Resuscitation Of The Patient With Massive Upper Gastrointestinal Bleeding

Abstract

Managing an unstable patient with massive gastrointestinal bleeding can be challenging, but effective management can optimize patient outcomes. These patients require prompt recognition of severe illness, resuscitation and stabilization, and diagnostic and treatment modalities directed at identification and control of the source of bleeding. Multiple consultants (including gastroenterology, general surgery, and interventional radiology) are often required, and early consultation of these subspecialists can improve morbidity and mortality. Underlying comorbidities add complexity to the management of the bleeding patient and are associated with increased mortality.

Most cases of massive bleeding are due to an upper gastrointestinal source, and these patients have a higher mortality rate than those presenting with lower gastrointestinal bleeding. Common sources of upper gastrointestinal bleeding include gastric and duodenal ulcers, esophageal and gastric varices, and erosive esophagitis. Depending on the source of the bleeding, medical and/or surgical treatment modalities may be required. Utilization of emergent upper endoscopy and other medical therapies as well as transfusion of blood products are often necessary to ensure optimal patient outcomes. Knowledge of the emergency procedures and medications available, as well as familiarity with the treatment modalities used by consultants, will help the emergency physician orchestrate the care necessary to ensure patient survival.
Case Presentation

You receive a medic call for a 45-year-old male who called 911 for massive hematemesis. The medics report that the patient is an alcoholic who has been binge drinking since losing his job 1 week ago. He has vomited a large amount of frank blood twice. He is hypotensive, with a MAP of 50 mm Hg and a pulse of 128 beats per minute. EMS personnel have established a peripheral IV line and are infusing normal saline as fast as possible; they are 5 minutes out. After you page the gastroenterologist and ask the unit secretary to activate your institution’s massive transfusion protocol, you consider the potential causes of massive GI bleeding, the available therapeutic options, and the interventions you should undertake in the next few minutes to improve this critically ill patient’s chance for survival...

Introduction

Providing optimal care in the emergency department (ED) for patients with massive gastrointestinal (GI) bleeding requires coordination of multiple consultants as well as knowledge of both medical and surgical therapeutic options. For the purposes of this review, massive GI bleeding is defined as bleeding in a patient that has resulted in hemodynamic instability, with signs and symptoms consistent with hemorrhagic shock and evidence of either hematemesis or hematochezia. In order to provide initial resuscitation in the ED (either as a bridge to continued resuscitation in the intensive care unit or to diagnostic/therapeutic interventions in the operating room or interventional radiology), emergency physicians must be familiar with the wide variety of both diagnostic and therapeutic options as well as the underlying pathophysiology for massive GI hemorrhage. The majority (>75%) of massive GI bleeding cases are due to an upper GI source.\(^1\) Mortality rates for patients with massive GI bleeding range from 20% to 39%.\(^2\) Treatment options depend upon the underlying cause of the bleeding, and early intervention can mean the difference between survival and death. This issue of EMCC provides an overview of the current evidence for the treatment of patients with massive upper GI bleeding.

Critical Appraisal Of The Literature

A literature search of PubMed, Ovid MEDLINE\(^\text{®}\), and the Cochrane Library was performed using the following keywords: GI bleeding, severe GI bleeding, upper GI bleeding, and variceal bleeding treatment. The search focused only on literature in the English language for adult patients. More than 100 articles were reviewed, which provided the background information and basis for further literature review. Additionally, both national and international guidelines as well as the Cochrane Database of Systematic Reviews were searched for guidelines for the acute treatment of severe GI bleeding. Table 1 summarizes the current guidelines related to GI bleeding.\(^3\) The literature review and these guidelines suggest a general consensus about the importance of early evaluation and resuscitation in the ED as well as early involvement of appropriate consultants in order to avoid negative outcomes in patients with massive GI bleeding.

Etiology And Pathophysiology

The presence of GI bleeding with evidence of hemorrhagic shock requires prompt identification of the bleeding source. The most common etiologies of upper GI bleeding include: duodenal ulcers (28%), gastric ulcers (26%), gastritis (13%), varices (12%), and esophagitis (8%).\(^1\) GI bleeding due to esophageal or gastric varices and peptic ulceration carry the highest mortality.\(^2\) Exclusion of other bleeding sources (such as pulmonary or intranasal sources) is important. As with management of all cases of undifferentiated hypotension, initiation of resuscitation based on hemodynamic decompensation should not be delayed, regardless of the underlying source of the bleeding.

Treatment

Stabilization And Monitoring

Initial resuscitation of patients with massive GI bleeding involves restoration of intravascular volume in order to achieve the relative hemodynamic stability required to proceed with diagnostic and therapeutic interventions. An initial primary survey to evaluate the airway, breathing, and circulation should almost always include endotracheal intubation in order to protect the patient’s airway in the setting of active hemorrhage and to assist in identifying the bleeding source and facilitating these additional interventions. Early resuscitation may start with 2 large-bore peripheral intravenous (IV) catheters, but resuscitation of massive hemorrhage may be aided by the use of larger, introducer-sized central venous catheters that can more efficiently restore lost intravascular volume during hemorrhagic shock.

The importance of early mobilization of specialists cannot be overemphasized. In patients with massive GI bleeding, medical therapy alone is unlikely to be sufficient to achieve hemostasis. Early mobilization of the available subspecialists expedites time to definitive therapy. The lack of available subspecialty support should prompt consideration for transfer to a tertiary care facility that can provide this care. Depending on the underlying cause of the bleeding as well as patient comorbidities, gastroenterology, general surgery, and interventional radiology may all play a role in the care of these patients.
Patients with massive GI bleeding require continuous cardiac and pulse oximetry monitoring, with frequent blood pressure checks. Central venous access may be required in the ED if the patient has poor peripheral access or if more the rapid infusion of fluids or blood products is needed. Invasive monitoring, including an arterial line, will enable prompt recognition and response to the rapid changes in physiology that may occur in massive GI hemorrhage.

Initial laboratory values can help guide initial therapy, but serial coagulation studies in the ED are not likely to aid in decision making and may be difficult to interpret in patients being treated with fresh frozen plasma (FFP), cryoprecipitate, or factor concentrate. Likewise, an initial D-dimer may offer prognostic information, but it should not influence immediate management in the acute setting.

Regardless of the source of upper GI bleeding, proton-pump inhibitors (PPIs) are typically recommended until the etiology of the bleeding can be identified. While PPIs will not impact the immediate stabilization of a hemodynamically unstable bleeding patient, their use may improve outcomes.

In cases of nonvariceal hemorrhage, a bolus of a PPI should be initiated and then followed by a continuous infusion. This is thought to promote ulcer healing and decrease the rate of rebleeding. Additionally, in cases of variceal hemorrhage with subsequent endoscopic intervention, patients who are given PPIs intravenously have demonstrated less rebleeding because PPIs work against stomach acid and pepsin, which are thought to destabilize platelet plugs and the hemostatic clot.

**Procedural Therapies**

**Endoscopy**

Endoscopy is an important diagnostic and therapeutic tool for upper GI hemorrhage and should be performed promptly upon patient stabilization. Endoscopy provides definitive therapy in many cases. The gastroenterologist may employ various endoscopic techniques, including clips, banding, thermocoagulation, or sclerosant injection with or without epinephrine. All patients with massive GI bleeding should be intubated to facilitate endoscopy, control the airway, reduce the risk of aspiration.

### Table 1. Current Guidelines For Gastrointestinal Bleeding

<table>
<thead>
<tr>
<th>Organization</th>
<th>Topic</th>
<th>Type of Guideline</th>
<th>Recommendations</th>
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| American College of Physicians            | Management of patients with nonvariceal upper GI bleeding              | Consensus         | 1. Early evaluation, resuscitation, and risk stratification is recommended.  
2. Nasogastric tube placement may have diagnostic value.  
3. Transfuse PRBCs to maintain hemoglobin > 7 gm/dL.  
4. Correction of coagulopathy is recommended but should not delay endoscopy.  
5. Pre-endoscopic PPIs may be considered.  
6. Early endoscopy (within 24 h) is recommended.  
7. Histamine-2 receptor antagonists are not recommended for patients with acute bleeding due to a gastric or duodenal ulcer.  
8. Somatostatin and octreotide are not routinely recommended for patients with acute ulcerative bleeding. |
| Cochrane Library                          | Pharmacologic management of patients with GI bleeding                 | Systematic review  | 1. Somatostatin analogues do not reduce mortality in variceal GI bleeding; however, they may reduce the need for blood transfusion by 0.5 units.  
2. PPI administration prior to endoscopy for upper GI bleeds significantly reduces the number of patients with recurrent serious bleeding and the need for endoscopic intervention, but they have no effect on the need for surgery or risk of death. |
| Scottish Intercollegiate Guidelines Network| Guidelines for initial treatment of patients with significant GI bleeding | Systematic review  | 1. PRBC transfusion should be considered after loss of 30% of the circulating blood volume.  
2. PPIs should not be used prior to endoscopic diagnosis in patients presenting with an acute upper GI bleed.  
3. Early endoscopic examination should be undertaken within 24 h of initial presentation, if possible.  
4. Prior to endoscopic diagnosis, terlipressin (vasopressin analogue) should be given to patients suspected of variceal hemorrhage.  
5. Antibiotic therapy should be initiated in patients with chronic liver disease who present with acute upper GI bleeding.  
6. Balloon tamponade should be considered as a temporizing measure to gain hemostasis in uncontrolled variceal hemorrhage. |

**Abbreviations:** GI, gastrointestinal; PPI, proton-pump inhibitor; PRBC, packed red blood cell.
Additional Procedural Therapies
In severe cases of massive GI bleeding, where urgent endoscopy is unable to provide a diagnosis or therapy, urgent or emergent angiography can be performed to identify the location of the hemorrhage. Angiography is indicated in patients with nonvariceal upper GI bleeding when medical management and endoscopic therapy have failed to control the bleeding. Angiographic intervention should be considered as an alternative to surgery in patients who are high risk for more invasive surgical intervention. Angiography can detect bleeding at rates of at least 0.5 mL/min with 100% specificity and variable sensitivity (based on the rate). During angiography, lesions can be treated by use of vasopressin or transcatheter embolization.

Another option for identifying the source of bleeding is radionuclide imaging, which can detect bleeding occurring at slower rates than can be detected by angiography. The primary purpose of these studies is to localize the source of the bleeding and to facilitate surgical intervention. An emergency surgical consult should be sought in patients with massive hemorrhage who cannot be stabilized in the ED, who have contraindications to endoscopy, or who have peritoneal signs on examination (which can occur from a ruptured peptic ulcer). While surgery is typically only considered as a last resort after medical, endoscopic, and interventional radiology approaches have been exhausted or are contraindicated, not all types of bleeding (such as variceal bleeding) are amenable to surgical intervention. Nonetheless, early involvement of surgery and gastroenterology services can facilitate a cooperative treatment approach and may make surgical intervention, if necessary, safer and less emergent.

Pharmacologic Therapies
There are multiple proposed pharmacologic treatments for the management of acute upper GI bleeding. Understanding the mechanism of action of these

Figure 1. Minnesota Tube For Balloon Tamponade
Note both the gastric and esophageal balloons. Types of tubes vary slightly. Be familiar with the type available in your institution.

Image courtesy of Ryan G. K. Mihata, MD
medications can facilitate a deeper understanding of the rationale for the existing guidelines for their use.

**Proton Pump Inhibitors**

PPIs, which include omeprazole, pantoprazole, and esomeprazole, are considered to be a mainstay of early therapy for acute GI bleeding in the ED. PPIs suppress gastric acid stimulation by inhibiting the parietal cell H+ / K+ ATPase (adenosine triphosphatase) pump. One potential benefit of the early use of PPIs may be the reduction of hemorrhage during endoscopy. Additionally, PPIs may create a more optimal environment for the stabilization of blood clots by neutralizing the intraluminal gastric acid that would otherwise serve to promote continued bleeding.

There has been concern about the potential increased risk of thrombotic stroke or myocardial infarction with the addition of PPIs to long-term clopidogrel therapy. Based on cohort studies, a 2009 United States Food and Drug Administration warning cited a 50% increased risk of these complications when PPIs and clopidogrel were taken concurrently. However, current data are inconclusive and suggest that these complications are typically associated with long-term use of PPIs and clopidogrel. These risks have not yet been described in the acute, short-term use of these medications during the stabilization of patients with massive GI bleeding.

At this time, guidelines continue to encourage the use of PPIs (either before or immediately following upper endoscopy) in patients with undifferentiated upper GI bleeding. However, there has not yet been a study showing a statistical improvement in the rate of rebleeding, the need for surgical intervention, or mortality from their use. Nonetheless, their cost effectiveness and excellent safety profile make the benefit of giving these agents greater than the potential risks, particularly in high-risk patient populations.

**Somatostatin**

Somatostatin and its analogues (including octreotide and vaptreotide) represent another class of pharmacologic agents that may be used early in the management of acute GI bleeding due to a suspected variceal source. Somatostatin (also known as “growth-hormone-inhibiting hormone”) suppresses the release of GI hormones such as gastrin, cholecystokinin, motilin, and secretin. The secondary effects of blocking the release of these hormones include a reduction of portal venous blood flow due to splanchnic vasoconstriction and subsequent decreased GI bleeding.

To date, there are minimal data regarding the use of somatostatin or its analogues for nonvariceal upper GI bleeding. The data on their use in severe variceal bleeding are also mixed. A Cochrane review found no significant change in mortality, the rebleeding rate, or the number of units of blood transfused with the use of somatostatin in patients with acute bleeding from esophageal varices. Some data suggest that the combination of these medications with upper endoscopy may be beneficial.

Given their relatively low-risk side-effect profiles, the use of these medications can be considered in patients with acute hemorrhage that is possibly caused by variceal bleeding.

**Vasopressin**

A third class of pharmacologic agents, vasopressin and its analogues, can reduce portal hypertension by causing systemic and splanchnic vasoconstriction. Much like somatostatin, the use of vasopressin in the management of upper GI bleeding is controversial. The risks (eg, myocardial infarction, arrhythmias, mesenteric ischemia, peripheral soft tissue necrosis, cardiac arrest) of using this medical therapy are similar to—but usually less severe than—those seen with the use of other vasopressor medications. In patients with variceal bleeding, adding nitroglycerin to vasopressin therapy appears to decrease systemic complications from the use of vasopressin alone while not affecting the efficacy of the vasopressin. In patients with massive GI bleeding, the risk of systemic vasoconstriction and the potential for end-organ ischemia must be weighed against the potential benefit of splanchnic vasoconstriction, which can help to achieve hemostasis in the actively bleeding patient.

In spite of the potential deleterious effects of vasopressin, use of vasopressin (or vasopressin plus nitroglycerin) in a patient in extremis due to massive GI bleeding may be justified.

**Blood Product Transfusion**

The goal of blood product transfusion for acute bleeding is to replace the blood cells and coagulation factors that are being lost. Whole blood is a complex heterogeneous solution that serves 3 basic functions: (1) to maintain intravascular volume, (2) to provide oxygen-carrying capacity, and (3) to provide the coagulation factors that are required to reverse coagulopathy and to assist in achieving (and maintaining) hemostasis. The ability to achieve hemostasis can be complicated by the use of anticoagulation or antiplatelet agents. Because there are inherent risks involved with transfusing blood products, the risks must be considered along with the potential benefits for each individual patient.

**Goal #1: Provide Intravascular Volume Resuscitation**

Prompt volume resuscitation with IV crystalloid fluids should be initiated in a bleeding, hypotensive patient in order to achieve a perfusing mean arterial pressure (MAP) until blood products are available. Given that it lacks oxygen-carrying capacity and does not replenish clotting factors, the role of crystalloid fluid for volume resuscitation should ideally

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be restricted to this early phase of resuscitation in a bleeding patient. In cirrhotic patients with suspected variceal hemorrhage, excessive crystalloid resuscitation may be particularly harmful due to their susceptibility to extravascular fluid shifts in the setting of underlying low intravascular oncotic pressure.

In patients with massive GI bleeding (especially those with hemoglobin < 8.0 gm/dL, coagulopathy, or persistent hypotension despite volume infusion), the use of blood products (such as PRBCs) is important in order to restore hemodynamic stability and to achieve hemostasis. However, transfusion of PRBCs alone is not sufficient because they do not contain the required coagulation factors. For this reason, PRBCs should be accompanied by FFP and/or platelets. The military literature on traumatic blood loss recommends a PRBC:FFP:platelet ratio approaching 1:1:1.19,20 Other sources disagree on the exact ratio, citing a lack of a specific survival benefit to a higher PRBC to FFP ratio.21 Many large centers have massive transfusion protocols that allow the rapid release of blood products in varying ratios. While the survival benefit remains unclear, most protocols have PRBC:FFP:platelet ratios near 1:1:1.

Goal #2: Optimize Oxygen-Carrying Capacity

The goal of improving oxygen-carrying capacity by transfusing PRBCs is to ensure sufficient oxygen delivery to the tissues in order to prevent end-organ ischemia and secondary dysfunction. Clinical signs and symptoms of decreased oxygen delivery can include:

- Decreased level of consciousness
- Evidence of cardiac ischemia (as shown by electrocardiographic changes or troponin elevation)
- Increasing serum lactate
- Decreased central venous oxygen saturation (ScvO2)

The International Consensus guidelines recommend maintenance of hemoglobin levels between 7 and 8 gm/dL.3 However, there are no randomized controlled trials to suggest an exact hemoglobin goal for transfusion. Target hemoglobin levels should be individualized for each patient and should be based upon the suspected etiology of the bleed (ie, variceal vs nonvariceal hemorrhage) as well as patient comorbidities. In the setting of ongoing massive bleeding, the recommended hemoglobin level of 7 to 8 gm/dL should not be considered to be an endpoint of resuscitation. Instead, a MAP of > 60 mm Hg and evidence of adequate end-organ perfusion should be the target.

Though the International Consensus guidelines suggest maintenance of hemoglobin between 7 and 8 gm/dL, patients with certain comorbidities may require greater hemoglobin goals. For instance, evidence suggests that patients with suspected active ischemic coronary artery disease may benefit from a target hemoglobin closer to 10 gm/dL to support myocardial oxygen delivery.22 However, for patients with suspected variceal bleeding (even with concomitant coronary artery disease), consider keeping the hemoglobin goal to between 7 and 8 gm/dL until hemostasis is achieved, as the additional blood volume required to achieve this higher target may lead to increased portal pressure and worsening bleeding or rebleeding.

When there is a need for emergent transfusion of PRBCs for acute blood loss anemia with signs of hypovolemic shock, O-negative PRBCs (or O-positive PRBCs in women past childbearing age and in men) can be given until type-specific crossmatched blood is available. For patients requiring massive transfusion of blood products, it is important to remember that the transfusion will have metabolic complications such as depletion of intravascular cations due to chelation by the sodium citrate used to anticoaggregate blood components during storage. Citrate is primarily metabolized by the liver; so in cirrhotic patients receiving massive transfusion, this effect may be more profound. For these patients, consider giving calcium replacement (calcium gluconate 1-2 g IV or calcium chloride 0.5-1 g IV per 500 mL of transfused blood). Magnesium can also be affected, and low levels have unwanted side effects and should be repleted as necessary, keeping in mind that overly rapid infusion of magnesium can exacerbate hypotension.

Goal #3: Reverse Coagulopathy

Treatment of coagulopathy can be tailored to its likely or known etiology. Coagulopathy may arise due to an underlying chronic disorder, but it may also be caused by metabolic acidosis due to tissue hypoxemia in the setting of acute hypotension.

To reverse coagulopathy, consider an initial transfusion of 10 mL/kg of FFP. Prothrombin time (PT)/international normalized ratio (INR), partial thromboplastin time (PTT), platelet count, and fibrinogen levels can be used to guide patient management. While there are no specific guidelines, keeping the platelet count > 50,000/mm3, PTT < 60 seconds, and INR < 1.8 is common practice and is recommended by the authors. Because D-dimer levels have been shown to be inversely related to prognosis in upper GI bleeding, a D-dimer may be drawn as a prognostic marker, but it has no role in guiding intervention.23 If available, the thromboelastography (TEG) test can evaluate platelet function and coagulation and may be more efficient to direct the type of transfusion products necessary.

The role of recombinant activated factor VII (rFVIIa) is continuing to evolve. While it may be useful in certain situations (eg, when a patient is on a direct thrombin inhibitor), it is expensive, it carries the risk of thromboembolic complications, and studies of its use have had conflicting results. A prospective randomized trial of 245 patients found no benefit with rFVIIa over standard therapy for
rate of 1 mg/min is necessary to achieve a sustained reversal of the INR; however, it is insufficient when given as a single agent, as it has an onset of action of 2 to 6 hours and requires up to 24 hours to achieve a complete response. For this reason, FFP must also be administered.

Although controversial, rFVIIa can be considered for reversal of effects of warfarin. However, it is expensive, it has minimal proven therapeutic benefit for warfarin reversal, it is not FDA approved for this use, and its use may cause thrombotic complications.

Dabigatran: Dabigatran (Pradaxa®) is a direct thrombin inhibitor. To quantify the degree of dabigatran coagulopathy, obtain a PTT and thrombin time (if rapidly available). PT/INR is not useful in assessing dabigatran coagulopathy. The PTT is only useful to qualitatively assess the degree of coagulopathy because it lacks sufficient sensitivity with therapeutic dabigatran levels. A very high PTT value should be confirmed by another method (eg, thrombin time), although this may be impractical in the emergent setting. Like other anticoagulant medications, even therapeutic levels may require reversal in the setting of active bleeding.

At this time, an evidence-based reversal strategy for dabigatran is not available. Many institutions have a pathway for bleeding patients who are on dabigatran that may inform local practice patterns. Management strategies may range from expectant management to aggressive use of blood products, including FFP and even hemodialysis.

Rivaroxaban: Rivaroxaban (Xarelto®) is a factor Xa inhibitor. Administer PCCs for reversal of rivaroxaban; if PCCs are not available, consider administration of FFP.

Reversal Of Medically Induced Coagulopathy

**Warfarin:** Warfarin (Coumadin®, Jantoven®) inhibits vitamin K-dependent coagulation factor synthesis. Prothrombin complex concentrates (PCCs) should be the first-line treatment in a patient who is on warfarin. PCCs do not require crossmatch and act more quickly than FFP alone; however, because PCCs in the United States do not contain significant amounts of factor VII, FFP must also be administered. Dosages are individually calculated based on weight and desired factor IX level; 1 unit/kg will increase the factor IX level approximately 1%.

If PCCs are not available, coagulopathy can be reversed by discontinuing warfarin therapy and administering vitamin K. Vitamin K at a dose of 5 to 10 mg diluted in D5W or D5 saline infused at a

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**Figure 2. Coagulopathy Chart**

Clinical Pathway For The Initial Management Of Patients With Massive Upper Gastrointestinal Bleeding

Initial actions: insert 2 large-bore IVs (Class I); establish monitoring (Class I); provide a PPI bolus & drip (Class I); consider insertion of a nasogastric tube (Class II); provide a crystalloid bolus (Class II); and keep the patient warm (Class II).

Are there signs of altered mental status and/or active hematemesis?

NO

Intubate (Class II)

YES

Are there signs or symptoms of shock?

NO

Transfuse PRBCs, FFP, and platelets (Class II)

YES

Is there a history of esophageal varices or signs of cirrhosis?

NO

Administer:

Octreotide 50-mcg bolus and 50-mcg/h infusion (Class II)

and

Ciprofloxacin 400 mg bid (Class I) or ceftriaxone 1g IV qd (Class II)

• Transfuse 2 units FFP (Class II)
• If there is severe liver disease with coagulopathy and refractory bleeding, consider PCCs or rFVIIa (Class II)
• If the patient is taking warfarin, add vitamin K 10 mg slow IV push (Class II)

Are there signs of altered mental status and/or active hematemesis?

NO

Intubate (Class II)

YES

Transfuse PRBCs, FFP, and platelets (Class II)

YES

Is there a history of esophageal varices or signs of cirrhosis?

NO

Administer:

Octreotide 50-mcg bolus and 50-mcg/h infusion (Class II)

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Ciprofloxacin 400 mg bid (Class I) or ceftriaxone 1g IV qd (Class II)

If there is severe liver disease with coagulopathy and refractory bleeding, consider PCCs or rFVIIa (Class II)

If the patient is taking warfarin, add vitamin K 10 mg slow IV push (Class II)

Coagulopathy (INR > 1.7 with active bleeding)

Anemia

• If hemoglobin < 7.0 gm/dL: transfuse PRBCs (Class II)
• If hemoglobin > 8.0 gm/dL (or with bleeding varices): do not transfuse PRBCs (Class II)

Abbreviations: ABG, arterial blood gas; bid, two times a day; BMP, basic metabolic panel; CBC, complete blood count; CXR, chest x-ray; ECG, electrocardiogram; FFP, fresh frozen plasma; INR, international normalized ratio; IV, intravenous; LFT, liver function test; PCCs, prothrombin complex concentrates; PPI, proton-pump inhibitor; PRBCs, packed red blood cells; PT, prothrombin time; PTT, partial thromboplastin time; qd, 1 time a day; rFVIIa, recombinant activated factor VII.

Class Of Evidence Definitions

Each action in the clinical pathways section of EM Critical Care receives a score based on the following definitions.

Class I
• Always acceptable, safe
• Definitely useful
• Proven in both efficacy and effectiveness

Level of Evidence:
• One or more large prospective studies are present (with rare exceptions)
• High-quality meta-analyses
• Study results consistently positive and compelling

Class II
• Safe, acceptable
• Probably useful

Level of Evidence:
• Generally higher levels of evidence
• Non-randomized or retrospective studies: historic, cohort, or case control studies
• Less robust randomized controlled trials
• Results consistently positive

Class III
• May be acceptable
• Possibly useful
• Considered optional or alternative treatments

Level of Evidence:
• Generally lower or intermediate levels of evidence
• Case series, animal studies, consensus panels
• Occasionally positive results

Indeterminate
• Continuing area of research
• No recommendations until further research

Level of Evidence:
• Evidence not available
• Higher studies in progress
• Results inconsistent, contradictory
• Results not compelling


This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient’s individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Unfractionated Heparin: Unfractionated heparin acts at multiple sites in the clotting cascade and serves as a catalyst for clotting factors. Administer protamine sulfate to reverse heparin.

Clopidogrel: Clopidogrel (Plavix®) is a nonreversible platelet inhibitor. Empiric transfusion of platelets can be considered for patients on clopidogrel or other platelet inhibitors (such as aspirin), although no randomized controlled trials have demonstrated a favorable impact with this approach.

Special Circumstances

When treating a patient who refuses blood based on religious beliefs (eg, Jehovah’s Witnesses), it is important to ask the patient or legal guardian whether they are willing to receive blood products. Even though the patient may be a Jehovah’s Witness, it is important to carefully establish and document the patient’s current medical wishes. In a small case study, investigators found a portion of a Jehovah’s Witness congregation who would accept blood transfusions because they disagreed with the official doctrine. Further, The Watchtower, a legal organization of the Jehovah’s Witnesses, asserts with respect to blood transfusion: “Ultimately, you (the patient or the parent) must decide. Because your (or your child’s) body, life, ethics, and relationship with God are involved.” Regardless of the patient’s or power of attorney’s decision, carefully document conversations about risk and benefit as well as the subsequent management based on that decision.

Disposition

Most patients who present to the ED with massive GI bleeding will ultimately require admission to the intensive care unit, although this will depend on the success of early intervention and the clinical stability of the patient. Consideration should be made early for involvement of subspecialists. Facilities without gastroenterology, general surgery, or interventional radiology backup should have a rapid interfacility transfer protocol in place to expedite patient transfer to a facility capable of providing the definitive management that will be needed after the initial resuscitation and stabilization.

Summary

The unstable patient with massive upper GI bleeding is a high-risk case for the emergency physician, requiring the coordination of resources and consideration of various treatment modalities. Initial resuscitation efforts should occur concomitantly, with diagnostic measures aimed at finding the etiology of the bleed.

The foundation of the resuscitation includes initial management of the airway, breathing, and circulation. Volume resuscitation requires crystalloid fluids and transfusion of blood products. Early consultation with gastroenterology, interventional radiology, and general surgery will improve the patient’s time to definitive care. Endoscopy should be performed promptly upon stabilization. In addition to volume resuscitation, medical therapies such as PPIs, somatostatin and its analogues, and vasopressin should be considered either empirically based upon hemodynamic stability of the patient or directed at the underlying etiology of the bleeding, if known. Surgical intervention may ultimately be necessary if less-invasive treatments fail or are contraindicated. Depending on the clinical scenario, balloon tamponade may be needed as a temporizing and life-sustaining measure until the resources required for definitive therapy become available.

Case Conclusion

The critically ill 45-year-old male patient arrived to your resuscitation bay with vital signs that were unchanged from the prehospital report. You made a decision to intubate the patient in order to protect his airway and to facilitate anticipated upper endoscopy. Shortly after intubation, the patient continued to have frank blood coming from his oropharynx. The nurses established a second large-bore peripheral IV, and emergency release blood was given. Each large-bore peripheral IV had blood infusing, and the initial hemoglobin came back at 5.8 g/dL. You ordered 4 more units of type-specific blood as well as 2 units of FFP and 2 single-donor units of platelets, and you subsequently placed an introducer catheter to expedite large volume infusion of blood products via a level 1 infuser, which warmed the infusing fluid. The gastroenterologist came and performed an endoscopy in the ED, during which he attempted to band the large esophageal varices. He was uncertain whether or not he was able to obtain complete hemostasis during the procedure. The patient initially improved with your resuscitation but continued to have a MAP in the 50s and a pulse rate in the 120s. You administered an octreotide bolus and infusion as well as a PPI infusion. In consultation with your intensivist team, you started a vasopressin drip with a goal MAP of > 60 mm Hg. This improved not only the variceal bleeding but also the patient’s hemodynamic stability. In total, the patient required 4 more units of PRBCs and 2 more units of FFP. He was admitted to the ICU, where the gastroenterologist repeated an endoscopy and found less blood but 2 more bleeding varices, which were subsequently banded. The patient developed renal failure and liver failure (acute on chronic) with ischemic hepatitis secondary to hemorrhagic shock. He had a prolonged course in the ICU but eventually improved and was ultimately discharged from the hospital.
Must-Do Markers Of Quality Care

- Control the airway by endotracheal intubation prior to endoscopy.
- Establish central venous access after inserting 2 large-bore peripheral IVs and utilizing an introducer to facilitate high-volume infusions and central venous pressure monitoring. Consider the internal jugular vein as the location for central line placement in coagulopathic patients due to vascular compressibility. Line placement with ultrasound guidance is the standard of care.
- Initiate volume resuscitation with crystalloid fluids and O-negative blood (if available). Switch to type-specific crossmatched blood as soon as it becomes available.
- Consider transfusion of other blood products or infusions of medications based on the suspected etiology of the bleed and the patient’s comorbidities.
- Aggressively treat hypothermia and acidosis in order to facilitate correction of coagulopathy.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.


Accessed March 1, 2013.
1. The most common cause of massive upper GI bleeding is:
   a. Esophageal varices
   b. Diverticula
   c. Arteriovenous malformations
   d. Gastritis
   e. Gastric and duodenal ulcers

2. In regard to the stabilization and monitoring of patients with massive upper GI bleeding, which of the following is TRUE?
   a. PPIs are not recommended.
   b. Initial and serial coagulation studies should be used to guide decision making.
   c. Continuous cardiac and pulse oximetry monitoring are required.
   d. Consultation with subspecialists should be delayed until after stabilization and resuscitation.

3. In regard to the use of angiography in patients with massive upper GI bleeding, which of the following is TRUE?
   a. Angiography is highly specific for detection of bleeding at rates of at least 0.5 mL/min.
   b. Angiography can detect bleeding at slower rates than can be detected by radionuclide imaging.
   c. Angiography has the disadvantage of being diagnostic but not therapeutic.
   d. Angiography is contraindicated in patients with nonvariceal upper GI bleeding.

4. In regard to blood product transfusion for patients with massive upper GI bleeding, which of the following is TRUE?
   a. A hemoglobin level of 7 to 8 gm/dL should be considered the endpoint of resuscitation.
   b. The recommended PRBC:FFP:platelet ratio is 1:2:1.
   c. Use of crystalloids should ideally be restricted to the initial phase of resuscitation.
   d. Calcium gluconate should be administered to all patients receiving PRBCs.
**CME Information**

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**Target Audience:** This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents as well as intensivists and hospitalists.

**Goals:** Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentation; and (3) describe the most common medicolegal pitfalls for each topic covered.

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