Chapter 75: Upper Gastrointestinal Bleeding

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FIGURE 75–1.

INTRODUCTION AND EPIDEMIOLOGY

Upper GI (UGI) bleeding is any GI bleeding originating proximal to the ligament of Treitz. The overall annual incidence of UGI bleeding ranges from 39 to 172 per 100,000 in Western countries.[1,2,3] Difference in prevalence between countries is attributed to variations in *Helicobacter pylori* rates, socioeconomic conditions, and prescription patterns of ulcer-healing and ulcer-promoting medications.[2] Increasing age, coexistent organ system disease, and recurrent hemorrhage are factors associated with increased morbidity and mortality.[3]

PATHOPHYSIOLOGY

PEPTIC ULCER DISEASE

Despite a downward trend in prevalence over the past 20 years, peptic ulcer disease, which includes gastric, duodenal, esophageal, and stomal ulcers, is still considered the most common cause of UGI bleeding.[2,4] However, the Analysis of Clinical Outcomes Research Initiative found gastric and duodenal ulcers in only 20.6% of 7822 endoscopies performed for suspected UGI bleeding.[4] This number is much lower than previous estimates of up to 50%.[5,6] Awareness that aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), and smoking cause bleeding and increased recognition and treatment of *H. pylori* infection may be responsible for decreased incidence.[7,8,9,10]

EROSIVE GASTRITIS AND ESOPHAGITIS

Erosive gastritis, esophagitis, and duodenitis are also common causes of GI hemorrhage.[11] Common predisposing factors include alcohol, salicylates, and NSAIDs. Infection, toxic ingestion, radiation, and stress from severe illness may also cause erosive gastritis. Stress-related mucosal disease occurs in patients with overwhelming sepsis, trauma, or respiratory failure requiring mechanical ventilation. *Candida*, herpes simplex virus, cytomegalovirus, and human immunodeficiency virus are potential sources of esophageal bleeding from infection.

ESOPHAGEAL AND GASTRIC VARICES

Esophageal and gastric varices result from portal hypertension and, in the United States, are most often a result of alcoholic liver disease.[12] Although varices account for a small percentage of all cases of UGI hemorrhage, they can rebleed and carry a high mortality rate. However, many patients with end-stage cirrhosis never develop varices;

many patients with documented varices never bleed; and many patients with a documented history of varices presenting with UGI bleeding will actually bleed from nonvariceal sites. Variceal bleeding is the cause of UGI bleeding in cirrhotics 59% of the time, followed by peptic ulcer disease in 16% of cases.[13] In-hospital mortality rates for any type of GI bleed in cirrhotics are essentially double those of noncirrhotic patients.[14]

MALLORY-WEISS SYNDROME

Mallory-Weiss syndrome is bleeding secondary to a longitudinal mucosal tear at the gastroesophageal junction. The classic history is repeated vomiting followed by bright red hematemesis. The syndrome can be associated with alcoholic binge drinking, diabetic ketoacidosis, or chemotherapy administration. The Valsalva maneuver, such as from coughing or seizures, is also a reported cause.

DIEULAFOY LESIONS

Dieulafoy lesions are arteries of the GI tract that protrude through the submucosa. They are most commonly found in the lesser curvature of the stomach but may be found anywhere in the GI tract; 80% to 95% are found within 6 cm of the gastroesophageal junction.[15] These lesions are characterized by intermittent massive GI bleeding, without the standard predisposing factors of liver disease or NSAID use. Dieulafoy lesions are difficult to diagnose endoscopically, and sometimes patients report multiple previous diagnostic maneuvers with negative results.

OTHER CAUSES

Arteriovenous malformation and malignancy are other causes of UGI hemorrhage. Significant bleeding from ear, nose, and throat sources can also masquerade as GI hemorrhage. An aortoenteric fistula secondary to a preexisting aortic graft is an unusual but important cause of bleeding to keep in mind. Classically, this presents as a self-limited "herald" bleed with hematemesis or hematochezia, which precedes massive hemorrhage and exsanguination.

DIAGNOSIS

HISTORY

Ask about hematemesis, coffee-ground emesis, or melena. Classically, hematemesis and coffee-ground emesis suggest a UGI source. The presence of melena and age <50 years old more likely indicate an upper GI bleed versus a lower GI bleed, even in patients without hematemesis.[16] Vomiting and retching, followed by hematemesis, suggest a Mallory-Weiss tear. Be sure to ask about prior episodes of GI bleeding and any interventions performed. A history of an aortic graft should suggest bleeding from an aortoenteric fistula. Review the patient's medication list carefully. Salicylates, glucocorticoids, NSAIDs, and anticoagulants all place the patient at high risk for GI bleed. Alcohol abuse is strongly associated with a number of causes of bleeding, including peptic ulcer disease, erosive gastritis, and esophageal varices. Ingestion of iron or bismuth can simulate melena. Liquid medications with red dye, as well as certain foods, such as beets, can simulate hematochezia. In such cases, stool guaiac testing will be negative. Inquire about past history of GI bleeding, even though recurrent bleeding episodes may originate from different sources.

Although the medical history may suggest the source of bleeding, history can also be misleading. For instance, what initially appears to be lower GI bleeding may actually be a UGI bleed in disguise. **Bright red or maroon rectal bleeding unexpectedly originates from UGI sources about 14% of the time**.[16] Although patients volunteer complaints of hematemesis or melena, if there is no vomiting or the patient has not noted tarry stools, signs may be subtle. Patients with hypotension, tachycardia, angina, syncope, weakness, confusion, or cardiac arrest may have underlying GI hemorrhage.

PHYSICAL EXAMINATION

Visual inspection of the vomitus for a bloody, maroon, or coffee-ground appearance is the most reliable way to diagnose UGI bleeding in the ED. Consider keeping a sample of the vomitus or nasogastric (NG) aspirate at bedside for the gastroenterologist to view.

Vital signs may reveal obvious hypotension and tachycardia or more subtle findings such as decreased pulse pressure or tachypnea. Younger patients and those without comorbidities can tolerate substantial volume loss with minimal or no changes in vital signs. Paradoxical bradycardia may occur even in the face of profound hypovolemia. Remember that comorbid conditions and medications may mask the body's physiologic response to volume loss. β-Blockers, for example, will prevent tachycardia. Patients with baseline hypertension may have relatively normal blood pressure in the setting of hypovolemia.

Cool, clammy skin is an obvious sign of shock. Spider angiomas, palmar erythema, jaundice, and gynecomastia suggest liver disease. Petechiae and purpura suggest an underlying coagulopathy. Facial lesions, cutaneous macules, or telangiectasias may be suggestive of the Peutz-Jeghers, Rendu-Osler-Weber, or Gardner's syndromes. A careful ear, nose, and throat examination can reveal an occult bleeding source that has resulted in swallowed blood and subsequent coffee-ground emesis. Abdominal examination may disclose tenderness, masses, ascites, or organomegaly.

Perform rectal examination to detect the presence of blood and its appearance, whether bright red, maroon, or melanotic.

LABORATORY TESTING

In patients with significant bleeding, the single most important laboratory test is to obtain blood for type and cross-match in case transfusion is needed. A CBC is also important, although the initial hematocrit level may not reflect the actual amount of acute blood loss. In addition, consider BUN, creatinine, electrolyte, glucose, coagulation, and liver function studies. UGI hemorrhage will elevate BUN levels through digestion and absorption of hemoglobin. A BUN:creatinine ratio ≥30 suggests a UGI source of bleeding.[17] Coagulation studies, including INR, partial thromboplastin time, and platelet count, are useful in patients taking anticoagulants and those with underlying hepatic disease. Obtain an ECG in patients with underlying coronary artery disease. Silent cardiac or mesenteric ischemia can develop if bleeding decreases cardiac or mesenteric perfusion. A single elevated lactate level is a sentinel sign of severe illness. The success or failure of resuscitation efforts can be assessed by following dynamic lactate levels, because a rising lactate level in the hospital setting is a clear predictor of in-hospital mortality.[18]

Routine abdominal and chest radiographs are of limited value and are not needed in the absence of specific clinical indications. Barium contrast studies are contraindicated because barium may hinder subsequent endoscopy or angiography.

In cases where traditional endoscopy is unavailable or endoscopic visualization is unable to find the source, consider tagged red-cell scintigraphy or visceral angiography. Both of these tests will demonstrate the source only in cases of active bleeding. Scintigraphy and angiography help localize the source of bleeding to determine whether medical or surgical management is optimal.

NASOGASTRIC LAVAGE

NG intubation and aspiration are diagnostic and therapeutic.[19] In patients without a history of hematemesis, a positive aspirate provides strong evidence for a UGI source of bleeding. High-risk lesions are more likely in patients with bloody aspirates. Visual inspection of the aspirate to identify bloody, maroon, or coffee-ground material verifies UGI bleeding. Early NG lavage is associated with decreased time to endoscopy.[20] NG tube placement and lavage can confirm the diagnosis of UGI bleeding and stratify risk.

A negative NG aspirate does not conclusively exclude a UGI source. Intermittent bleeding, pyloric spasm, or edema preventing reflux of duodenal blood can cause false-negative results. Ultimately, NG aspiration yields a positive result in only 23% of patients without hematemesis who have occult UGI bleeding.[21]

Guaiac testing of NG aspirate can yield both false-negative and false-positive results. Conventional stool guaiac cards may be falsely negative. However, guaiac cards specifically designed for UGI sources are available. Conversely, even minimally traumatic NG intubation can result in positive guaiac testing even in the face of a clear aspirate. **Visual inspection of the aspirate for a bloody, maroon, or coffee-ground appearance is the most reliable way to diagnose UGI bleeding in the ED**.

If bright red blood or clots are found in the NG aspirate, perform gentle gastric lavage. Room temperature water is the preferred irrigant. Maintain the NG tube on mild, intermittent suction. Suction that is too vigorous may produce gastric erosions that can confuse findings on subsequent endoscopy.

As of this writing, there is no evidence to support concerns that NG tube passage may provoke bleeding in patients with varices.

RISK STRATIFICATION

Risk stratification depends on clinical judgment. There are no universally accepted pre-endoscopy risk stratification practice guidelines. However, the literature does seem to agree on those individuals that qualify as very low risk (**Table 75–1**). Pre-endoscopic predictors of higher risk include advanced age, comorbidities, red hematemesis, hematochezia, red blood on NG aspirate, hemodynamic instability, and abnormal laboratory studies.[22,23,24,25,26] Other high-risk factors include prior variceal banding, clamping or cauterization of an ulcer bed, or the transjugular intrahepatic portosystemic shunt procedure.

Table 75–1

Upper GI Bleeding Risk

Very Low Risk	High Risk
<60 y old	Advanced age
No major comorbidities	Comorbidities and prior endoscopic or transjugular intrahepatic portosystemic shunt procedures
No history of red hematemesis	Red hematemesis
No hematochezia	Hematochezia or melena
Negative nasogastric (NG) aspirate	Positive NG aspirate
Hemodynamically stable at ED presentation	Hemodynamically unstable
Normal laboratory studies	Abnormal laboratory studies

TREATMENT

Initial management is stabilization. Patients in hemorrhagic shock require emergent resuscitation, including two large-bore IVs, typed and cross-matched blood with the consideration of massive transfusion protocols, and in selected cases, early airway management. Intubating a patient with a UGI bleed who is hemodynamically unstable can be a perilous procedure. Aggressively resuscitate prior to intubation, and consider using smaller doses of the induction agent to minimize peri-intubation hypotension or arrest.[27] ED treatment is summarized in **Table 75–2**.

Table 75–2

Treatment of Upper GI Bleed

Treatment	Dose	Comments
Blood transfusion		Transfuse if ≤7 grams/dL in most; ≤9 grams/dL in older patients or patients with comorbidities
Correct coagulopathy		Correct if INR is elevated or platelets <50,000; or if bleeding severe, correct coagulopathy unless contraindications to correction (e.g., stents)
Omeprazole	80-milligram IV bolus then infusion of 8 milligrams/h	Labeled use for ulcer bleeding
Octreotide	50-microgram bolus then infusion of 25–50 micrograms/h	Unlabeled use for varices; for elderly, begin at lower dose range of 25-microgram bolus and infusion of 25 micrograms/h
Antibiotics	Ciprofloxacin 400 milligrams IV or ceftriaxone 1 gram IV	Antibiotics for cirrhotics with UGI bleeding

BLOOD TRANSFUSIONS

When UGI bleeding is severe, blood transfusions can be lifesaving. If a large amount of blood product is anticipated, use massive transfusion protocols.[28] See chapter 13, "Fluid and Blood Resuscitation in Traumatic Shock" for discussion of massive transfusion. In less severe cases, the decision to transfuse can be difficult because hemoglobin concentrations do not fall until after hemodilution has occurred. Individualize thresholds for transfusion based on underlying comorbidities and hemodynamic status.[29] Liberally transfusing all bleeding patients using a high threshold (hemoglobin <9 grams/dL) can cause harm.[5] A restrictive transfusion threshold using hemoglobin concentrations of <7 grams/dL in most patients and <9 grams/dL in older patients with comorbidities who are not tolerating the acute anemia is recommended.[5,29]

COAGULOPATHY

In patients with life-threatening bleeding receiving anticoagulants, reverse the coagulopathy without concern for the INR unless there are contraindications to reversal, such as cardiac or vascular stents. In less severe bleeding, carefully consider the risks of reversal therapy. An INR ≥1.5 is a significant predictor of mortality in patients with a UGI bleed who are receiving anticoagulants.[30] International consensus guidelines recommend reversal of

coagulopathy for UGI bleed patients who have an elevated INR or platelet counts <50,000/μL.[29,31] Coagulopathies from other causes such as the newer oral antithrombin and Xa inhibitors should be managed according to institutional protocols. See Figure 239–1 and Table 239–4 in chapter titled "Thrombotics and Antithrombotics" for recommendations for anticoagulant reversal. Reversal should not delay time to endoscopy. Tranexamic acid, an antifibrinolytic agent, has shown no benefit in the management UGI bleeding.[32]

PROTON PUMP INHIBITORS

Consensus guidelines continue to recommend proton pump inhibitors for patients with nonvariceal bleeding from peptic ulcer disease.[23] When proton pump inhibitors are given at high dose, the gastric pH remains neutral. Clot formation from platelet aggregation is dependent on a pH >6.0.[33] Administer a high-dose proton pump inhibitor such as omeprazole 80 milligrams IV bolus followed by infusion of 8 milligrams/h[34] because the cause of bleeding cannot be determined without endoscopy. In patients with peptic ulcer bleeding, proton pump inhibitors reduce the need for surgery, the length of stay in the hospital, and signs of bleeding.[35]

SOMATOSTATIN ANALOGS/OCTREOTIDE

Octreotide is a long-acting analog of somatostatin that elicits several actions in patients with UGI bleeding. It inhibits the secretion of gastric acid, reduces blood flow to the gastroduodenal mucosa, and causes splanchnic vasoconstriction.[36] The dose is a 50-microgram bolus followed by a continuous infusion of 25 to 50 micrograms/h. Despite its widespread use in the United States, octreotide does not appear to provide a clear benefit on mortality.[37] In contrast, terlipressin is preferred in countries where it is available because it is the only drug treatment associated with a reduction in mortality.[38]

ANTIBIOTICS

Patients with cirrhosis have an impaired immune system and have an increased risk of gut bacterial translocation during an acute bleeding episode. Prophylactic antibiotics (e.g., ciprofloxacin 400 milligrams IV or ceftriaxone 1 gram IV) reduce infectious complications, may decrease mortality,[39] and should be started as soon as possible.

PROMOTILITY AGENTS

Erythromycin and metoclopramide are examples of promotility agents used to enhance endoscopic visualization.[40] However, they are not recommended for routine use, but may be considered if the patient is undergoing endoscopy in the ED and the patient is suspected to have large amounts of blood in the UGI tract.[29]

ENDOSCOPY

UGI endoscopy is the diagnostic study of choice. Endoscopy allows visualization of the source of bleeding (in most cases) and administration of hemostatic therapy.[41] The optimal timing relates to the severity of the bleeding. Early endoscopy (within 24 hours of presentation) is recommended for most patients because it is associated with a

significant cost reduction and decreased length of stay.[19,20,41,42,43] An unstable patient may benefit from emergent endoscopy immediately following resuscitation.

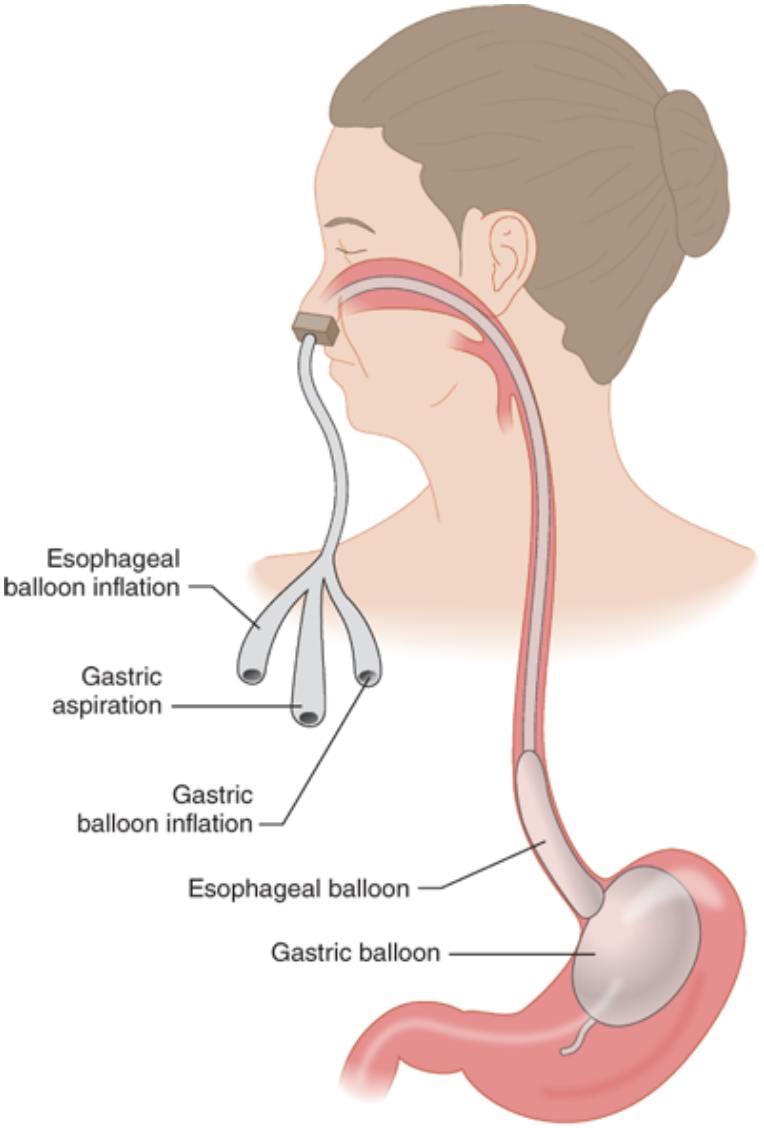
Endoscopic treatment options commonly used for variceal bleeding include variceal ligation and sclerotherapy. Clips, thermocoagulation, and sclerosant injections alone or in combination with epinephrine injections are commonly used in ulcerative lesions.

In some practices, the ED physician is asked to provide sedation for the endoscopist. Pretreat with an antiemetic such as ondansetron. Use short-acting titratable drugs with both analgesic properties (fentanyl) and sedative properties (versed or propofol). Ideal agents can be reversed if the patient's condition changes.[44,45,46] In unstable patients, one must consider using cardiovascular stable agents such as etomidate or ketamine. While providing sedation, consider that the most noxious part of the procedure is when the scope is passed around the tongue.

BALLOON TAMPONADE

Balloon tamponade is an effective short-term solution for life-threatening variceal bleeding. Because of the high rate of complications, it should be reserved for temporary stabilization of patients for transfer to an appropriate institution or until endoscopy can be done. The **Sengstaken-Blakemore tube** (which has a 250-cc gastric balloon, an esophageal balloon, and a single gastric suction port) (**Figure 75–1**) and the **Minnesota tube** (with an added esophageal suction port above the esophageal balloon) are examples of balloons that have been used. Adverse reactions include mucosal ulceration, esophageal or gastric rupture, asphyxiation from tracheal compression, and aspiration. Strongly consider intubation prior to balloon tamponade.

FIGURE 75–1.
Sengstaken-Blakemore tube.



The device can be inserted either nasally or orally. The gastric balloon is inflated first. It is critical to know that one is in the stomach before fully inflating the gastric balloon. If bleeding does not stop, inflate the esophageal balloon while using a manometer to ensure the pressure does not exceed 50 mm Hg. Confirm tube placement by x-ray. To secure the tube, apply 1 kg of traction by attaching the distal end of the tube to a 1-L bag of saline hung from an IV pole. Alternatively, the tube can be secured directly to a football helmet.

SURGERY

Patients who do not respond to both pharmacologic and endoscopic treatments may require emergent surgery. In patients with variceal bleeding, there are two basic types of operations: shunt operations (trans-jugular intrahepatic portosystemic shunt procedure) and nonshunt operations (esophageal transection or gastroesophageal junction devascularization). In nonvariceal bleeding, percutaneous embolization or subtotal or total gastrectomy can be performed. Emergent surgical consultation is considered prudent in case of uncontrolled bleeding.

DISPOSITION AND FOLLOW-UP

Patients with significant UGI bleeding require intensive care unit admission and early endoscopy. Very-low-risk patients (Table 75–1) may be eligible for ED observation or be discharged home with adequate outpatient follow-up.

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