Approach to acute upper gastrointestinal bleeding in adults

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INTRODUCTION

Patients with acute upper gastrointestinal (GI) bleeding commonly present with hematemesis (vomiting of blood or coffee-ground-like material) and/or melena (black, tarry stools). The initial evaluation of patients with acute upper GI bleeding involves an assessment of hemodynamic stability and resuscitation if necessary. Diagnostic studies (usually endoscopy) follow, with the goals of diagnosis, and when possible, treatment of the specific disorder.

The diagnostic and initial therapeutic approach to patients with clinically significant (ie, the passage of more than a scant amount of blood) acute upper GI bleeding will be reviewed here. While there is variability among guidelines, this approach is generally consistent with a multidisciplinary international consensus statement updated in 2019, a 2012 guideline issued by the American Society for Gastrointestinal Endoscopy, a 2021 guideline issued by the American College of Gastroenterology, a 2015 guideline issued by the European Society of Gastrointestinal Endoscopy, and a 2021 update issued by the European Society of Gastrointestinal Endoscopy [1-5]. The causes of upper GI bleeding, the endoscopic management of acute upper GI bleeding, and the management of active variceal hemorrhage are discussed separately. (See "Causes of upper gastrointestinal bleeding in adults" and "Overview of the treatment of bleeding peptic ulcers" and "Overview of the management of patients with variceal bleeding" and "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

A table outlining the major causes, clinical features, and emergency management of acute severe upper gastrointestinal bleeding in adults is provided (table 1).

INITIAL EVALUATION

The initial evaluation of a patient with a suspected clinically significant acute upper GI bleed includes a history, physical examination, and laboratory tests. The goal of the evaluation is to assess the severity of the bleed, identify potential sources of the bleed, and determine if there are conditions present that may affect subsequent management. The information gathered as part of the initial evaluation is used to guide decisions regarding triage, resuscitation, empiric medical therapy, and diagnostic testing.

Factors that are predictive of a bleed coming from an upper GI source identified in a meta-analysis included a patient-reported history of melena (likelihood ratio [LR] 5.1-5.9), melenic stool on examination (LR 25), blood or coffee grounds detected during nasogastric lavage (LR 9.6), and a ratio of blood urea nitrogen to serum creatinine greater than 30 (LR 7.5) [6]. On the other hand, the presence of blood clots in the stool made an upper GI source less likely (LR 0.05). Factors associated with severe bleeding included red blood detected during nasogastric lavage (LR 3.1), tachycardia (LR 4.9), or a hemoglobin level of less than 8 g/dL (LR 4.5-6.2).

Bleeding manifestations — Hematemesis (either red blood or coffee-ground emesis) suggests bleeding proximal to the ligament of Treitz. The presence of frankly bloody emesis suggests moderate to severe bleeding that may be ongoing, whereas coffee-ground emesis suggests more limited bleeding.

The majority of melena (black, tarry stool) originates proximal to the ligament of Treitz (90 percent), though it may also originate from the oropharynx or nasopharynx, small bowel, or colon [7]. Melena may be seen with variable degrees of blood loss, being seen with as little as 50 mL of blood [8].

Hematochezia (red or maroon blood in the stool) is usually due to lower GI bleeding. However, it can occur with massive upper GI bleeding [9], which is typically associated with orthostatic hypotension. (See 'Physical examination' below.)

Past medical history — Patients should be asked about prior episodes of upper GI bleeding, since up to 60 percent of patients with a history of an upper GI bleed are bleeding from the same lesion [10]. In addition, the patient's past medical history should be reviewed to identify important comorbid conditions that may lead to upper GI bleeding or may influence the patient's subsequent management.

Potential bleeding sources suggested by a patient's past medical history include:

- Varices or portal hypertensive gastropathy in a patient with a history of liver disease or excess alcohol use
- Aorto-enteric fistula in a patient with a history of an abdominal aortic aneurysm or an aortic graft
- Angiodysplasia in a patient with renal disease, aortic stenosis, or hereditary hemorrhagic telangiectasia
- Peptic ulcer disease in a patient with a history of *Helicobacter pylori* (*H. pylori*) infection, nonsteroidal anti-inflammatory drug (NSAIDs) use, antithrombotic use, or smoking
- Malignancy in a patient with a history of smoking, excess alcohol use, or *H. pylori* infection
- Marginal ulcers (ulcers at an anastomotic site) in a patient with a gastroenteric anastomosis

Comorbid illnesses may influence patient management in the setting of an acute upper GI bleed. Comorbid illnesses may:

- Make patients more susceptible to adverse effects of anemia (eg, coronary artery disease, pulmonary disease). Such patients may need to be maintained at higher hemoglobin levels than patients without these disorders. (See 'Blood product transfusions' below.)

- Predispose patients to volume overload in the setting of vigorous fluid resuscitation or blood transfusions (eg, renal disease, heart failure). Such patients may need more invasive monitoring during resuscitation. (See 'General support' below.)

- Result in bleeding that is more difficult to control (eg, coagulopathies, thrombocytopenia, significant hepatic dysfunction). Such patients may need additional hemostatic therapies. (See 'Blood product transfusions' below.)

- Predispose to aspiration of GI contents into the lungs (eg, dementia, hepatic encephalopathy). Endotracheal intubation should be considered in such patients. (See 'General support' below.)

Medication history — A thorough medication history should be obtained, with particular attention paid to drugs that:

- Predispose to peptic ulcer formation, such as aspirin and other NSAIDs, including COX-2 inhibitors (see "NSAIDs (including aspirin): Pathogenesis and risk factors for gastroduodenal toxicity").

- Are associated with pill esophagitis (see "Pill esophagitis").

- Increase risk of bleeding, such as anticoagulants (including warfarin and the direct oral anticoagulants) and antiplatelet agents (eg, P2Y12 inhibitors and aspirin).

- Have been associated with GI bleeding, including selective serotonin reuptake inhibitors (SSRI), calcium channel blockers, and aldosterone antagonists.

- May alter the clinical presentation, such as bismuth, charcoal, licorice, and iron, which can turn the stool black.

Symptom assessment — Patients should be asked about symptoms as part of the assessment of the severity of the bleed and as a part of the evaluation for potential bleeding sources. Symptoms that suggest the bleeding is severe include orthostatic dizziness, confusion, angina, severe palpitations, and cold/clammy extremities.

Specific causes of upper GI bleeding may be suggested by the patient's symptoms [7]:

- Peptic ulcer – Upper abdominal pain
- Esophageal ulcer – Odynophagia, gastroesophageal reflux, dysphagia
- Mallory-Weiss tear – Emesis, retching, or coughing prior to hematemesis
- Variceal hemorrhage or portal hypertensive gastropathy: Jaundice, abdominal distention (ascites)
- Malignancy – Dysphagia, early satiety, involuntary weight loss, cachexia

Physical examination — The physical examination is a key component of the assessment of hemodynamic stability. Signs of hypovolemia include [7]:

- Mild to moderate hypovolemia (less than 15 percent of blood volume lost) – Resting tachycardia.

- Blood volume loss of at least 15 percent – Orthostatic hypotension (a decrease in the systolic blood pressure of more than 20 mmHg and/or an increase in heart rate of 20 beats per minute when moving from recumbency to standing).

- Blood volume loss of at least 40 percent – Supine hypotension.

Examination of the stool color may provide a clue to the location of the bleeding, but it is not a reliable indicator. In a series of 80 patients with severe hematochezia (red or maroon blood in the stool), 74 percent had a colonic lesion, 11 percent had an upper GI lesion, 9 percent had a presumed small bowel source, and no site was identified in 6 percent [9]. Nasogastric lavage may be carried out if there is doubt as to whether a bleed originates from the upper GI tract, although is not a sensitive or specific test. (See 'Nasogastric lavage' below.)

The presence of abdominal pain, especially if severe and associated with rebound tenderness or involuntary guarding, raises concern for perforation. If any signs of an acute abdomen are present, further evaluation to exclude a perforation is required prior to endoscopy.

Finally, as with the past medical history, the physical examination should include a search for evidence of significant comorbid illnesses. (See 'Past medical history' above.)

Laboratory data — Laboratory tests that should be obtained in patients with acute upper gastrointestinal bleeding include a complete blood count, serum chemistries, liver tests, and coagulation studies. In addition, serial electrocardiograms and cardiac enzymes may be indicated in patients who are at risk for a myocardial infarction, such as older adults, patients with a history of coronary artery disease, or patients with symptoms such as chest pain or dyspnea. (See "Diagnosis of acute myocardial infarction".)

The initial hemoglobin level in patients with acute upper GI bleeding may be at the patient's baseline because the patient is losing whole blood. With time, the hemoglobin level will decline as the blood is diluted by the influx of extravascular fluid into the vascular space and by fluid administered during resuscitation. The hemoglobin level should initially be monitored every two to eight hours, depending upon the severity of the bleed.

Acute bleeding does not alter the mean corpuscular volume (MCV). If the MCV is low, it may suggest iron deficiency, which could be caused by chronic bleeding. Anemia or other abnormalities on the CBC that persist after recovery from the acute bleeding event should be evaluated. (See "Diagnostic approach to anemia in adults".)

Because blood is absorbed as it passes through the small bowel and patients may have decreased renal perfusion, patients with acute upper GI bleeding typically have an elevated blood urea nitrogen (BUN)-to-creatinine or urea-to-creatinine ratio. Values >30:1 or >100:1, respectively, suggest upper GI bleeding as the cause [6,11-13]. The higher the ratio, the more likely the bleeding is from an upper GI source [11].

Nasogastric lavage — The use of nasogastric tube (NGT) placement in patients with suspected acute upper GI bleeding is not recommended, as studies have failed to demonstrate a benefit with regard to clinical outcomes [5,14,15]. As an example, a retrospective study looked at whether there were clinical benefits from NGT lavage in 632 patients admitted with gastrointestinal bleeding [16]. Patients who underwent NGT lavage were matched with patients with similar characteristics who did not undergo NGT lavage. NGT lavage was associated with a shorter time to endoscopy. However, there were no differences between those who underwent NGT lavage and those who did not with regard to mortality, length of hospital stay, surgery, or transfusion requirement. Similarly, in a randomized trial with 280 patients with upper GI

bleeding, there were no differences in rebleeding rates or mortality between patients who underwent NGT lavage and those who did not [17].

NGT lavage may be used when it is unclear if a patient has ongoing bleeding and thus might benefit from an early endoscopy. In addition, NGT lavage can be used to remove particulate matter, fresh blood, and clots from the stomach to facilitate endoscopy. (See "Inpatient placement and management of nasogastric and nasoenteric tubes in adults", section on 'Tube placement'.)

The presence of red blood or coffee ground material in the nasogastric aspirate also confirms an upper GI source of bleeding and predicts whether the bleeding is caused by a lesion at increased risk for ongoing or recurrent bleeding [16,18]. However, lavage may not be positive if bleeding has ceased or arises beyond a closed pylorus. Nasogastric aspiration of nonbloody bilious fluid suggests that the pylorus is open and that there is no active upper GI bleeding distal to the pylorus [9].

We suggest that patients only undergo NGT lavage if particulate matter, fresh blood, or clots need to be removed from the stomach to facilitate endoscopy. An alternative to NGT lavage in this situation is to use a prokinetic such as erythromycin. (See 'Prokinetics' below.)

GENERAL MANAGEMENT

Hemodynamically unstable patients — While the principles behind the management of all patients with upper gastrointestinal bleeding are similar, there are some special considerations when it comes to patients presenting with hemodynamic instability (shock, orthostatic hypotension) (table 1).

Intravenous access — Adequate peripheral access should be attained with either two 18 gauge or larger intravenous catheters and/or a large-bore, single-lumen central cordis.

Fluid resuscitation — Fluid resuscitation should begin immediately and should not be delayed pending transfer of the patient to an intensive care unit. The approach to fluid resuscitation in patients who are hemodynamically unstable is discussed in detail elsewhere. (See "Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock", section on 'Hemodynamic support'.)

Transfusion — For patients with active/brisk bleeding and hypovolemia, transfusion should be guided by hemodynamic parameters (eg, pulse and blood pressure), the pace of the bleeding, estimated blood loss, and the ability to stop the bleeding, rather than by serial hemoglobin

measurements. If the initial hemoglobin level is low (<7 g/dL) transfusions should be initiated [19,20]. In an acutely hemorrhaging patient, however, transfusion support should not be delayed while awaiting laboratory test results. (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult", section on 'Acute bleeding'.)

Patients without active bleeding who become hemodynamically stable with fluid resuscitation are managed like other patients who are hemodynamically stable. For most stable patients, a restrictive transfusion strategy is appropriate (transfuse if hemoglobin is <7g/dL [<70 g/L] rather than at a higher hemoglobin) (algorithm 1 and table 2). (See 'Blood product transfusions' below.)

Medications and endoscopy — The approach to medications (eg, proton pump inhibitors) and endoscopy are similar for patients with hemodynamic instability compared with patients who are hemodynamically stable. It is particularly important to ensure that these patients are adequately resuscitated prior to undergoing upper endoscopy. (See 'Medications' below and 'Upper endoscopy' below.)

Triage — All patients with hemodynamic instability or active bleeding (manifested by hematemesis, bright red blood per nasogastric tube, or hematochezia) should be admitted to an intensive care unit for resuscitation and close observation with automated blood pressure monitoring, electrocardiographic monitoring, and pulse oximetry.

A table outlining the emergency management of acute severe upper gastrointestinal bleeding is provided (table 1).

Other patients can be admitted to a regular medical ward, though we suggest that all admitted patients with the exception of low-risk patients receive electrocardiographic monitoring. Outpatient management may be appropriate for some low-risk patients. Determining the appropriate site of care for a patient can be facilitated using risk stratification scores, such as the Glasgow-Blatchford score. Use of these scores is recommended in the International Consensus Group guideline [1]. (See 'Risk stratification' below.)

General support — Patients should receive supplemental oxygen by nasal cannula and should receive nothing per mouth. Two large caliber (18 gauge or larger) peripheral intravenous catheters or a central venous line should be inserted. For patients who are hemodynamically unstable, two 16 gauge intravenous catheters and/or a large-bore, single-lumen central cordis should be placed.

Elective endotracheal intubation in patients with ongoing hematemesis or altered respiratory or mental status may facilitate endoscopy and decrease the risk of aspiration. However, among

patients who are critically ill, elective endotracheal intubation has been associated with worse outcomes. Our approach is to proceed with intubation in patients deemed high-risk for aspiration, including those with massive upper gastrointestinal (GI) bleeding or altered mental status.

A case control study with 200 patients with upper GI bleeding who were critically ill found that patients who had elective endotracheal intubation were more likely than patients who were not intubated to have adverse cardiopulmonary outcomes based on a composite outcome that included pneumonia, pulmonary edema, acute respiratory distress syndrome, and cardiac arrest [21]. Of note, the presence of respiratory distress prior to intubation was not reported and the mean Glasgow Coma Scale score was 14.7 (+/- 0.95), indicating that altered mental status was absent in the majority of patients. Patients who were electively intubated were more likely to suffer cardiopulmonary complications compared with patients who were not intubated (20.0 versus 6.0 percent). In particular, patients who were intubated were more likely to be diagnosed with pneumonia within 48 hours (14.0 versus 2.0 percent).

Fluid resuscitation — Adequate resuscitation and hemodynamic stabilization is essential prior to endoscopy to minimize treatment-associated complications [22]. Patients with active bleeding should receive intravenous fluids (eg, 500 mL of normal saline or lactated Ringer's solution over 30 minutes) while being typed and cross-matched for blood transfusion. The rate of fluid resuscitation will in part depend on whether the patient is hemodynamically unstable. Patients at risk of fluid overload may require intensive monitoring.

If the blood pressure fails to respond to initial resuscitation efforts, the rate of fluid administration should be increased. In some patients, temporary support with vasopressor drugs may be required. (See "Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock", section on 'Hemodynamic support' and "Treatment of severe hypovolemia or hypovolemic shock in adults", section on 'Initial rate of fluid repletion'.)

Blood product transfusions

Anemia

General approach — The decision to initiate blood transfusion must be individualized (algorithm 1). Our approach is to initiate blood transfusion if the hemoglobin is <7 g/dL (<70-g/L) [1,23-26]. For most patients, our goal is to maintain the hemoglobin at a level ≥7 g/dL (70 g/L), rather than at a higher level. However, for patients at increased risk of adverse events in the setting of significant anemia, such as those with coronary artery disease or in those with evidence of ongoing active bleeding, our goal is to maintain the hemoglobin at a level of ≥8 g/dL (80 g/L). We do not have an age cutoff for determining which patients should have a goal

hemoglobin of ≥8 g/dL (80 g/L), and instead base the decision on the patient's comorbid conditions. Hemoglobin thresholds for individuals with other features, such as acute coronary syndrome (ACS), are summarized in the table (table 2) and discussed separately. (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult", section on 'ACS (including MI)'.)

Using a restrictive transfusion strategy for most hemodynamically stable patients was supported by a meta-analysis of five randomized trials with a total of 1965 patients with acute upper gastrointestinal bleeding [19]. Patients assigned to a restrictive transfusion strategy had a lower all-cause mortality than those assigned to a liberal transfusion strategy (absolute risk reduction [ARR] 2.2 percent, relative risk [RR] 0.65, 95% CI 0.44-0.97) and rebleeding (ARR 4.4 percent, RR 0.58, 95% CI 0.40-0.84). While two of the studies used a cutoff of 7 g/dL (70 g/L) for a restrictive transfusion strategy and three used a cutoff of 8 g/dL (80 g/L), all of the studies favored a restrictive transfusion strategy. There were no differences between patients with cirrhosis and those with non-variceal bleeding. On subgroup analysis, there were non-statistically significant trends toward a higher risk of mortality with a restrictive transfusion strategy among patients with ischemic heart disease (RR 4.4, 95% CI 0.27-22) and a lower risk of mortality among patients without ischemic heart disease (RR 0.58, 95% CI 0.86-1.3). Rebleeding risk was similar between those with and without ischemic heart disease (RR 0.50 and 0.69, respectively).

The meta-analysis did not detect a difference between a restrictive and a liberal transfusion strategy in the risk of myocardial infarction (RR 0.79, 95% CI 0.33-1.89), stroke (RR 0.49, 95% CI 0.12-2.01), or acute kidney injury (RR 0.77, 0.56-1.05), but not all of the included trials reported these outcomes.

Suspected variceal bleeding — It is important to avoid overtransfusion in patients with suspected variceal bleeding. In patients with variceal bleeding, we transfuse once the hemoglobin is <7 g/dL (<70 g/L), with the goal of increasing the hemoglobin to ≥7 g/dL (70 g/L). We do not use a higher transfusion threshold (eg, <9 g/dL [90 g/L]), as transfusion can precipitate worsening of the bleeding [25,27]. (See "Overview of the management of patients with variceal bleeding", section on 'Resuscitation and support'.)

Active bleeding and hypovolemia — For patients with active/brisk bleeding and hypovolemia, decisions about transfusion are guided by hemodynamic parameters (eg, pulse and blood pressure), the pace of the bleeding, estimated blood loss, and the ability to stop the bleeding, rather than by serial hemoglobin measurements. Patients who require massive transfusion (defined by institutional protocols, often >3 units of RBCs in an hour or 10 units of RBCs in 24 hours) may also need replacement of coagulation factors and/or platelets. (See

"Indications and hemoglobin thresholds for red blood cell transfusion in the adult", section on 'Acute bleeding' and "Massive blood transfusion", section on 'Approach to volume and blood replacement'.)

Thrombocytopenia — Patients with critical or life-threatening bleeding and a low platelet count (<50,000/microL) should be transfused with platelets. Limited data suggest that proceeding with upper endoscopy in patients with thrombocytopenia is generally safe [28], though whether there is a lower limit below which endoscopy should be delayed is unclear [29]. Our approach is to perform an upper endoscopy if the platelet count is >20,000/microL, though if the patient is suspected to have active bleeding, we attempt to raise the platelet count to >50,000/microL prior to endoscopy.

In the past, platelet transfusions were considered in non-thrombocytopenic or mildly thrombocytopenic patients with life-threatening bleeding who had been taking antiplatelet agents such as aspirin or clopidogrel [30]. However, high-quality evidence regarding the benefit of platelet transfusion is lacking, and some evidence suggests that platelet transfusion may be deleterious [31]. Because these cases can be complex, an individualized approach based on the complete clinical picture is required. Potential adverse effects of platelet transfusion are discussed separately. (See "Platelet transfusion: Indications, ordering, and associated risks", section on 'Complications'.)

If the patient is taking antiplatelet medications because of a recent (less than one year) vascular stent placement or acute coronary syndrome, when possible, a cardiologist should be consulted prior to stopping the medications.

Managing anticoagulants, antiplatelet agents, and coagulopathies

Anticoagulants and antiplatelet agents — The approach to management of anticoagulants and antiplatelet agents depends on the medications being used and their indications, how severe the bleeding is, and how quickly reversal of anticoagulation is needed. The medications and products that may be used to reverse anticoagulation are discussed in the tables and separate topic reviews:

- Warfarin (table 3) (see "Management of warfarin-associated bleeding or supratherapeutic INR", section on 'Treatment of bleeding')
- Direct oral anticoagulants (DOACs) (table 4) (see "Management of bleeding in patients receiving direct oral anticoagulants", section on 'Major bleeding')
- Heparins (see "Heparin and LMW heparin: Dosing and adverse effects", section on 'Reversal')

For most patients, endoscopy should not be delayed because of anticoagulant or antiplatelet agent use [1]. Provided the patient is hemodynamically stable, urgent endoscopy can usually proceed simultaneously with management of antithrombotic medications. However, for patients undergoing upper endoscopy, we wait until the INR is <2.5 to perform the endoscopy, if possible [32]. This approach is based on data that suggest endoscopy is safe and endoscopic therapy effective in patients who are mildly to moderately anticoagulated [33].

When possible, anticoagulants and antiplatelet agents should be held in patients with acute upper GI bleeding. In patients with severe, ongoing bleeding who are taking an anticoagulant, administration of a reversal agent or intravenous prothrombin complex concentrate may be indicated. However, the thrombotic risk of reversing anticoagulation should be weighed against the risk of continued bleeding without reversal, and thus the decision to discontinue medications or administer reversal agents needs to be individualized.

For antiplatelet agents, the decision to discontinue may be straightforward (eg, stopping a nonsteroidal anti-inflammatory drug in a patient who is taking it for mild joint pain). However, in more complicated cases, consultation with the provider who prescribed the antiplatelet medication should be considered. (See "Management of anticoagulants in patients undergoing endoscopic procedures", section on 'Urgent procedures' and "Management of antiplatelet agents in patients undergoing endoscopic procedures" and "Gastrointestinal endoscopy in patients with disorders of hemostasis".)

When to resume these medications once hemostasis has been achieved will depend on the patient's risks for thrombosis and recurrent bleeding. (See "Management of anticoagulants in patients undergoing endoscopic procedures", section on 'Resuming anticoagulants after hemostasis' and "Overview of the treatment of bleeding peptic ulcers", section on 'Risk factors for persistent or recurrent bleeding'.)

Coagulopathies related to cirrhosis — The management of coagulopathies in patients with cirrhosis is particularly complicated. In patients with cirrhosis, the INR is not an accurate measure of coagulation because it only reflects changes in procoagulant factors, and both procoagulant and anticoagulant factors are reduced. These issues are discussed separately. (See "Hemostatic abnormalities in patients with liver disease", section on 'Bleeding'.)

Other bleeding disorders — For individuals with other bleeding disorders, consultation with hematology (the patient's primary hematologist if possible) is prudent. They can advise regarding specific products, dosing, and monitoring based on the individual's specific disorder and medical history.

Dilutional coagulopathy — Patients who require massive transfusion (defined by institutional protocols, often >3 units RBCs in an hour or 10 units RBCs in 24 hours) may also need replacement of coagulation factors and/or platelets. (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult", section on 'Acute bleeding' and "Massive blood transfusion", section on 'Approach to volume and blood replacement'.)

Medications

Acid suppression — Patients admitted to the hospital with acute upper GI bleeding are typically treated with a proton pump inhibitor (PPI). The optimal approach to PPI administration prior to endoscopy is unclear. Options include giving an IV PPI every 12 hours or starting a continuous infusion. Our approach is to give a high-dose bolus (eg, esomeprazole 80 mg) to patients with signs of active bleeding (eg, hematemesis, hemodynamic instability).

Typically, we try to perform endoscopy on patients with suspected ongoing active bleeding after resuscitation within 12 hours. If endoscopy is performed after 12 hours, a second dose of an IV PPI should be given 12 hours later (eg, esomeprazole 40 mg). For patients who may have stopped bleeding (eg, patients who are hemodynamically stable with melena), we give an IV PPI every 12 hours (eg, esomeprazole 40 mg). Subsequent dosing will then depend on the endoscopic findings. Oral formulations (eg, esomeprazole 40 mg orally twice daily) are a reasonable alternative if IV formulations are not available. Pantoprazole and esomeprazole are the only intravenous formulations available in the United States, and intravenous lansoprazole has been removed from the world market. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Acid suppression'.)

Several studies have examined the role of acid suppression given before or after endoscopy (with or without therapeutic intervention) [34-45]. In the setting of active upper GI bleeding from an ulcer, acid suppressive therapy with H2 receptor antagonists has **not** been shown to significantly lower the rate of ulcer rebleeding [39,42,46]. By contrast, high dose antisecretory therapy with an intravenous infusion of a PPI significantly reduces the rate of rebleeding compared with standard treatment in patients with bleeding ulcers [47]. Oral and intravenous PPI therapy also decrease the length of hospital stay, rebleeding rate, and need for blood transfusion in patients with high-risk ulcers treated with endoscopic therapy. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Acid suppression'.)

PPIs may also promote hemostasis in patients with lesions other than ulcers. This likely occurs because neutralization of gastric acid leads to the stabilization of blood clots [45].

Prokinetics — Both erythromycin and metoclopramide have been studied in patients with acute upper GI bleeding. The goal of using a prokinetic agent is to improve gastric visualization

at the time of endoscopy by clearing the stomach of blood, clots, and food residue. We suggest that erythromycin be used before endoscopy. A reasonable dose is 250 mg intravenously over 20 to 30 minutes. Endoscopy is performed 20 to 90 minutes following completion of the erythromycin infusion. Patients receiving erythromycin need to be monitored for QTc prolongation. In addition, drug-drug interactions should be evaluated before giving erythromycin because it is a cytochrome P450 3A inhibitor (table 5).

Erythromycin promotes gastric emptying based upon its ability to be an agonist of motilin receptors. Using erythromycin to improve gastric visualization has been studied in several randomized controlled trials and meta-analyses [48-56]. The randomized trials were included in a 2016 meta-analysis that examined the role of pre-endoscopic erythromycin [55]. The meta-analysis included eight randomized trials with 598 patients with upper gastrointestinal bleeding and compared patients who received erythromycin with those who did not. Patients who received erythromycin were more likely to have adequate gastric visualization (77 versus 51 percent, odds ratio [OR] 4.14; 95% CI 2.01-8.53), were less likely to require second-look endoscopy (15 versus 26 percent, OR 0.51; 95% CI 0.34-0.77), and had shorter hospital stays (mean difference -1.75 days, 95% CI -2.43 to -1.06). There were no differences in units of blood transfused, endoscopy duration, or need for emergent surgery between those who received erythromycin and those who did not. A second meta-analysis also found better visualization with the use of erythromycin [56]. In two trials with 195 patients, patients who received erythromycin scored higher than patients who received placebo on a 16-point ordinal scale, with higher scores indicating better visualization (mean difference 3.63 points, 95% CI 2.20-5.05).

Some trials have compared pre-endoscopy erythromycin with nasogastric lavage. In one trial, 253 patients were assigned to receive erythromycin alone, nasogastric lavage alone, or nasogastric lavage plus erythromycin. It found that the quality of visualization did not differ significantly among the three groups [52]. In addition, there were no differences among the groups with regard to procedure duration, rebleeding rates, need for second endoscopy, number of transfused units of blood, and mortality. A meta-analysis also failed to show a significant difference between erythromycin and nasogastric lavage [56]. (See 'Nasogastric lavage' above.)

Vasoactive medications — Somatostatin, its analog octreotide, and terlipressin are used in the treatment of variceal bleeding and may also reduce the risk of bleeding due to nonvariceal causes. In patients with suspected variceal bleeding, octreotide is given as an intravenous bolus of 50 mcg, followed by a continuous infusion at a rate of 50 mcg per hour. (See "Methods to

achieve hemostasis in patients with acute variceal hemorrhage", section on 'Somatostatin and its analogs'.)

Octreotide is not recommended for routine use in patients with acute nonvariceal upper GI bleeding, but it can be used as adjunctive therapy in some cases. Its role is generally limited to settings in which endoscopy is unavailable or as a means to help stabilize patients before definitive therapy can be performed. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Somatostatin and octreotide'.)

Antibiotics for patients with cirrhosis — Bacterial infections are present in up to 20 percent of patients with cirrhosis who are hospitalized with gastrointestinal bleeding; up to an additional 50 percent develop an infection while hospitalized. Such patients have increased mortality.

Multiple trials evaluating the effectiveness of prophylactic antibiotics in cirrhotic patients hospitalized for GI bleeding suggest an overall reduction in infectious complications and possibly decreased mortality. Antibiotics may also reduce the risk of recurrent bleeding in hospitalized patients who bled from esophageal varices. A reasonable conclusion from these data is that patients with cirrhosis who present with acute upper GI bleeding (from varices or other causes) should be given prophylactic antibiotics, preferably before endoscopy (although effectiveness has also been demonstrated when given after endoscopy). (See "Overview of the management of patients with variceal bleeding".)

Ineffective treatments — Tranexamic acid is an antifibrinolytic agent that has been studied in patients with upper GI bleeding and does not appear to be beneficial [57,58]. A meta-analysis that included eight randomized trials of tranexamic acid in patients with upper GI bleeding found a benefit with regard to mortality but not with regard to bleeding, surgery, or transfusion requirements [57]. However, when only studies that used antiulcer drugs and/or endoscopic therapy were included, there was no beneficial effect.

In a subsequent randomized trial with 12,009 patients with GI bleeding (most of whom had evidence of upper GI bleeding), use of tranexamic acid did not reduce the risk of death due to bleeding within five days (4 percent with tranexamic acid, 4 percent with placebo) [58]. Tranexamic acid use was associated with an increase in venous thromboembolic events (deep vein thrombosis, pulmonary embolism) and seizures compared with placebo (relative risks of 1.85 and 1.73, respectively).

The randomized trial and meta-analysis suggest that there is no role for tranexamic acid in the treatment of upper GI bleeding.

Consultations

- **Gastroenterology** – Gastroenterological consultation should be obtained in all patients with suspected clinically significant acute upper GI bleeding.
- **Transfusion medicine (if available)** – The transfusion medicine service or clinical pathology/blood bank physician should be alerted if the patient may require a massive transfusion protocol so they can ensure that adequate blood products are available.
- **Hematology** – Hematology consultation can help if emergency reversal of anticoagulation is indicated and/or if abnormal clotting tests are unexplained.
- **Surgery/interventional radiology** – The decision to obtain surgical and interventional radiology consultations prior to endoscopy should be based upon the likelihood of persistent or recurrent bleeding, or risks/complications stemming from endoscopic therapy (perforation, precipitation of massive bleeding).
- **Clinician who prescribed an anticoagulant or antiplatelet agent (if relevant)** – If an anticoagulant or antiplatelet agent needs to be discontinued and/or reversed due to serious or life-threatening bleeding, the clinician who prescribed the medication should be contacted to discuss plans for resuming it.

As a general rule, we obtain surgical and interventional radiology consultation if endoscopic therapy is unlikely to be successful, if the patient is deemed to be at high risk for rebleeding or complications associated with endoscopy, or if there is concern that the patient may have an aorto-enteric fistula. In addition, a surgeon and an interventional radiologist should be promptly notified of all patients with severe acute upper GI bleeding, such as those presenting with hemodynamic instability that fails to respond to resuscitation [59].

DIAGNOSTIC STUDIES

An algorithm providing an overview of the diagnostic approach to patients with suspected upper gastrointestinal bleeding is provided (algorithm 2).

Upper endoscopy — Upper endoscopy is the diagnostic modality of choice for acute upper GI bleeding (algorithm 2) [60,61]. Endoscopy has a high sensitivity and specificity for locating and identifying bleeding lesions in the upper GI tract. In addition, once a bleeding lesion has been identified, therapeutic endoscopy can achieve acute hemostasis and prevent recurrent bleeding in most patients. Early endoscopy (within 24 hours) is recommended for most patients with acute upper GI bleeding. For patients with suspected variceal bleeding, we perform

endoscopy within 12 hours of presentation. (See 'Early endoscopy' below and "Methods to achieve hemostasis in patients with acute variceal hemorrhage", section on 'Initial management' and "Overview of the treatment of bleeding peptic ulcers", section on 'Endoscopic therapy' and "Methods to achieve hemostasis in patients with acute variceal hemorrhage", section on 'Management of esophageal varices'.)

Endoscopic findings in patients with bleeding peptic ulcers are described using the modified Forrest classification [62]. Findings include spurting hemorrhage (class Ia) (picture 1), oozing hemorrhage (class Ib), a nonbleeding visible vessel (class IIa) (picture 2), an adherent clot (class IIb) (picture 3), a flat pigmented spot (class IIc) (picture 4), and a clean ulcer base (class III) (picture 4). The endoscopic appearance helps determine which lesions require endoscopic therapy. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Endoscopic therapy'.)

It may be helpful to give a prokinetic agent such as erythromycin or to irrigate the stomach prior to endoscopy to help remove residual blood and other gastric contents. However, despite prokinetic administration or irrigation, the stomach can be obscured with blood, potentially making it difficult to establish a clear diagnosis and/or perform therapeutic maneuvers. In patients in whom blood obscures the source of bleeding, a second endoscopy may be required to establish a diagnosis and to potentially apply therapy, but routine second-look endoscopy is not recommended. (See 'Nasogastric lavage' above and "Overview of the treatment of bleeding peptic ulcers", section on 'Second-look endoscopy'.)

Early endoscopy — Our approach is to perform upper endoscopy within 24 hours for most patients with upper GI bleeding, but only after adequate resuscitation has been provided. For patients with suspected variceal bleeding, we perform endoscopy within 12 hours of presentation.

Studies have reached variable conclusions when determining whether the application of early endoscopy (typically within 24 hours) for risk stratification and treatment reduces resource utilization or affects patient outcomes [63-74]. Some studies have demonstrated reduced resource utilization and improved outcomes from early endoscopy [67,68,70,72-74], while other studies, including a randomized trial, did not [64,65,69].

Retrospective studies have suggested that emergency endoscopy (within 12 hours) may be associated with poor outcomes [69,71], possibly due to inadequate resuscitation in patients undergoing emergency endoscopy. However, a randomized trial with 516 patients with upper GI bleeding who were at increased risk for death or further bleeding (Glasgow-Blatchford score ≥12) did not find a difference in outcomes between those who underwent "urgent" endoscopy

(within six hours of gastroenterology consultation) and "early" endoscopy (between 6 and 24 hours after gastroenterology consultation), though there was a trend toward worse outcomes in the urgent endoscopy group [63]. Outcomes examined in the study included 30-day mortality (8.9 versus 6.6 percent with urgent and early endoscopy, respectively; hazard ratio 1.35; 95% CI 0.72 to 2.54) and further bleeding within 30 days (10.9 versus 7.8 percent; hazard ratio 1.45; 95% CI 0.83 to 2.58). Of note, because there was a lag between presentation and gastroenterology consultation, the patients in the urgent endoscopy group underwent endoscopy a mean of 10 hours after presentation and those in the early endoscopy group underwent endoscopy a mean of 25 hours after presentation (with 55 percent undergoing endoscopy >24 hours after presentation). In addition, patients with hemodynamic instability who could not be stabilized were excluded from the study, so the results may not apply to this group of patients.

Risks of endoscopy — Risks of upper endoscopy include pulmonary aspiration, adverse reactions to medications used to achieve conscious sedation, GI perforation, and increasing bleeding while attempting therapeutic intervention.

While patients need to be hemodynamically stable prior to undergoing endoscopy, data suggest that patients do not need to have a normal hematocrit in order to safely undergo endoscopy [75]. In addition, endoscopy appears to be safe in patients who are mildly to moderately anticoagulated [33]. In a retrospective study of 920 patients with upper GI bleeding undergoing upper endoscopy, patients with low hematocrits (<30 percent) were similar to those with high hematocrits (>30 percent) with regard to cardiovascular complications and mortality [75]. In another retrospective study with 233 patients with upper GI bleeding who received endoscopic therapy, an elevated INR was not associated with an increased risk of rebleeding, transfusion requirement, surgery, length of stay, or mortality [33]. The INR was between 1.3 and 2.7 in 95 percent of the patients, so the results of the study may only apply to patients who are mildly to moderately anticoagulated.

The risks versus benefits of upper endoscopy should be considered in high-risk patients, such as those who have had a recent myocardial infarction. In one study, for example, 200 patients who underwent endoscopy within 30 days after myocardial infarction (MI) were compared with 200 controls matched for age, sex, and endoscopic indication [76]. Complications (including fatal ventricular tachycardia, near respiratory arrest, and mild hypotension) occurred more often in patients who had a recent MI (8 versus 2 percent). Complications occurred more often (21 versus 2 percent) in patients who were very ill (Apache II score >16 or hypotension prior to endoscopy). However, such patients are at increased risk for complications even without endoscopy and may be particularly vulnerable to complications from continued bleeding without endoscopy. (See "Predictive scoring systems in the intensive care unit".)

Other diagnostic tests — Other diagnostic tests for acute upper GI bleeding include CT angiography (CTA using an overt GI bleeding protocol) and angiography, which can detect active bleeding [77,78], deep small bowel enteroscopy, and rarely, intraoperative enteroscopy (algorithm 2). Upper GI barium studies are **contraindicated** in the setting of acute upper GI bleeding because they will interfere with subsequent endoscopy, angiography, or surgery [60]. There is also interest in using wireless capsule endoscopy for patients who have presented to the emergency department with suspected upper GI bleeding. An esophageal capsule (which has a recording time of 20 minutes) can be given in the emergency department and reviewed immediately for evidence of bleeding. Confirming the presence of blood in the stomach or duodenum may aid with patient triage and identify patients more likely to benefit from early endoscopy [79-83]. Small bowel capsule endoscopy has also been employed to help localize bleeding in patients with acute gastrointestinal bleeding without hematemesis. (See "Angiographic control of nonvariceal gastrointestinal bleeding in adults" and "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)" and "Wireless video capsule endoscopy", section on 'Esophageal capsule endoscopy' and "Wireless video capsule endoscopy", section on 'Indications' and "Wireless video capsule endoscopy", section on 'Acute gastrointestinal bleeding'.)

A colonoscopy is generally required for patients with hematochezia and a negative upper endoscopy unless an alternative source for the bleeding has been identified. In addition, patients with melena and a negative upper endoscopy frequently undergo colonoscopy to rule out a colonic source for the bleeding, as right-sided lesions may present with melena. In a study that included 1743 colonoscopies performed for the evaluation of melena following a non-diagnostic upper endoscopy, a suspected bleeding source was identified in 5 percent of patients, a rate that was higher than that seen in 194,979 average-risk screening controls (1 percent) [84]. Despite the relatively low yield in patients with melena, we routinely perform a colonoscopy in patients with melena and a negative upper endoscopy, as well as in patients with hematochezia. (See "Approach to acute lower gastrointestinal bleeding in adults", section on 'Colonoscopy'.)

RISK STRATIFICATION

Endoscopic, clinical, and laboratory features may be useful for risk stratification of patients who present with acute upper GI bleeding (table 6 and picture 1 and picture 2 and picture 3 and picture 4) [85-94], and the use of risk stratification tools is recommended by the International Consensus Group [1]. Factors associated with rebleeding identified in a meta-analysis included [95]:

- Hemodynamic instability (systolic blood pressure less than 100 mmHg, heart rate greater than 100 beats per minute)
- Hemoglobin less than 10 g/L
- Active bleeding at the time of endoscopy
- Large ulcer size (greater than 1 to 3 cm in various studies)
- Ulcer location (posterior duodenal bulb or high lesser gastric curvature)

An increase in the blood urea nitrogen (BUN) level at 24 hours compared with baseline may be another predictor of poor outcomes. A study of 357 patients with acute nonvariceal upper GI bleeding found that an increase in the BUN at 24 hours was a predictor of a composite outcome that included rebleeding and mortality [96]. The authors speculate that the association of poor outcomes with an increase in BUN may be the result of inadequate resuscitation.

Several investigators have developed decision rules and predictive models that permit identification of patients who are at low risk for recurrent or life-threatening hemorrhage [97]. Such patients may be suitable for early hospital discharge or even outpatient care. The effectiveness of such rules has been evaluated in a variety of clinical settings, with most studies suggesting that patients deemed to be low-risk can safely be discharged early or treated as outpatients [65,85-91,97-104]. In addition, this approach is associated with reduced resource utilization compared with universal hospitalization of patients with acute upper GI bleeding.

Risk scores — Two commonly cited scoring systems are the Rockall score and the Blatchford score. The International Consensus Group suggests using a Glasgow Blatchford score (GBS) of ≤1 to identify patients who are very low risk for rebleeding or mortality and who can be considered for outpatient management [1].

- The Rockall score which is calculated after endoscopy is based upon age, the presence of shock, comorbidity, diagnosis, and endoscopic stigmata of recent hemorrhage (calculator 1) [85]. In one validation study, only 32 of 744 patients (4 percent) who scored 2 or less (out of a maximum of 11) rebled and only one died.

  On the other hand, in a later study of 247 patients who underwent endoscopic therapy for bleeding peptic ulcers, the model performed poorly when predicting recurrent bleeding, underscoring the need for validation of this model [105].

- The GBS, unlike the Rockall score, does not take endoscopic data into account and thus can be calculated when the patient first presents (calculator 2) [90]. The score is based upon the blood urea nitrogen, hemoglobin, systolic blood pressure, pulse, and the presence of melena, syncope, hepatic disease, and/or cardiac failure. The score ranges from zero to 23 and the risk of requiring endoscopic intervention increases with increasing

score. One meta-analysis found that a Blatchford score of zero was associated with a low likelihood of the need for urgent endoscopic intervention (likelihood ratio 0.02, 95% confidence interval [CI] 0-0.05) [6]. A second study with 3012 patients found that a score ≤1 could be used to identify a low-risk cohort [106].

A simpler version of the score, known as the modified GBS, is calculated using only the blood urea nitrogen, hemoglobin, systolic blood pressure, and pulse. The score ranges from 0 to 16. A prospective study of the modified score found that it performed as well as the full GBS and that it outperformed the Rockall score with regard to predicting the need for clinical intervention, rebleeding, and mortality [107].

AIMS65 is another scoring system that uses data available prior to endoscopy (serum albumin, INR, presence of altered mental status, systolic blood pressure, and age), but it is less sensitive than the Blatchford and preendoscopic Rockall scores for identifying low-risk patients [106,108]. A newer score, the Age, Blood tests and Comorbidities score, was developed to predict mortality in patients with upper GI bleeding and lower GI bleeding [109]. Initial data from the validation cohort, which included 4019 patients with upper GI bleeding and 2336 patients with lower GI bleeding suggests good performance for the score (AUROC 0.81 to 0.84).

Implementation — The data presented above suggest that risk stratification is feasible and permits identification of patients who can be managed safely without hospitalization. However, for these systems to be successful, the risk stratification system must be tied directly to decisions regarding patient discharge. None of the published risk scores has yet been adopted widely.

As a general rule, we discharge patients following endoscopy if they have a likely bleeding source identified on upper endoscopy that is not associated with a high risk of rebleeding provided they:

- Have no comorbidities
- Have stable vital signs
- Have a normal hemoglobin level

High-risk bleeding sources include variceal bleeding, active bleeding, bleeding from a Dieulafoy's lesion, or an ulcer bleeding with high-risk stigmata (table 6).

Prior to endoscopy, we will discharge patients with a GBS of 0-1 provided they have no significant comorbidities, have stable vital signs, have a normal hemoglobin level, do not live far from medical care, and have a mechanism for prompt outpatient gastroenterology

evaluation/endoscopy (ie, within three days). We discharge the patient on a proton pump inhibitor (eg, omeprazole or esomeprazole 20 mg once daily, pantoprazole 40 mg once daily).

Ultimately, the decision to discharge the patient also depends on individual-patient factors, such as reliability for follow-up and confidence of the endoscopists in the diagnosis.

If patients do not meet the above criteria we admit them to a monitored setting or intensive care unit (depending upon the severity of bleeding, comorbidities, and stability of vital signs). Most patients who have received endoscopic treatment for high-risk stigmata should be hospitalized for 72 hours to monitor for rebleeding, since most rebleeding occurs during this time [110].

TREATMENT

The treatment of patients with upper GI bleeding is discussed separately:

- (See "Overview of the treatment of bleeding peptic ulcers".)
- (See "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)
- (See "Angiodysplasia of the gastrointestinal tract", section on 'Treatment'.)
- (See "Portal hypertensive gastropathy", section on 'Management'.)
- (See "Causes of upper gastrointestinal bleeding in adults", section on 'Vascular lesions'.)
- (See "Causes of upper gastrointestinal bleeding in adults", section on 'Aortoenteric fistulas'.)
- (See "Causes of upper gastrointestinal bleeding in adults", section on 'Upper gastrointestinal tumors'.)
- (See "Argon plasma coagulation in the management of gastrointestinal hemorrhage".)
- (See "Angiographic control of nonvariceal gastrointestinal bleeding in adults".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Gastrointestinal bleeding in adults".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Upper endoscopy (The Basics)" and "Patient education: GI bleed (The Basics)")
- Beyond the Basics topics (see "Patient education: Upper endoscopy (Beyond the Basics)" and "Patient education: Peptic ulcer disease (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Rapid overview** – A table outlining the emergency management of acute severe upper gastrointestinal bleeding is provided (table 1). (See 'Introduction' above.)
- **History and examination** – The history and physical examination can help identify potential sources of the upper GI bleed, assess the severity of the bleed, and identify comorbid conditions that may influence the patient's subsequent management. (See 'Initial evaluation' above.)

  The presence of abdominal pain, especially if severe and associated with rebound tenderness or involuntary guarding, raises concern for perforation. If any signs of an acute abdomen are present, further evaluation to exclude a perforation is required prior to endoscopy. (See 'Physical examination' above.)
- **Laboratory testing** – Laboratory tests that should be obtained in patients with acute upper GI bleeding include a complete blood count, serum chemistries, liver tests, and coagulation studies. In addition, we suggest ruling out a myocardial infarction in older adult patients and those with known cardiovascular disease who have severe bleeding, especially if there has been hemodynamic instability. (See 'Laboratory data' above.)

- **Risk stratification and triage –** We suggest incorporation of a validated risk score for upper gastrointestinal bleeding into routine clinical practice to facilitate optimal triage decisions. Patients with a Glasgow Blatchford Score (GBS) of 0-1 may be considered for out-patient management. Others should be admitted to a monitored bed or intensive care unit depending upon the severity of bleeding. (See 'Risk scores' above and 'Triage' above.)

- **Pre-endoscopic management** – Prior to endoscopy, patients should receive general supportive measures, including:

  - Provision of supplemental oxygen by nasal cannula
  - Nothing per mouth
  - Two large caliber (18 gauge or larger) peripheral catheters or a central venous line

  Nasogastric lavage is **NOT** a routine part of the management of acute upper GI bleeding. (See 'Nasogastric lavage' above.)

- **Pharmacologic therapy** – Medications that should be started prior to endoscopy include a proton pump inhibitor, erythromycin, antibiotics (for patients with cirrhosis), and somatostatin or one of its analogs (for patients with suspected variceal bleeding).

  - We suggest that patients admitted to the hospital with acute upper GI bleeding receive an IV proton pump inhibitor (PPI) (**Grade 2B**). The optimal approach to PPI administration prior to endoscopy is unclear. Our approach is to give a high-dose bolus (eg, esomeprazole 80 mg) to patients with signs of active bleeding (eg, hematemesis, hemodynamic instability). If endoscopy is delayed beyond 12 hours, a second dose of an IV PPI should be given (eg, esomeprazole 40 mg). For patients who may have stopped bleeding (eg, patients who are hemodynamically stable with melena), we give an IV PPI every 12 hours (eg, esomeprazole 40 mg). Subsequent dosing will then depend on the endoscopic findings. (See 'Acid suppression' above and "Overview of the treatment of bleeding peptic ulcers", section on 'Acid suppression'.)

    We suggest that erythromycin be given prior to endoscopy to help improve visualization (**Grade 2C**). A reasonable dose is 250 mg intravenously over 20 to 30 minutes. Endoscopy is performed 20 to 90 minutes following completion of the erythromycin infusion. Patients receiving erythromycin need to be monitored for QTc prolongation. In addition, drug-drug interactions should be evaluated before giving erythromycin because it is a cytochrome P450 3A inhibitor (table 5). (See 'Prokinetics' above.)

- Patients with cirrhosis who present with acute upper GI bleeding (from varices or other causes) should be given prophylactic antibiotics.

- Somatostatin, its analog octreotide, or terlipressin should be started if variceal bleeding is suspected.

- **Blood transfusion** – Transfusion is guided by the hemoglobin level and the presence of comorbid conditions:

  - The decision to initiate blood transfusion must be individualized (algorithm 1 and table 2). Our approach is to initiate blood transfusion if the hemoglobin is <7 g/dL (70 g/L) for most patients. Higher transfusion thresholds may be indicated for patients at increased risk of adverse events in the setting of significant anemia or those with coronary artery disease. (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult", section on 'Overview of our approach'.)

  - It is important to avoid overtransfusion in patients with suspected variceal bleeding because transfusion can precipitate worsening of the bleeding; the blood transfusion goal for variceal bleeding is <7 g/dL (70 g/L). (See "Overview of the management of patients with variceal bleeding", section on 'Blood products'.)

  - For patients with active/brisk bleeding and hypovolemia, decisions about transfusion are guided by hemodynamic parameters (eg, pulse and blood pressure), the pace of the bleeding, estimated blood loss, and the ability to stop the bleeding, rather than by serial hemoglobin measurements. (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult", section on 'Acute bleeding' and "Massive blood transfusion", section on 'Approach to volume and blood replacement'.)

- **Anticoagulants, antiplatelet agents, and coagulopathies** – The approach to management of anticoagulants and antiplatelet agents depends on the specific medications being used, the reason they are being used, and how quickly reversal of anticoagulation is needed. Management of coagulopathy depends on the underlying etiology. Hematology input may be advisable. (See 'Managing anticoagulants, antiplatelet agents, and coagulopathies' above and 'Consultations' above.)

- **Endoscopy** – We recommend upper endoscopy within 24 hours for the evaluation and management of patients admitted with acute upper GI bleeding. For patients with suspected variceal bleeding, we perform endoscopy within 12 hours of presentation.

Additional diagnostic tests may be required for some patients. An algorithm providing an overview of the diagnostic approach to patients with suspected upper gastrointestinal bleeding is provided (algorithm 2).

- **Treatment of the underlying lesion** – The approaches to achieve hemostasis for variceal bleeding and bleeding peptic ulcers are discussed separately.

REFERENCES

1. Barkun AN, Almadi M, Kuipers EJ, et al. Management of Nonvariceal Upper Gastrointestinal Bleeding: Guideline Recommendations From the International Consensus Group. Ann Intern Med 2019; 171:805.
2. Hwang JH, Fisher DA, Ben-Menachem T, et al. The role of endoscopy in the management of acute non-variceal upper GI bleeding. Gastrointest Endosc 2012; 75:1132.
3. Laine L, Barkun AN, Saltzman JR, et al. ACG Clinical Guideline: Upper Gastrointestinal and Ulcer Bleeding. Am J Gastroenterol 2021; 116:899.
4. Gralnek IM, Dumonceau JM, Kuipers EJ, et al. Diagnosis and management of nonvariceal upper gastrointestinal hemorrhage: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. Endoscopy 2015; 47:a1.
5. Gralnek IM, Stanley AJ, Morris AJ, et al. Endoscopic diagnosis and management of nonvariceal upper gastrointestinal hemorrhage (NVUGIH): European Society of Gastrointestinal Endoscopy (ESGE) Guideline - Update 2021. Endoscopy 2021; 53:300.
6. Srygley FD, Gerardo CJ, Tran T, Fisher DA. Does this patient have a severe upper gastrointestinal bleed? JAMA 2012; 307:1072.
7. Cappell MS, Friedel D. Initial management of acute upper gastrointestinal bleeding: from initial evaluation up to gastrointestinal endoscopy. Med Clin North Am 2008; 92:491.
8. Current diagnosis and treatment: Surgery, 13, Doherty G (Ed), McGraw-Hill Companies, 201 0. p.493.
9. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. Gastroenterology 1988; 95:1569.
10. Palmer ED. The vigorous diagnostic approach to upper-gastrointestinal tract hemorrhage. A 23-year prospective study of 1,4000 patients. JAMA 1969; 207:1477.
11. Richards RJ, Donica MB, Grayer D. Can the blood urea nitrogen/creatinine ratio distinguish

upper from lower gastrointestinal bleeding? J Clin Gastroenterol 1990; 12:500.

12. Mortensen PB, Nøhr M, Møller-Petersen JF, Balslev I. The diagnostic value of serum urea/creatinine ratio in distinguishing between upper and lower gastrointestinal bleeding. A prospective study. Dan Med Bull 1994; 41:237.
13. Ernst AA, Haynes ML, Nick TG, Weiss SJ. Usefulness of the blood urea nitrogen/creatinine ratio in gastrointestinal bleeding. Am J Emerg Med 1999; 17:70.
14. Pallin DJ, Saltzman JR. Is nasogastric tube lavage in patients with acute upper GI bleeding indicated or antiquated? Gastrointest Endosc 2011; 74:981.
15. Karakonstantis S, Tzagkarakis E, Kalemaki D, et al. Nasogastric aspiration/lavage in patients with gastrointestinal bleeding: a review of the evidence. Expert Rev Gastroenterol Hepatol 2018; 12:63.
16. Huang ES, Karsan S, Kanwal F, et al. Impact of nasogastric lavage on outcomes in acute GI bleeding. Gastrointest Endosc 2011; 74:971.
17. Rockey DC, Ahn C, de Melo SW Jr. Randomized pragmatic trial of nasogastric tube placement in patients with upper gastrointestinal tract bleeding. J Investig Med 2017; 65:759.
18. Aljebreen AM, Fallone CA, Barkun AN. Nasogastric aspirate predicts high-risk endoscopic lesions in patients with acute upper-GI bleeding. Gastrointest Endosc 2004; 59:172.
19. Odutayo A, Desborough MJ, Trivella M, et al. Restrictive versus liberal blood transfusion for gastrointestinal bleeding: a systematic review and meta-analysis of randomised controlled trials. Lancet Gastroenterol Hepatol 2017; 2:354.
20. Carson JL, Guyatt G, Heddle NM, et al. Clinical Practice Guidelines From the AABB: Red Blood Cell Transfusion Thresholds and Storage. JAMA 2016; 316:2025.
21. Hayat U, Lee PJ, Ullah H, et al. Association of prophylactic endotracheal intubation in critically ill patients with upper GI bleeding and cardiopulmonary unplanned events. Gastrointest Endosc 2017; 86:500.
22. Baradarian R, Ramdhaney S, Chapalamadugu R, et al. Early intensive resuscitation of patients with upper gastrointestinal bleeding decreases mortality. Am J Gastroenterol 2004; 99:619.
23. Laine L, Jensen DM. Management of patients with ulcer bleeding. Am J Gastroenterol 2012; 107:345.
24. Duggan JM. Gastrointestinal hemorrhage: should we transfuse less? Dig Dis Sci 2009; 54:1662.

25. Villanueva C, Colomo A, Bosch A, et al. Transfusion strategies for acute upper gastrointestinal bleeding. N Engl J Med 2013; 368:11.
26. Qaseem A, Humphrey LL, Fitterman N, et al. Treatment of anemia in patients with heart disease: a clinical practice guideline from the American College of Physicians. Ann Intern Med 2013; 159:770.
27. McCormick PA, Jenkins SA, McIntyre N, Burroughs AK. Why portal hypertensive varices bleed and bleed: a hypothesis. Gut 1995; 36:100.
28. Ramos GP, Binder M, Hampel P, et al. Outcomes of endoscopic intervention for overt GI bleeding in severe thrombocytopenia. Gastrointest Endosc 2018; 88:55.
29. Laine L. Treatment of thrombocytopenic patients with GI bleeding. Gastrointest Endosc 2018; 88:62.
30. ASGE Standards of Practice Committee, Anderson MA, Ben-Menachem T, et al. Management of antithrombotic agents for endoscopic procedures. Gastrointest Endosc 2009; 70:1060.
31. Zakko L, Rustagi T, Douglas M, Laine L. No Benefit From Platelet Transfusion for Gastrointestinal Bleeding in Patients Taking Antiplatelet Agents. Clin Gastroenterol Hepatol 2017; 15:46.
32. ASGE Standards of Practice Committee, Acosta RD, Abraham NS, et al. The management of antithrombotic agents for patients undergoing GI endoscopy. Gastrointest Endosc 2016; 83:3.
33. Wolf AT, Wasan SK, Saltzman JR. Impact of anticoagulation on rebleeding following endoscopic therapy for nonvariceal upper gastrointestinal hemorrhage. Am J Gastroenterol 2007; 102:290.
34. Kanno T, Yuan Y, Tse F, et al. Proton pump inhibitor treatment initiated prior to endoscopic diagnosis in upper gastrointestinal bleeding. Cochrane Database Syst Rev 2022; 1:CD005415.
35. Leontiadis GI, Sharma VK, Howden CW. Systematic review and meta-analysis of proton pump inhibitor therapy in peptic ulcer bleeding. BMJ 2005; 330:568.
36. Aoki T. Intravenous administration of lansoprazole: a preliminary study of dose ranging and efficacy in upper gastrointestinal bleeding. Aliment Pharmacol Ther 1995; 9 Suppl 1:51.
37. Lin HJ, Lo WC, Lee FY, et al. A prospective randomized comparative trial showing that omeprazole prevents rebleeding in patients with bleeding peptic ulcer after successful endoscopic therapy. Arch Intern Med 1998; 158:54.

38. Khuroo MS, Yattoo GN, Javid G, et al. A comparison of omeprazole and placebo for bleeding peptic ulcer. N Engl J Med 1997; 336:1054.

39. Lau JY, Sung JJ, Lee KK, et al. Effect of intravenous omeprazole on recurrent bleeding after endoscopic treatment of bleeding peptic ulcers. N Engl J Med 2000; 343:310.

40. Schaffalitzky de Muckadell OB, Havelund T, Harling H, et al. Effect of omeprazole on the outcome of endoscopically treated bleeding peptic ulcers. Randomized double-blind placebo-controlled multicentre study. Scand J Gastroenterol 1997; 32:320.

41. Hasselgren G, Lind T, Lundell L, et al. Continuous intravenous infusion of omeprazole in elderly patients with peptic ulcer bleeding. Results of a placebo-controlled multicenter study. Scand J Gastroenterol 1997; 32:328.

42. Gisbert JP, González L, Calvet X, et al. Proton pump inhibitors versus H2-antagonists: a meta-analysis of their efficacy in treating bleeding peptic ulcer. Aliment Pharmacol Ther 2001; 15:917.

43. Sung JJ, Chan FK, Lau JY, et al. The effect of endoscopic therapy in patients receiving omeprazole for bleeding ulcers with nonbleeding visible vessels or adherent clots: a randomized comparison. Ann Intern Med 2003; 139:237.

44. Lau JY, Leung WK, Wu JC, et al. Omeprazole before endoscopy in patients with gastrointestinal bleeding. N Engl J Med 2007; 356:1631.

45. Green FW Jr, Kaplan MM, Curtis LE, Levine PH. Effect of acid and pepsin on blood coagulation and platelet aggregation. A possible contributor prolonged gastroduodenal mucosal hemorrhage. Gastroenterology 1978; 74:38.

46. Kaviani MJ, Hashemi MR, Kazemifar AR, et al. Effect of oral omeprazole in reducing re-bleeding in bleeding peptic ulcers: a prospective, double-blind, randomized, clinical trial. Aliment Pharmacol Ther 2003; 17:211.

47. Chan WH, Khin LW, Chung YF, et al. Randomized controlled trial of standard versus high-dose intravenous omeprazole after endoscopic therapy in high-risk patients with acute peptic ulcer bleeding. Br J Surg 2011; 98:640.

48. Frossard JL, Spahr L, Queneau PE, et al. Erythromycin intravenous bolus infusion in acute upper gastrointestinal bleeding: a randomized, controlled, double-blind trial. Gastroenterology 2002; 123:17.

49. Coffin B, Pocard M, Panis Y, et al. Erythromycin improves the quality of EGD in patients with acute upper GI bleeding: a randomized controlled study. Gastrointest Endosc 2002; 56:174.

50. Altraif I, Handoo FA, Aljumah A, et al. Effect of erythromycin before endoscopy in patients presenting with variceal bleeding: a prospective, randomized, double-blind, placebo-

controlled trial. Gastrointest Endosc 2011; 73:245.

51. Carbonell N, Pauwels A, Serfaty L, et al. Erythromycin infusion prior to endoscopy for acute upper gastrointestinal bleeding: a randomized, controlled, double-blind trial. Am J Gastroenterol 2006; 101:1211.
52. Pateron D, Vicaut E, Debuc E, et al. Erythromycin infusion or gastric lavage for upper gastrointestinal bleeding: a multicenter randomized controlled trial. Ann Emerg Med 2011; 57:582.
53. Rudzki S, Czekalowski S, Michalak K, et al. [Erythromycin improves quality of endoscopy for acute upper gastrointestinal bleeding]. Wiad Lek 2006; 59:490.
54. Javad Ehsani Ardakani M, Zare E, Basiri M, Mohaghegh Shalmani H. Erythromycin decreases the time and improves the quality of EGD in patients with acute upper GI bleeding. Gastroenterol Hepatol Bed Bench 2013; 6:195.
55. Rahman R, Nguyen DL, Sohail U, et al. Pre-endoscopic erythromycin administration in upper gastrointestinal bleeding: an updated meta-analysis and systematic review. Ann Gastroenterol 2016; 29:312.
56. Adão D, Gois AF, Pacheco RL, et al. Erythromycin prior to endoscopy for acute upper gastrointestinal haemorrhage. Cochrane Database Syst Rev 2023; 2:CD013176.
57. Bennett C, Klingenberg SL, Langholz E, Gluud LL. Tranexamic acid for upper gastrointestinal bleeding. Cochrane Database Syst Rev 2014; :CD006640.
58. HALT-IT Trial Collaborators. Effects of a high-dose 24-h infusion of tranexamic acid on death and thromboembolic events in patients with acute gastrointestinal bleeding (HALT-IT): an international randomised, double-blind, placebo-controlled trial. Lancet 2020; 395:1927.
59. Kolkman JJ, Meuwissen SG. A review on treatment of bleeding peptic ulcer: a collaborative task of gastroenterologist and surgeon. Scand J Gastroenterol Suppl 1996; 218:16.
60. Jutabha R, Jensen DM. Management of upper gastrointestinal bleeding in the patient with chronic liver disease. Med Clin North Am 1996; 80:1035.
61. Adang RP, Vismans JF, Talmon JL, et al. Appropriateness of indications for diagnostic upper gastrointestinal endoscopy: association with relevant endoscopic disease. Gastrointest Endosc 1995; 42:390.
62. Forrest JA, Finlayson ND, Shearman DJ. Endoscopy in gastrointestinal bleeding. Lancet 1974; 2:394.
63. Lau JYW, Yu Y, Tang RSY, et al. Timing of Endoscopy for Acute Upper Gastrointestinal Bleeding. N Engl J Med 2020; 382:1299.

64. Sarin N, Monga N, Adams PC. Time to endoscopy and outcomes in upper gastrointestinal bleeding. Can J Gastroenterol 2009; 23:489.

65. Bjorkman DJ, Zaman A, Fennerty MB, et al. Urgent vs. elective endoscopy for acute non-variceal upper-GI bleeding: an effectiveness study. Gastrointest Endosc 2004; 60:1.

66. Tsoi KK, Ma TK, Sung JJ. Endoscopy for upper gastrointestinal bleeding: how urgent is it? Nat Rev Gastroenterol Hepatol 2009; 6:463.

67. Tsoi KK, Chiu PW, Chan FK, et al. The risk of peptic ulcer bleeding mortality in relation to hospital admission on holidays: a cohort study on 8,222 cases of peptic ulcer bleeding. Am J Gastroenterol 2012; 107:405.

68. Wysocki JD, Srivastav S, Winstead NS. A nationwide analysis of risk factors for mortality and time to endoscopy in upper gastrointestinal haemorrhage. Aliment Pharmacol Ther 2012; 36:30.

69. Kumar NL, Cohen AJ, Nayor J, et al. Timing of upper endoscopy influences outcomes in patients with acute nonvariceal upper GI bleeding. Gastrointest Endosc 2017; 85:945.

70. Garg SK, Anugwom C, Campbell J, et al. Early esophagogastroduodenoscopy is associated with better Outcomes in upper gastrointestinal bleeding: a nationwide study. Endosc Int Open 2017; 5:E376.

71. Laursen SB, Leontiadis GI, Stanley AJ, et al. Relationship between timing of endoscopy and mortality in patients with peptic ulcer bleeding: a nationwide cohort study. Gastrointest Endosc 2017; 85:936.

72. Cho SH, Lee YS, Kim YJ, et al. Outcomes and Role of Urgent Endoscopy in High-Risk Patients With Acute Nonvariceal Gastrointestinal Bleeding. Clin Gastroenterol Hepatol 2018; 16:370.

73. Jeong N, Kim KS, Jung YS, et al. Delayed endoscopy is associated with increased mortality in upper gastrointestinal hemorrhage. Am J Emerg Med 2019; 37:277.

74. Guo CLT, Wong SH, Lau LHS, et al. Timing of endoscopy for acute upper gastrointestinal bleeding: a territory-wide cohort study. Gut 2022; 71:1544.

75. Balderas V, Bhore R, Lara LF, et al. The hematocrit level in upper gastrointestinal hemorrhage: safety of endoscopy and outcomes. Am J Med 2011; 124:970.

76. Cappell MS, Iacovone FM Jr. Safety and efficacy of esophagogastroduodenoscopy after myocardial infarction. Am J Med 1999; 106:29.

77. Barth KH. Radiological intervention in upper and lower gastrointestinal bleeding. Baillieres Clin Gastroenterol 1995; 9:53.

78. Emslie JT, Zarnegar K, Siegel ME, Beart RW Jr. Technetium-99m-labeled red blood cell scans in the investigation of gastrointestinal bleeding. Dis Colon Rectum 1996; 39:750.

79. Gralnek IM, Ching JY, Maza I, et al. Capsule endoscopy in acute upper gastrointestinal hemorrhage: a prospective cohort study. Endoscopy 2013; 45:12.

80. Meltzer AC, Ali MA, Kresiberg RB, et al. Video capsule endoscopy in the emergency department: a prospective study of acute upper gastrointestinal hemorrhage. Ann Emerg Med 2013; 61:438.

81. Chandran S, Testro A, Urquhart P, et al. Risk stratification of upper GI bleeding with an esophageal capsule. Gastrointest Endosc 2013; 77:891.

82. Meltzer AC, Pinchbeck C, Burnett S, et al. Emergency physicians accurately interpret video capsule endoscopy findings in suspected upper gastrointestinal hemorrhage: a video survey. Acad Emerg Med 2013; 20:711.

83. Sung JJ, Tang RS, Ching JY, et al. Use of capsule endoscopy in the emergency department as a triage of patients with GI bleeding. Gastrointest Endosc 2016; 84:907.

84. Etzel JP, Williams JL, Jiang Z, et al. Diagnostic yield of colonoscopy to evaluate melena after a nondiagnostic EGD. Gastrointest Endosc 2012; 75:819.

85. Rockall TA, Logan RF, Devlin HB, Northfield TC. Selection of patients for early discharge or outpatient care after acute upper gastrointestinal haemorrhage. National Audit of Acute Upper Gastrointestinal Haemorrhage. Lancet 1996; 347:1138.

86. Corley DA, Stefan AM, Wolf M, et al. Early indicators of prognosis in upper gastrointestinal hemorrhage. Am J Gastroenterol 1998; 93:336.

87. Stanley AJ, Robinson I, Forrest EH, et al. Haemodynamic parameters predicting variceal haemorrhage and survival in alcoholic cirrhosis. QJM 1998; 91:19.

88. Hay JA, Maldonado L, Weingarten SR, Ellrodt AG. Prospective evaluation of a clinical guideline recommending hospital length of stay in upper gastrointestinal tract hemorrhage. JAMA 1997; 278:2151.

89. Hay JA, Lyubashevsky E, Elashoff J, et al. Upper gastrointestinal hemorrhage clinical--guideline determining the optimal hospital length of stay. Am J Med 1996; 100:313.

90. Blatchford O, Murray WR, Blatchford M. A risk score to predict need for treatment for upper-gastrointestinal haemorrhage. Lancet 2000; 356:1318.

91. Cipolletta L, Bianco MA, Rotondano G, et al. Outpatient management for low-risk nonvariceal upper GI bleeding: a randomized controlled trial. Gastrointest Endosc 2002; 55:1.

92. Marmo R, Koch M, Cipolletta L, et al. Predicting mortality in non-variceal upper gastrointestinal bleeders: validation of the Italian PNED Score and Prospective Comparison with the Rockall Score. Am J Gastroenterol 2010; 105:1284.

93. Pang SH, Ching JY, Lau JY, et al. Comparing the Blatchford and pre-endoscopic Rockall score in predicting the need for endoscopic therapy in patients with upper GI hemorrhage. Gastrointest Endosc 2010; 71:1134.
94. Saltzman JR, Tabak YP, Hyett BH, et al. A simple risk score accurately predicts in-hospital mortality, length of stay, and cost in acute upper GI bleeding. Gastrointest Endosc 2011; 74:1215.
95. García-Iglesias P, Villoria A, Suarez D, et al. Meta-analysis: predictors of rebleeding after endoscopic treatment for bleeding peptic ulcer. Aliment Pharmacol Ther 2011; 34:888.
96. Kumar NL, Claggett BL, Cohen AJ, et al. Association between an increase in blood urea nitrogen at 24 hours and worse outcomes in acute nonvariceal upper GI bleeding. Gastrointest Endosc 2017; 86:1022.
97. Das A, Wong RC. Prediction of outcome of acute GI hemorrhage: a review of risk scores and predictive models. Gastrointest Endosc 2004; 60:85.
98. Brullet E, Campo R, Calvet X, et al. A randomized study of the safety of outpatient care for patients with bleeding peptic ulcer treated by endoscopic injection. Gastrointest Endosc 2004; 60:15.
99. Gralnek IM, Dulai GS. Incremental value of upper endoscopy for triage of patients with acute non-variceal upper-GI hemorrhage. Gastrointest Endosc 2004; 60:9.
100. Longstreth GF, Feitelberg SP. Successful outpatient management of acute upper gastrointestinal hemorrhage: use of practice guidelines in a large patient series. Gastrointest Endosc 1998; 47:219.
101. Lee JG, Turnipseed S, Romano PS, et al. Endoscopy-based triage significantly reduces hospitalization rates and costs of treating upper GI bleeding: a randomized controlled trial. Gastrointest Endosc 1999; 50:755.
102. Imperiale TF, Dominitz JA, Provenzale DT, et al. Predicting poor outcome from acute upper gastrointestinal hemorrhage. Arch Intern Med 2007; 167:1291.
103. Das A, Ben-Menachem T, Farooq FT, et al. Artificial neural network as a predictive instrument in patients with acute nonvariceal upper gastrointestinal hemorrhage. Gastroenterology 2008; 134:65.
104. Stanley AJ, Ashley D, Dalton HR, et al. Outpatient management of patients with low-risk upper-gastrointestinal haemorrhage: multicentre validation and prospective evaluation. Lancet 2009; 373:42.
105. Church NI, Dallal HJ, Masson J, et al. Validity of the Rockall scoring system after endoscopic therapy for bleeding peptic ulcer: a prospective cohort study. Gastrointest Endosc 2006;

63:606.

106. Stanley AJ, Laine L, Dalton HR, et al. Comparison of risk scoring systems for patients presenting with upper gastrointestinal bleeding: international multicentre prospective study. BMJ 2017; 356:i6432.
107. Cheng DW, Lu YW, Teller T, et al. A modified Glasgow Blatchford Score improves risk stratification in upper gastrointestinal bleed: a prospective comparison of scoring systems. Aliment Pharmacol Ther 2012; 36:782.
108. Ramaekers R, Mukarram M, Smith CA, Thiruganasambandamoorthy V. The Predictive Value of Preendoscopic Risk Scores to Predict Adverse Outcomes in Emergency Department Patients With Upper Gastrointestinal Bleeding: A Systematic Review. Acad Emerg Med 2016; 23:1218.
109. Laursen SB, Oakland K, Laine L, et al. ABC score: a new risk score that accurately predicts mortality in acute upper and lower gastrointestinal bleeding: an international multicentre study. Gut 2021; 70:707.
110. Barkun AN, Bardou M, Kuipers EJ, et al. International consensus recommendations on the management of patients with nonvariceal upper gastrointestinal bleeding. Ann Intern Med 2010; 152:101.

Topic 2548 Version 95.0

GRAPHICS

Upper GI bleeding in adults: Rapid overview of emergency management

Major causes*
Peptic ulcer, esophagogastric varices, arteriovenous malformation, tumor, esophageal (Mallory-Weiss) tear
Clinical features
History
Use of: NSAIDs, aspirin, anticoagulants, antiplatelet agents
Alcohol abuse, previous GI bleed, liver disease, coagulopathy
Symptoms and signs: Abdominal pain, hematemesis or "coffee ground" emesis, passing melena/tarry stool (stool may be frankly bloody or maroon with massive or brisk upper GI bleeding)
Examination
Tachycardia; orthostatic blood pressure changes suggest moderate to severe blood loss; hypotension suggests life-threatening blood loss (hypotension may be late finding in healthy younger adult)
Rectal examination is performed to assess stool color (melena versus hematochezia versus brown)
Significant abdominal tenderness accompanied by signs of peritoneal irritation (eg, involuntary guarding) suggests perforation
Diagnostic testing
Obtain type and crossmatch for hemodynamic instability, severe bleeding, or high-risk patient; obtain type and screen for hemodynamically stable patient without signs of severe bleeding
Obtain hemoglobin concentration (note that measurement may be inaccurate with acute severe hemorrhage), platelet count, coagulation studies (prothrombin time with INR), liver enzymes (AST, ALT), albumin, BUN, and creatinine
Nasogastric lavage may be helpful if the source of bleeding is unclear (upper or lower GI tract) or to clean the stomach prior to endoscopy
Treatment
Closely monitor airway, clinical status, vital signs, cardiac rhythm, urine output, nasogastric output (if nasogastric tube in place)
Do **NOT** give patient anything by mouth
Establish two large bore IV lines (16 gauge or larger)

Provide supplemental oxygen (goal oxygen saturation ≥94% for patients without COPD)
Treat hypotension initially with rapid, bolus infusions of isotonic crystalloid (eg, 500 to 1000 mL per bolus; use smaller boluses and lower total volumes for patients with compromised cardiac function)
Transfusion:
For severe, ongoing bleeding, immediately transfuse blood products in 1:1:1 ration of RBCs, plasma, and platelets, as for trauma patients
For hemodynamic instability despite crystalloid resuscitation, transfuse 1 to 2 units RBCs
For hemoglobin <8 g/dL (80 g/L) in high-risk patients (eg, older adult, coronary artery disease), transfuse 1 unit RBCs and reassess the patient's clinical condition
For hemoglobin <7 g/dL (70 g/L) in low-risk patients, transfuse 1 units RBCs and reassess the patient's clinical condition
Avoid over-transfusion with possible variceal bleeding
Give plasma for coagulopathy or after transfusing four units of RBCs; give platelets for thrombocytopenia (platelets <50,000) or platelet dysfunction (eg, chronic aspirin therapy) or after transfusing four units of RBCs
Obtain immediate consultation with gastroenterologist; obtain surgical and interventional radiology consultation for any large-scale bleeding¶
Pharmacotherapy for all patients with suspected or known severe bleeding:
Give a proton pump inhibitor:
Evidence of active bleeding (eg, hematemesis, hemodynamic instability), give esomeprazole or pantoprazole, 80 mg IV
No evidence of active bleeding, give esomeprazole or pantoprazole, 40 mg IV
Endoscopy delayed beyond 12 hours, give second dose of esomeprazole or pantoprazole, 40 mg IV
Pharmacotherapy for known or suspected esophagogastric variceal bleeding and/or cirrhosis:
Give somatostatin or an analogue (eg, octreotide 50 mcg IV bolus followed by 50 mcg/hour continuous IV infusion)
Give an IV antibiotic (eg, ceftriaxone or fluoroquinolone)
Balloon tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using any of several devices (eg, Sengstaken-Blakemore tube, Minnesota tube); tracheal intubation is necessary if such a device is to be placed; ensure proper device placement prior to inflation to avoid esophageal rupture

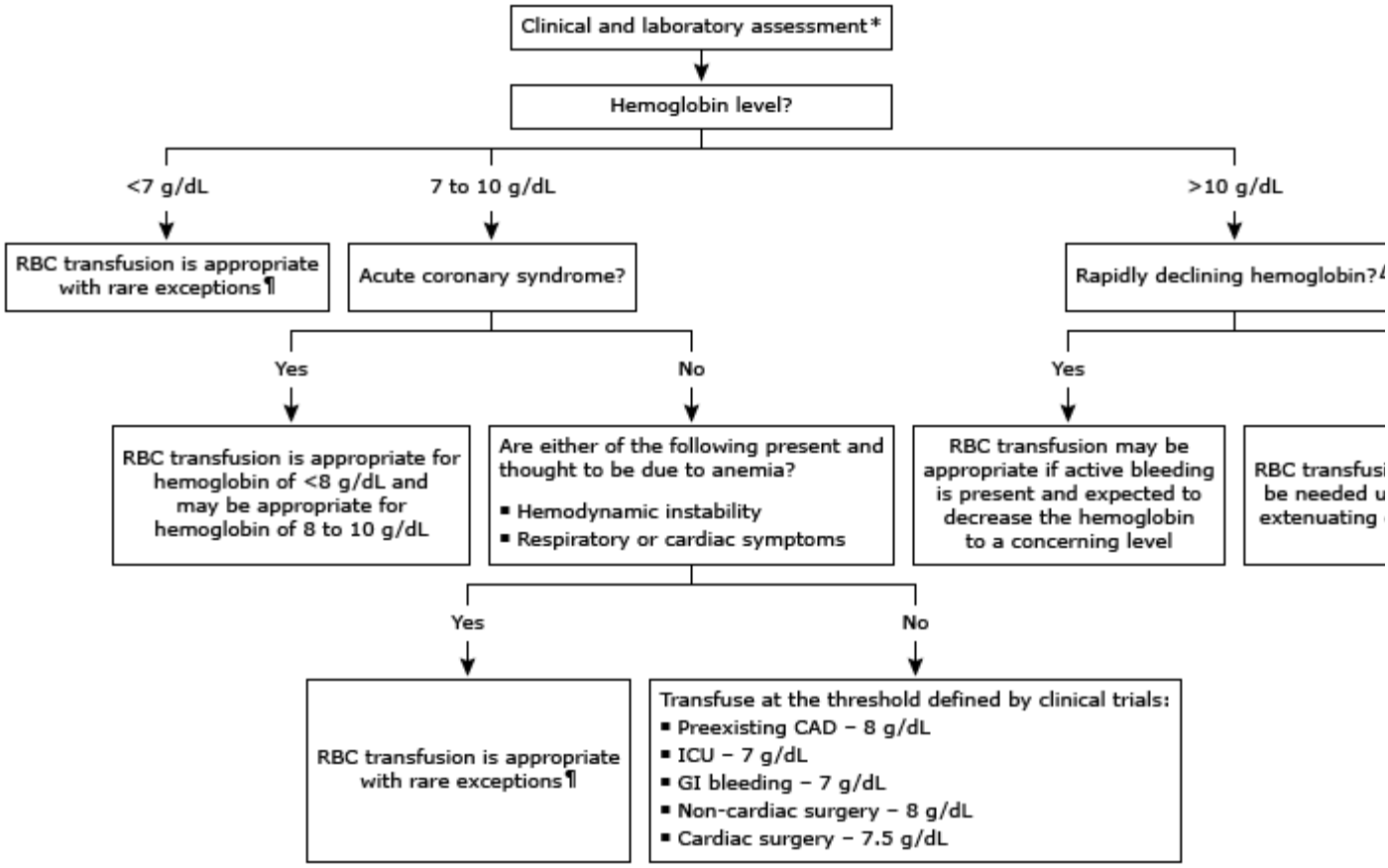
COPD: chronic obstructive pulmonary disease; GI: gastrointestinal; INR: international normalized ratio; AST: aspartate aminotransferase; ALT: alanine aminotransferase; BUN: blood urea nitrogen; IV: intravenous; RBC: red blood cells.

* An important but uncommon cause of gastrointestinal hemorrhage is vascular-enteric fistula, typically aortoduodenal fistula related to erosion of a prosthetic aortic graft.

¶ Minimally invasive techniques to control bleeding include sclerotherapy, embolization, and other vascular occlusion techniques. For patients with massive hemorrhage, resuscitative endovascular balloon occlusion of the aorta (REBOA) can be used to limit blood loss and support perfusion of vital organs until the sites of bleeding can be directly controlled.

Graphic 72195 Version 16.0

Red blood cell (RBC) transfusion decisions in adults



The decision to transfuse always incorporates an assessment by the clinician caring for the patient. Thresho here are based on data from clinical trials; refer to UpToDate for details. This algorithm does **not** apply to in with hemoglobinopathies (sickle cell disease, transfusion-dependent thalassemia); separate criteria apply to individuals as discussed in UpToDate. To convert hemoglobin to g/L, multiply by 10 (hemoglobin of 7 g/dL = Refer to UpToDate topics on indications for transfusion for further details and discussions.

RBC: red blood cell; CAD: coronary artery disease; ICU: intensive care unit; GI: gastrointestinal.

* Assessment includes:
- Symptoms (and whether attributable to anemia)
- Clinical status (vital signs, signs of hemodynamic instability, cardiac and respiratory examination)
- Underlying comorbidities
- Hemoglobin level
- Rate of hemoglobin decline and cause (active bleeding versus ongoing hemolysis versus decreased R production)

¶ Rarely, an individual with hemoglobin below an accepted threshold may decline transfusion (Jehovah's Wit healthy young adult); it is important that they understand the risks and alternatives. Rarely, an individual wit hemoglobin above 10 g/dL may warrant transfusion, such as if there are clear symptoms attributable to ane cause of anemia cannot otherwise be rapidly treated.

Δ Rapidly declining hemoglobin includes rapid bleeding associated with hemodynamic instability or a fall in of ≥2 g/dL per day.

Graphic 131907 Version 2.0

Thresholds for red blood cell transfusion in adults

Condition	Hemoglobin threshold for transfusion
Symptomatic patient (eg, myocardial ischemia, hemodynamic instability)	10 g/dL*[1]
Hospitalized patient	
Preexisting coronary artery disease	8 g/dL*
Acute coronary syndromes, including acute MI	8 to 10 g/dL¶[2]
ICU (hemodynamically stable)	7 g/dL*[3,4]
Gastrointestinal bleeding (hemodynamically stable)	7 g/dL*[5,6]
Orthopedic surgery	8 g/dL*[1]
Cardiac surgery	7.5 g/dL*[7,8]
Ambulatory outpatient	
Oncology patient in treatment	7 to 8 g/dL¶
Palliative care setting	As needed for symptoms; hospice benefits may vary

These thresholds are not a substitute for direct assessment of the patient and clinical judgment. Refer to UpToDate topics on red blood cell transfusion and specific clinical settings for further details. Hospitalized patients with heart failure are an especially challenging case because there are no data from large randomized trials, and the improvement in oxygenation from transfusion must be balanced against the risks of worsening heart failure due to the volume of the transfused blood. The authors generally use a threshold of 7 to 8 g/dL in this population, erring on the side of a higher hemoglobin level in those who are expected to be able to better tolerate the volume load. In patients who do not fit into these clinical subgroups, we recommend that transfusion based on the location of care (ICU versus other) or the similarity of their underlying disease to those patient groups where data are available. In most cases, a 7 or 8 g/dL threshold is appropriate.

MI: myocardial infarction; ICU: intensive care unit.

* Based on results from clinical trial(s). Some experts may use different values. As an example, in individuals with gastrointestinal bleeding, it is often difficult, if not impossible, to estimate what the nadir hemoglobin will be, and some experts recommend a transfusion threshold of 8 g/dL[6].

¶ There are no large clinical trials yet performed in this setting. These recommendations are based on the authors' opinions.

References:

1. *Carson JL, Terrin ML, Noveck H, et al. Liberal or restrictive transfusion in high-risk patients after hip surgery. N Engl J Med 2011; 365:2453.*

2. *Ducrocq G, Gonzalez-Juanetey JR, Puymirat E, et al. Effect of a restrictive vs liberal blood transfusion strategy on major cardiovascular events among patients with acute myocardial infarction and anemia: The REALITY randomized clinical trial. JAMA 2021; 325:552.*
3. *Hébert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. Transfusion Requirements in Critical Care Investigators, Canadian Critical Care Trials Group. N Engl J Med 1999; 340:409.*
4. *Lacroix J, Hebert PC, Hutchison JS, et al. Transfusion strategies for patients in pediatric intensive care units. N Engl J Med 2007; 356:1609.*
5. *Villanueva C, Colomo A, Bosch A, et al. Transfusion strategies for acute upper gastrointestinal bleeding. N Engl J Med 2013; 368:11.*
6. *Barkun AN, Almadi M, Kuipers EJ, et al. Management of nonvariceal upper gastrointestinal bleeding: Guideline recommendations from the International Consensus Group. Ann Intern Med 2019; 171:805.*
7. *Hajjar LA, Vincent JL, Galas FR, et al. Transfusion requirements after cardiac surgery: the TRACS randomized controlled trial. JAMA 2010; 304:1559.*
8. *Mazer CD, Whitlock RP, Fergusson DA, et al. Restrictive or liberal red-cell transfusion for cardiac surgery. N Engl J Med 2017; 377:2133.*

Graphic 93934 Version 9.0

Emergency reversal of anticoagulation from warfarin for life-threatening hemorrhage in adults: Suggested approaches based upon available resources

A. If 4-factor prothrombin complex concentrate (4F PCC) is available (preferred approach):
1. Give 4F PCC* 1500 to 2000 units¶ IV over 10 minutes. Check INR 15 minutes after completion of the infusion. If INR is not ≤1.5, give additional 4F PCC (refer to topic or drug reference for details).
2. Give vitamin K 10 mg IV over 10 to 20 minutes.
B. If 3-factor prothrombin complex concentrate (3F PCC) is available but 4F PCC is not available:
1. Give 3F PCC* 1500 to 2000 units¶ IV over 10 minutes. Check INR 15 minutes after completion of the infusion. If INR is not ≤1.5, give additional 3F PCC (refer to topic or drug reference for details).
2. Give Factor VIIa 20 mcg/kg IV **OR** give FFP 2 units IV by rapid infusion. Factor VIIa may be preferred if volume overload is a concern.
3. Give vitamin K 10 mg IV over 10 to 20 minutes.
C. If neither 3F PCC nor 4F PCC is available:
1. Give FFP 2 units IV by rapid infusion. Check INR 15 minutes after completion of infusion. If INR ≥1.5, administer 2 additional units of FFP IV rapid infusion. Repeat process until INR ≤1.5. May wish to administer loop diuretic between FFP infusions if volume overload is a concern.
2. Give vitamin K 10 mg IV over 10 to 20 minutes.

These products and doses are for use in life-threatening bleeding only. Evidence of life-threatening bleeding and over-anticoagulation with a vitamin K antagonist (eg, warfarin) are required. Anaphylaxis and transfusion reactions can occur.

It may be reasonable to thaw 4 units of FFP while awaiting the PT/INR. The transfusion service may substitute other plasma products for FFP (eg, Plasma Frozen Within 24 Hours After Phlebotomy [PF24]); these products are considered clinically interchangeable. PCC will reverse anticoagulation within minutes of administration; FFP administration can take hours due to the volume required; vitamin K effect takes 12 to 24 hours, but administration of vitamin K is needed to counteract the long half-life of warfarin. Subsequent monitoring of the PT/INR is needed to guide further therapy. Refer to topics on warfarin reversal in individual situations for further management.

PCC: unactivated prothrombin complex concentrate; 4F PCC: PCC containing coagulation factors II, VII, IX, X, protein S and protein C; 3F PCC: PCC containing factors II, IX, and X and only trace factor VII; FFP: fresh frozen plasma; PT: prothrombin time; INR: international normalized ratio; FEIBA: factor eight inhibitor bypassing agent.

* Before use, check product label to confirm factor types (3 versus 4 factor) and concentration. Activated complexes and single-factor IX products (ie, FEIBA, AlphaNine, Mononine, Immunine, BeneFix) are NOT used for warfarin reversal.

¶ PCC doses shown are those suggested for initial treatment of emergency conditions. Subsequent treatment is based on INR and patient weight if available. Refer to topic and Lexicomp drug reference included with UpToDate for INR-based dosing.

Graphic 89478 Version 10.0

Direct oral anticoagulant-associated bleeding reversal strategies

Type of bleeding	Agent	Possible interventions
Life-threatening or imminently fatal bleeding (eg, intracranial, retroperitoneal, compartment syndrome, massive gastrointestinal)	Dabigatran (Pradaxa)	▪ Idarucizumab ▪ Activated PCC* (eg, FEIBA) ▪ Antifibrinolytic agent (eg, tranexamic acid, epsilon-aminocaproic acid) ▪ Anticoagulant discontinuation ▪ Oral activated charcoal (if last dose within prior two hours) ▪ Hemodialysis ▪ RBC transfusions if needed for anemia ▪ Platelet transfusions if needed for thrombocytopenia or impaired platelet function (eg, due to aspirin) ▪ Surgical/endoscopic intervention if appropriate
	Rivaroxaban (Xarelto), apixaban (Eliquis), edoxaban (Lixiana)	▪ Andexanet alfa (AndexXa) **or** a 4-factor unactivated PCC (eg, Kcentra) ▪ Antifibrinolytic agent (eg, tranexamic acid, epsilon-aminocaproic acid) ▪ Anticoagulant discontinuation ▪ Oral activated charcoal (if last dose recent enough) ▪ RBC transfusions if needed for anemia ▪ Platelet transfusions if needed for thrombocytopenia or impaired platelet function (eg, due to aspirin) ▪ Surgical/endoscopic intervention if appropriate
Minor bleeding (eg, epistaxis, uncomplicated soft tissue bleeding, minor [slow] gastrointestinal bleeding)	Dabigatran (Pradaxa)	▪ Local hemostatic measures ▪ Possible anticoagulant discontinuation • Half-life (normal renal function¶): 12 to 17 hours ▪ Possible antifibrinolytic agent (eg, tranexamic acid, epsilon-aminocaproic acid)
	Rivaroxaban (Xarelto), apixaban (Eliquis), edoxaban (Lixiana)	▪ Local hemostatic measures ▪ Possible anticoagulant discontinuation • Half-lives (normal renal function¶): ○ Rivaroxaban 5 to 9 hours

		◦ Apixaban 8 to 15 hours ◦ Edoxaban 6 to 11 hours ▪ Possible antifibrinolytic agent (eg, tranexamic acid, epsilon-aminocaproic acid)

The table describes measures that may be used to manage bleeding associated with DOACs. Clinical judgment is essential in all cases of DOAC-associated bleeding in order to assess the risks of bleeding and weigh these against the risks of thrombosis if anticoagulation is discontinued or reversed. Refer to UpToDate topics on the use of direct thrombin inhibitors and direct factor Xa inhibitors and management of DOAC-associated bleeding for further details and dosing. The onset of all of the agents discussed herein is approximately 2 to 4 hours.

PCC: prothrombin complex concentrate; FEIBA: factor eight inhibitor bypassing activity; RBC: red blood cell; DOAC: direct oral anticoagulant.

* Use activated PCC only if idarucizumab is unavailable and/or if continued bleeding is reasonably likely to be fatal within hours.

¶ The anticoagulant effect of these agents (especially dabigatran) will dissipate more slowly as renal function declines. Severe hepatic failure may also prolong the half-life for apixaban, edoxaban, and rivaroxaban.

Graphic 96230 Version 18.0

Cytochrome P450 3A (including 3A4) inhibitors and inducers

Strong inhibitors	Moderate inhibitors	Strong inducers	Moderate inducers
▪ Adagrasib ▪ Atazanavir ▪ Ceritinib ▪ Clarithromycin ▪ Cobicistat and cobicistat-containing coformulations ▪ Darunavir ▪ Idelalisib ▪ Indinavir ▪ Itraconazole ▪ Ketoconazole ▪ Levoketoconazole ▪ Lonafarnib ▪ Lopinavir ▪ Mifepristone* ▪ Nefazodone ▪ Nelfinavir ▪ Nirmatrelvir-ritonavir ▪ Ombitasvir-paritaprevir-ritonavir ▪ Ombitasvir-paritaprevir-ritonavir plus dasabuvir ▪ Posaconazole ▪ Ritonavir and ritonavir-containing coformulations ▪ Saquinavir ▪ Tucatinib ▪ Voriconazole	▪ Amiodarone¶ ▪ Aprepitant ▪ Berotralstat ▪ Cimetidine¶ ▪ Conivaptan ▪ Crizotinib ▪ Cyclosporine¶ ▪ Diltiazem ▪ Duvelisib ▪ Dronedarone ▪ Erythromycin ▪ Fedratinib ▪ Fluconazole ▪ Fosamprenavir ▪ Fosaprepitant¶ ▪ Fosnetupitant-palonosetron ▪ Grapefruit juice ▪ Imatinib ▪ Isavuconazole (isavuconazonium sulfate) ▪ Lefamulin ▪ Letermovir ▪ Netupitant ▪ Nilotinib ▪ Ribociclib ▪ Schisandra ▪ Verapamil	▪ Apalutamide ▪ Carbamazepine ▪ Enzalutamide ▪ Fosphenytoin ▪ Lumacaftor ▪ Lumacaftor-ivacaftor ▪ Mitotane ▪ Phenobarbital ▪ Phenytoin ▪ Primidone ▪ Rifampin (rifampicin)	▪ Bexarotene ▪ Bosentan ▪ Cenobamate ▪ Dabrafenib ▪ Dexamethasone$^{\Delta}$ ▪ Dipyrone ▪ Efavirenz ▪ Elagolix, estradiol, and norethindrone therapy pack$^{\diamond}$ ▪ Eslicarbazepine ▪ Etravirine ▪ Lorlatinib ▪ Mitapivat ▪ Modafinil ▪ Nafcillin ▪ Pexidartinib ▪ Rifabutin ▪ Rifapentine ▪ Sotorasib ▪ St. John's wort

- For drug interaction purposes, the inhibitors and inducers of CYP3A metabolism listed above can alter serum concentrations of drugs that are dependent upon the CYP3A subfamily of liver enzymes, including CYP3A4, for elimination or activation.
- These classifications are based upon US Food and Drug Administration (FDA) guidance.[1,2] Other sources may use a different classification system resulting in some agents being classified differently.
- Data are for systemic drug forms. Degree of inhibition or induction may be altered by dose, method, and timing of administration.
- Weak inhibitors and inducers are not listed in this table with exception of a few examples. Clinically significant interactions can occasionally occur due to weak inhibitors and inducers (eg, target drug is highly dependent on CYP3A4 metabolism and has a narrow therapeutic index). Accordingly, specific interactions should be checked using a drug interaction program such as the Lexicomp drug interactions program included within UpToDate.
- Refer to UpToDate topics on specific agents and indications for further details.

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* Mifepristone is a significant inhibitor of CYP3A4 when used chronically (eg, for hyperglycemia in patients with Cushing syndrome); not in single-dose use.

¶ Classified as a weak inhibitor of CYP3A4 according to FDA system.[1]

Δ Classified as a weak inducer of CYP3A4 according to FDA system.[1]

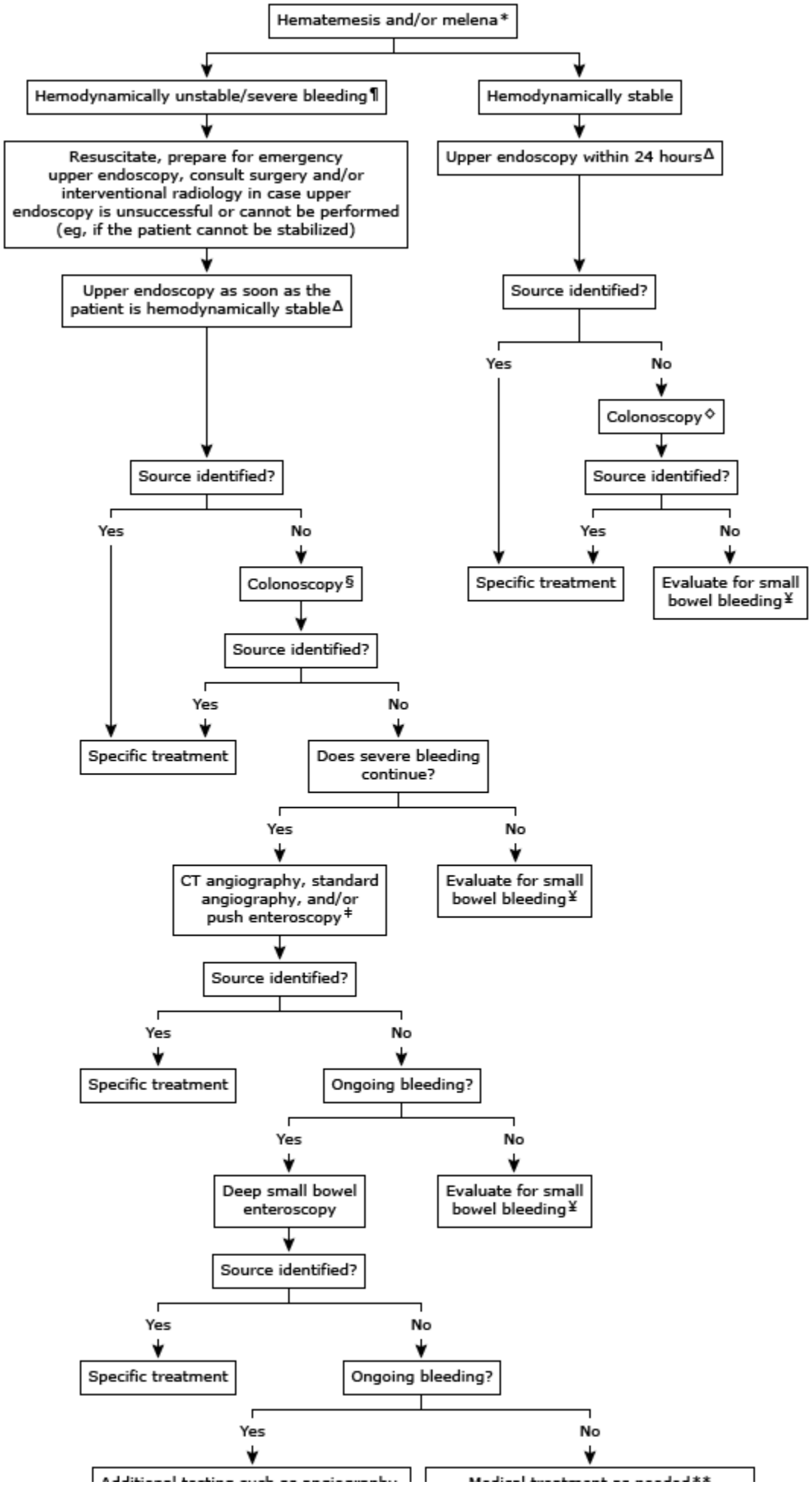
◇ The fixed-dose combination therapy pack taken in the approved regimen has moderate CYP3A4 induction effects. When elagolix is used as a single agent, it is a weak CYP3A4 inducer. Norethindrone and estradiol are not CYP3A4 inducers.

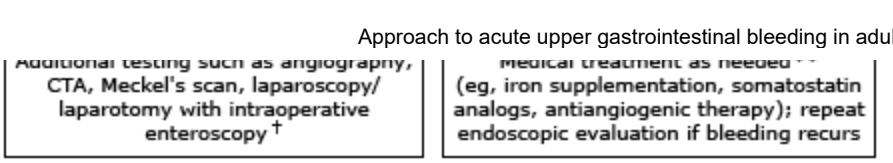
References:

1. *Clinical Drug Interaction Studies — Cytochrome P450 Enzyme- and Transporter-Mediated Drug Interactions Guidance for Industry (January 2020) available at: https://www.fda.gov/regulatory-information/search-fda-guidance-documents/clinical-drug-interaction-studies-cytochrome-p450-enzyme-and-transporter-mediated-drug-interactions.*
2. *US Food & Drug Administration. Drug Development and Drug Interactions: Table of Substrates, Inhibitors and Inducers. Available at: FDA.gov website.*

Graphic 76992 Version 95.0

Evaluation of suspected upper gastrointestinal bleeding


Hematemesis and/or melena*
Hemodynamically unstable/severe bleeding¶
Hemodynamically stable
Resuscitate, prepare for emergency upper endoscopy, consult surgery and/or interventional radiology in case upper endoscopy is unsuccessful or cannot be performed (eg, if the patient cannot be stabilized)
Upper endoscopy within 24 hours△
Upper endoscopy as soon as the patient is hemodynamically stable△
Source identified?
Yes
No
Colonoscopy◇
Source identified?
Yes
No
Specific treatment
Evaluate for small bowel bleeding¥
Source identified?
Yes
No
Colonoscopy§
Source identified?
Yes
No
Specific treatment
Does severe bleeding continue?
Yes
No
CT angiography, standard angiography, and/or push enteroscopy‡
Evaluate for small bowel bleeding¥
Source identified?
Yes
No
Specific treatment
Ongoing bleeding?
Yes
No
Deep small bowel enteroscopy
Evaluate for small bowel bleeding¥
Source identified?
Yes
No
Specific treatment
Ongoing bleeding?
Yes
No



GI: gastrointestinal; CT: computed tomographic; CTA: computed tomographic angiography; MR: magnetic resonance.

* The presence of both hematemesis and melena suggests that brisk bleeding is present.

¶ Bleeding associated with signs such as hypotension, tachycardia, or orthostatic hypotension.

Δ Consider evaluation with a side-viewing duodenoscope if there are risk factors for hemobilia or hemosuccus pancreaticus; consider CTA (followed by push enteroscopy if the CTA is negative) in patients at risk for an aortoenteric fistula. Conventional angiography is typically performed if the patient remains hemodynamically unstable despite attempts at resuscitation.

◇ Patients who present with hematemesis do not need to undergo colonoscopy, since hematemesis suggests the bleeding is proximal to the ligament of Treitz. They should proceed directly to an evaluation for small bowel bleeding if upper endoscopy is negative. Colonoscopy is the next step in the evaluation of patients with melena.

§ If the patient becomes hemodynamically unstable following initial resuscitation, conventional angiography can be performed. Patients who present with hematemesis do not need to undergo colonoscopy and can skip this step in the evaluation because hematemesis suggests the bleeding is proximal to the ligament of Treitz.

¥ If the initial endoscopic evaluation was inadequate (eg, fair or poor visualization, failure to reach the cecum), repeat examination should be considered before initiating an evaluation for small bowel bleeding. Refer to UpToDate topic review on suspected small bowel bleeding for details.

‡ If not already done. If the patient remains hemodynamically stable and does not have evidence of aggressive bleeding (eg, ongoing hematochezia), perform a CTA or push enteroscopy (CTA is the initial test of choice if there is concern for an aortoenteric fistula). If the patient becomes hemodynamically unstable following initial resuscitation or has signs of aggressive bleeding, perform conventional angiography.

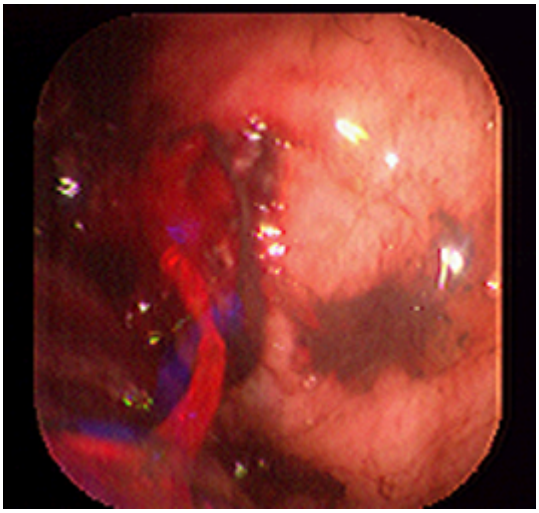
† If not already done, angiography or CTA may be obtained. If angiography or CTA has been performed and no source is identified, a Meckel's scan should be obtained in younger patients with overt bleeding, unless the only manifestation of bleeding was hematemesis. Surgical exploration is appropriate if no other studies have revealed a source and significant bleeding continues or if there is high suspicion for a small bowel neoplasm.

** If the deep small bowel enteroscopy was incomplete, a video capsule endoscopy study should be obtained, followed by CT enterography or MR enterography if the

capsule endoscopy is negative.

Graphic 105093 Version 4.0

Bleeding gastric ulcer

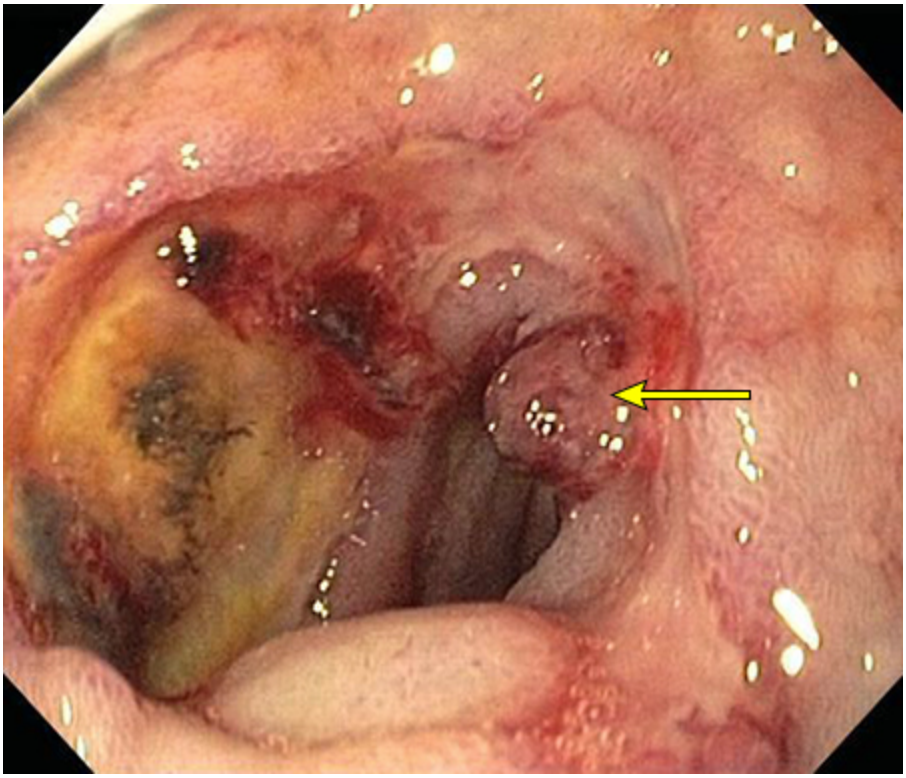


Endoscopy shows an actively bleeding gastric ulcer (Forrest classification Ia) along the lesser curvature.

Courtesy of Rome Jutabha, MD and Dennis M Jensen, MD.

Graphic 61646 Version 2.0

Duodenal ulcer with visible vessel

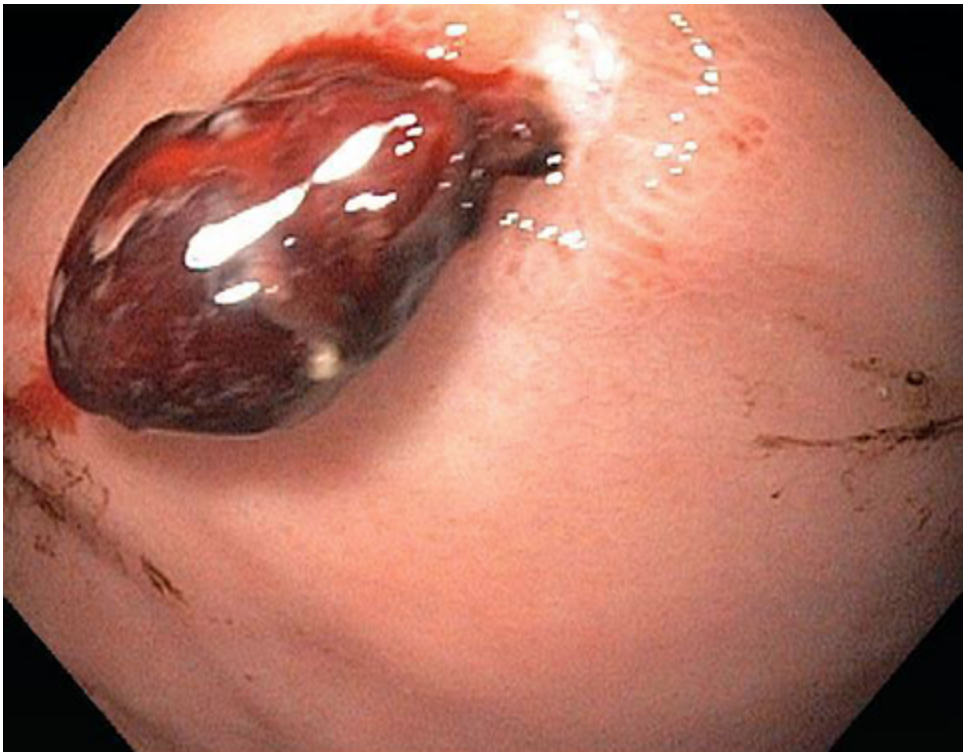


Upper endoscopy showing a duodenal ulcer with a nonbleeding visible vessel (arrow) in a large circumferential ulcer (Forrest classification IIa).

Courtesy of Rome Jutabha.

Graphic 54960 Version 4.0

Gastric ulcer with adherent clot

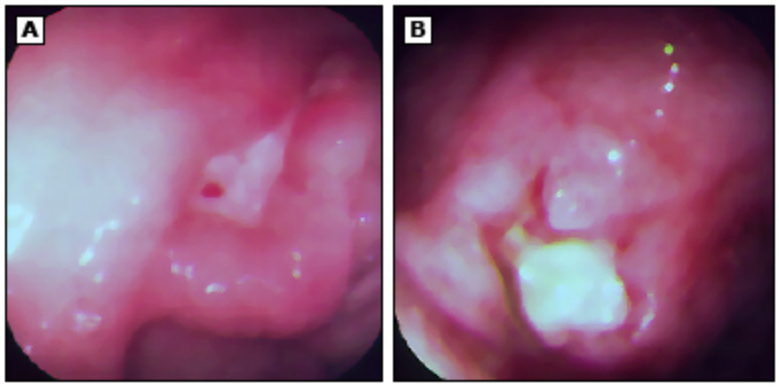


Upper endoscopy showing a gastric ulcer with an adherent clot (Forrest classification IIb).

Courtesy of Rome Jutabha, MD.

Graphic 76246 Version 1.0

Peptic ulcers at low risk for rebleeding



Ulcers with a flat pigmented spot (Forrest classification IIc; panel A) or a clean base (Forrest classification III, panel B) are at low risk for rebleeding and do not need to be treated endoscopically.

Courtesy of Rome Jutabha, MD and Dennis M Jensen, MD.

Graphic 52497 Version 2.0

Endoscopic predictors of recurrent peptic ulcer hemorrhage[1,2]

Endoscopic stigmata of recent hemorrhage	Prevalence, percent	Risk of rebleeding on medical management, percent
Active arterial bleeding (Forrest Ia)	12% (arterial bleeding + oozing)	55 (arterial bleeding + oozing)
Oozing without visible vessel (Forrest Ib)		
Non-bleeding visible vessel (Forrest IIa)	8	43
Adherent clot (Forrest IIb)	8	22
Flat spot (Forrest IIc)	16	10
Clean ulcer base (Forrest III)	55	5

References:

1. Katschinski B, Logan R, Davies J, et al. Prognostic factors in upper gastrointestinal bleeding. Dig Dis Sci 1994; 39:706.
2. Laine L, Jensen DM. Management of patients with ulcer bleeding. Am J Gastroenterol 2012; 107:345.

Graphic 78607 Version 8.0

Contributor Disclosures

John R Saltzman, MD, FACP, FACG, FASGE, AGAF No relevant financial relationship(s) with ineligible companies to disclose. **Mark Feldman, MD, MACP, AGAF, FACG** No relevant financial relationship(s) with ineligible companies to disclose. **Anne C Travis, MD, MSc, FACG, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

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