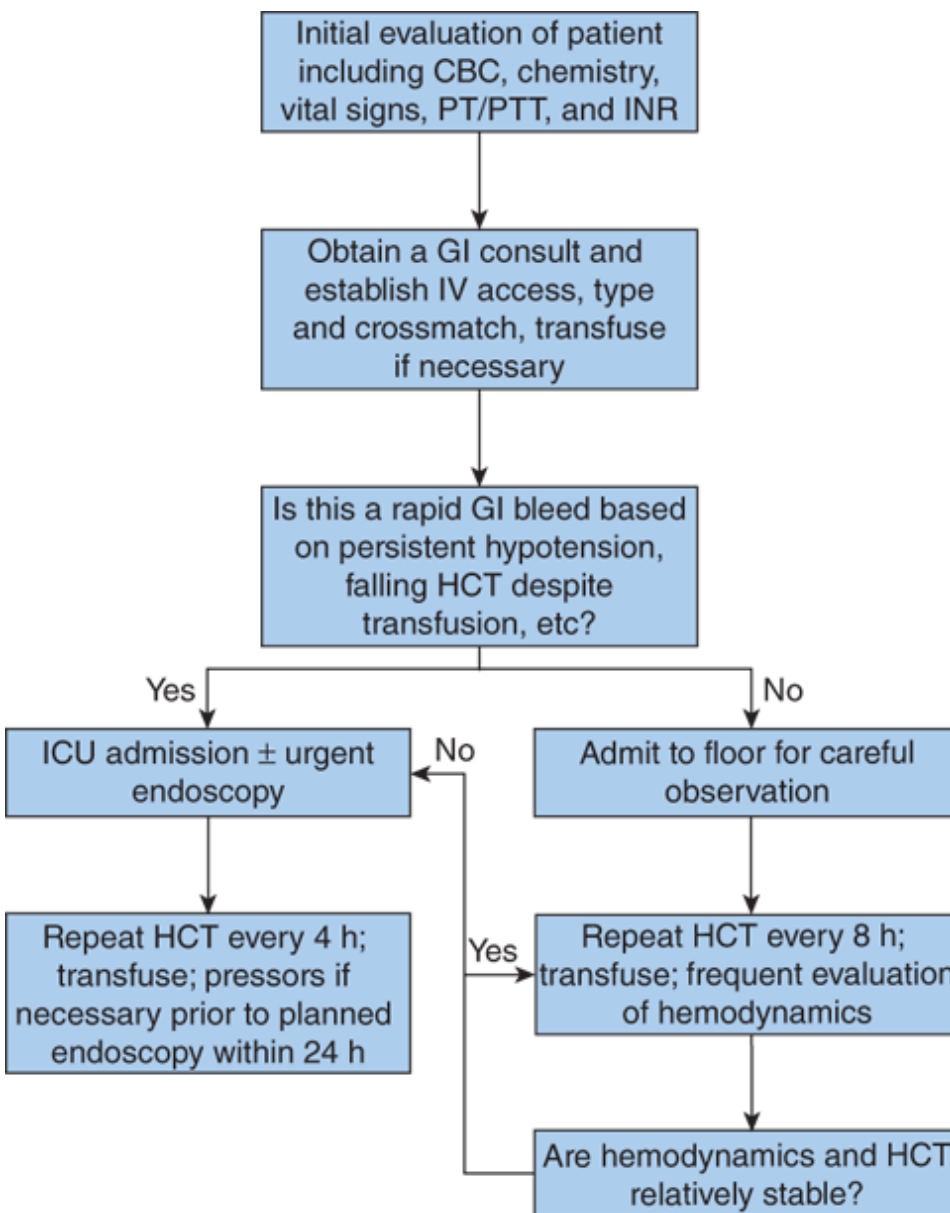
CONSULTATION

Hospitalists should involve a gastroenterologist early in the course of upper GI bleeding. Emergent consultation is appropriate with rapid bleeds in the setting of hypotension, a very low hematocrit, known varices, or other instances when urgent endoscopy might be required. In most cases, however, patient

stabilization is a crucial first step before gastroenterology intervention can be considered (**Figure 156-7**). A gastroenterology consultant can provide diagnostic and therapeutic advice early in the course of treatment and may be called as soon as a suspected upper GI bleeding patient is identified. In addition, IR angiography and surgery consultations are indicated in certain circumstances including patients requiring the ICU, those who require multiple blood transfusions or those who undergo endoscopy without identification of a source of bleed or ability to stop the bleeding. In small bowel or lower GI bleeding patients, the patient may benefit from a tagged red blood cell scan and subsequent angiography. **The key to consultation is to involve the consultant early and to communicate any significant clinical changes.**

Figure 156-7

Initial diagnostic algorithm for upper GI bleeding. (CBC, complete blood count; HCT, hematocrit; ICU, intensive care unit; INR, international normalized ratio; IV, intravenous; PT, prothrombin time; PTT, partial thromboplastin time.)



Source: Sylvia C. McKean, John J. Ross, Daniel D. Dressler, Danielle B. Scheurer: Principles and Practice of Hospital Medicine, Second Edition, www.accessmedicine.com Copyright © McGraw-Hill Education. All rights reserved.

COMPLICATIONS AND PROGNOSIS

COMPLICATIONS OF GI BLEEDING

The reported mortality of upper GI bleeding generally ranges from 2% to 14%. Complications of GI bleeding include hypotension and subsequent shock, which can lead to organ failure and death if not addressed in a timely manner. Aspiration of blood contents and subsequent hypoxemia and respiratory failure represents another adverse outcome. Tachycardia in the setting of pre-existing coronary heart disease can lead to ischemia. Anemia can also further lead to underperfusion of vital organs and some patients die of complications subsequent to inadequate resuscitation with blood and fluids as discussed above.

COMPLICATIONS OF TREATMENT

The majority of the complications associated with treatment of upper GI bleeding stem from endoscopic treatments. Major complications include myocardial infarction, perforation, aspiration, hemorrhage, and death. Minor complications of endoscopy include mucosal tears, medication reactions, and hypoxemia. In addition, a small risk exists of infection or bleeding from the endoscopic intervention itself. As sedatives are utilized for the procedure, the individual risks of each medication are added to the overall risk of the procedure. Many of the complications of GI endoscopy are cardiopulmonary in origin.

The initial evaluation of patients with upper GI bleeding should include risk assessment to triage patients to appropriate levels of care and perform resuscitation measures. Elective endotracheal intubation should be considered in patients at high risk of aspiration, such as those with massive upper GI bleeding. Attention must be given to provide the proper level of sedation required to perform the procedure.

Epinephrine is a vasoconstrictor that can lead to local ischemia and can also cause tachycardia. Large doses of injected **epinephrine** during endoscopic therapy especially in the region of the esophagus and upper stomach are associated with significant elevations of systolic blood pressure and tachycardia. The use of cautery can lead to direct tissue injury, which may further damage the GI tract. The use of cautery may lead to perforation, especially if excessive pressure is exerted on the site of therapy. The depth of penetration of tissue injury may be lessened by the use of injection therapy that lifts the tissue and provides a safety barrier.

PROGNOSIS

In patients presenting with upper GI bleeding, a number of clinical prognostic factors have been shown to be helpful in predicting a poor outcome. These include older age (age >60 years), the presence of shock, hematemesis and/or hematochezia, onset of bleeding in a patient hospitalized for other reasons, bleeding from malignancies of the upper GI tract or varices, increasing comorbid diseases, and patients with severe coagulopathies.

DISCHARGE PLANNING

RESTARTING ANTICOAGULATION IN UPPER GI BLEEDING

Patients who require anticoagulants (eg, warfarin) or platelet function inhibitors (eg, **clopidogrel**, aspirin) may develop signs of upper GI bleeding. Most GI bleeding episodes on antiplatelet agents or anticoagulants occur within the first year of therapy. Once a source of bleeding is identified, the timing of restarting

anticoagulants represents a difficult decision. Part of the decision-making process is evaluating the benefit of the anticoagulant or platelet inhibitor compared to the risk of bleeding. A patient with high-risk thromboembolic disease should have careful consideration of reversal of anticoagulation in consultation with the prescribing cardiologist or neurologist before endoscopy. A patient with low-risk thromboembolic disease can have complete reversal of anticoagulation followed by endoscopy. When to resume anticoagulation depends on the source and severity of bleeding as well as the underlying condition being treated. After hemostasis is achieved, patients who need to be restarted on anticoagulants have to be monitored carefully for subsequent bleeding.

OUTPATIENT SYMPTOM MONITORING

Patients discharged after upper GI bleeding require education about signs and symptoms to help identify any early bleeding. Patients should look for the return of black stool as a sign of a developing bleed. However, for the first 24 to 48 hours following active bleeding, patients can expect to see subsequent passage of blood, which may be maroon or black. Resting heart rate with evaluation for tachycardia can be measured by patients themselves. In addition, patients require education about over-the-counter medications to avoid that may affect GI bleeding, including drugs that contain NSAIDs, aspirin, or SSRIs. Bismuth subsalicylate (eg, Pepto-Bismol) should also be avoided as it causes the stool to appear dark and the stool color can be confused with melena.

OUTPATIENT LAB MONITORING

Patients who had bleeding related to supratherapeutic levels of warfarin need careful monitoring of the INR if the medication is subsequently resumed. Clinics that specialize in anticoagulation can reduce bleeding outcomes. In addition, anemic patients should have hemoglobin levels checked in the outpatient setting 1 to 2 weeks following discharge, as asymptomatic recurrent bleeding can present with worsening anemia. For upper GI bleeding, an elevated BUN can be a useful marker of recurrent bleeding. Patients with *H. pylori*-related bleeding should have an *H. pylori* breath test or stool antigen performed if upper endoscopy is not scheduled to confirm that *H. pylori* has been successfully eradicated.

QUALITY IMPROVEMENT TO ADDRESS PERFORMANCE GAPS

PREVENTION

Primary and secondary prevention of upper GI bleeding requires careful evaluation of a patient's underlying medical problems and current medications. NSAID use should be minimized whenever possible and, if necessary, one should monitor frequently for symptoms and signs of GI bleeding, which may indicate peptic ulcer disease, esophagitis, gastritis, or duodenitis, which can follow NSAID use. β -blocker therapy with propranolol or nadolol should be used in patients with cirrhosis and a prior history of esophageal variceal bleeding (secondary prophylaxis) regardless of grade, or large esophageal varices without previous hemorrhage (primary prophylaxis). The comparison of nonselective β -blockers and placebo in several randomized controlled trials demonstrates a greater than 20% absolute risk reduction in subsequent bleeding from esophageal varices. Prevention of GI bleeding in admitted patients requires careful monitoring of medications (eg, anticoagulants and antiplatelet agents) and comorbidities. Patients at high risk for stress ulcer bleeding (eg, mechanical ventilation) benefit from prophylactic PPI dosing.

TRANSITIONS OF CARE

As patients transition from in-hospital care to outpatient management, several factors must be considered. Medications must be adjusted from the inpatient setting to appropriate outpatient regimens. For example, although patients with upper GI bleeding are often managed with high-dose proton-pump inhibitors in the hospital, at discharge, most patients can be managed on a once daily standard dose oral proton-pump inhibitor or twice daily for high-risk bleeding lesions.

Findings at endoscopy may require changes in the medical regimen or subsequent endoscopies. In patients with peptic ulcer disease, biopsy results positive for *H. pylori* infection indicate a necessary outpatient antibiotic regimen. Most patients with significant gastric ulcers will require repeat endoscopy examinations in 8 to 12 weeks to confirm healing of the ulcer and for biopsy to exclude malignancy.

Communication of in-hospital caregivers with outpatient physicians is critical to the successful management of patients with significant upper GI bleeding. Follow-up with a primary physician should take place for repeat blood draws and physical examination within 7 to 14 days of hospital discharge, and follow-up with a gastroenterologist either for a planned repeat endoscopy or office visit should take place within 2 to 4 weeks depending on the source of bleeding. For example, variceal bleeding may require a 2-week follow-up endoscopy for further banding and assessment of healing. Outpatient planning for follow-up with gastroenterology, general surgery, and primary care should take place prior to discharge with the consulting specialists and the outpatient primary care provider.

DISPARITIES IN HEALTH CARE

While scarce literature identifies disparities in health care in gastroenterology, one recent study showed that significant proportion of visits to US emergency departments for acute GI illnesses are associated with a delay in initial clinical assessment. Of the patient visits analyzed, there were an estimated 1.6 million emergency department visits for acute pancreatitis, 2.2 million visits for appendicitis, 1.2 million visits for cholecystitis, and 3.9 million visits for upper GI bleeding. The study showed that Hispanic patients waited longer and had a higher frequency of delays compared with other racial and ethnic groups. Future investigations into racial, ethnic, gender and other disparities in health delivery and performance in upper GI bleeding are needed.

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