EM:RAP C3 April 2016 Written Summary

Massive GI Bleed

Mizuho Spangler DO, Stuart Swadron MD, Jess Mason MD, and Mel Herbert MD

** Drug doses are a guide only, always check second source and follow local practice guidelines*

Take Home Points:

- **A-B-C**
- **If unstable assume UPPER GI Bleeding**
- **Intubate early for control and help with Dx**
- **Replace blood with blood**
- **Reverse Bleeding disorders**
  - **Vit K if INR high (eg: 10 mg IV),**
  - **DDAVP (0.4mcg/kg IV over 10 minutes) if plt or renal disorder**
- **PPI eg: Pantoprazole 80 mg IV, then 8 mg/hr**
- **Octreotide eg: 50 mcg IV bolus**
- **Call GI stat**

- **Consider:**
  - **Early Scope**
  - **Antibiotics eg: Ceftriaxone 1 gram IV**
  - **Linton Tube or similar**
  - **Call Surgery if needed**
  - **Call Interventional Radiology if needed**

Introduction

The unstable patient with massive gastrointestinal (GI) bleeding is one of the most dramatic we will encounter in the ED. Patients with underlying liver disease are especially

at risk. Patients on cardiac medications, such as beta-blockers, may not demonstrate the tachycardia that is commonly seen in hemorrhagic shock. Once identified these patients warrant aggressive resuscitation with control of the airway, stabilizing hemodynamics, and attempts to control ongoing bleeding. At the same time, we coordinate efforts to bring in consultants who can get these patients the definitive care they need.

Confirming the source

First let's quickly discuss the source of blood and briefly mention the mimics of a GI bleed. When someone has a massive amount of blood coming from the rectum, it is pretty obvious, that the source is gastrointestinal. However when blood is coming from the mouth, it can sometimes be tricky differentiating hematemesis (from the upper GI tract) from other sources of bleeding, such as epistaxis (nosebleed) or hemoptysis (from the lungs). Here are a few identifying features of each:

- **Hematemesis:** usually large volume, with vomit, coffee ground/brownish, thick/chunky.
- **Hemoptysis:** usually smaller/lesser volume, with cough, bright red blood, thin and frothy.
- **Epistaxis:** volume is variable (still boluses are < hematemesis), thin/bright red +/- clots. Can be seen dripping down posterior oropharynx.

The fact of the matter is that in the vast majority of cases, it's going to be very obvious. And in those patients in whom it is not, if they are really unstable, you're going to be intubating them soon regardless. Then if there was any question of the source, once you have the tube in the trachea, if should resolve the issue of where the blood is coming from.

Massive vs. Minor GI bleed

Severe gastrointestinal hemorrhage can be quantified by objective criteria which include:

- an acute marked decrease in hemoglobin
- vital sign changes consistent with hemorrhagic shock
- copious amounts of hematemesis and/or hematochezia

These objective measures may be preceded by a toxic appearance and changes in mental status.

Upper vs. Lower:

There are two major classifications of GI bleed: Upper and Lower GI bleed

- **Upper GI bleed:** typically originates proximal to the ligament of Treitz in the distal duodenum. Most common sources of UGIB (in order of frequency) are: duodenal ulcers, gastric ulcers, gastritis, esophageal varices and esophagitis [1].
- **Lower GI bleed:** bleeding starts distal to the ligament of Treitz. Most common causes (in order of frequency) are diverticula, arteriovenous malformations, polyps and cancer [1].

It is fairly obvious when someone is vomiting bright red blood that it's an upper GI bleed. Confusion can arise however, when when the blood is coming from rectum. When blood is dark, tarry and malodorous we call that melena. It has a very distinguishable smell because it is blood that has been digested and passed through the gut - so it's usually from upper GI source. Sometimes, an upper GI bleed is so brisk, that no digestion has had time to occur, and so you have upper GI bleed that presents with bright red blood per rectum (hematochezia). That is something to consider if bleeding is really brisk.

This leads to our whole clinical approach to severe GI bleeding. We initially need to focus on the **upper sources** first for a number of reasons: they're more common, they tend to bleed brisker, they're more life threatening, and there's more that we can actually do about them in the ED.

Initially, focus your efforts and presume bleeding is from an UGIB source, and here's why:

- An upper source is responsible for 76%-90% of all GIB [1, 2]
- UGIB has a poorer prognosis and higher mortality
- Peptic ulcers (duodenal and gastric) represent over half of UGIB and 60% of patients that die from GIB do so from esophageal varices or peptic ulcers [3].

- (LGIB): Lower GI bleed
  - In contrast to UGIB, the overall mortality for LGIB is comparatively low at 4%.

- Even in cases of seemingly "clear cut" LGIB with frank hematochezia, over 10% will have an upper source as well [4].

BOTTOM LINE: when you are dealing with a patient who is hemodynamically unstable, it is safest to first consider an upper source of GIB. Specifically we really need to focus on two critical diagnosis: esophageal varices and bleeding peptic ulcers. Those are the two most common causes of life-threatening GI bleeding.

MANAGEMENT

Management for the unstable GI bleeder is complicated, but as with everything in emergency medicine a stepwise systematic approach is always best.
"Airway-breathing-circulation" and "IV-O2-Monitor" remains our starting points.

Airway/Breathing:

Early intubation with massive GI bleed has several advantages:

1. It helps you diagnose the source of bleed (i.e.: GI source vs. other)
2. Helps prevent aspiration (hematemesis = high risk for aspiration)
3. Gives you control of the critically ill patient and allows you to monitor bleeding (via NG tube)

Key points to remember when intubating these ill patients:

- Remember to pre-oxygenate these patients, as they will have little O2 reserve. You want to limit the amount of bag valve mask ventilation required, as this leads to abdominal distention, which increases the risk of aspiration, and preoxygenation helps limit BVM use.
- RSI: choose a hemodynamically stable drug (i.e.: Swadron prefers etomidate; Miz likes ketamine).

Circulation:

So now let's talk about "**C**"irculation. Patients in hemorrhagic shock secondary to a GIB may fail to mount tachycardia. In fact, patients with GIB may actually present with paradoxical bradycardia for a few reasons:

1. Primary or secondary prophylaxis with beta blocker therapy and/or nitrate [5], which will invariably blunt a normal tachycardic response to acute shock and lower baseline blood pressures.

2. Vagal response - more common with intra-abdominal hemorrhage

Although resuscitation may begin with crystalloids, blood must be transfused as quickly as possible in all patients with massive GIB. Instituting your hospital's massive transfusion protocol is critical for optimal resuscitation. Initially when there is no time to cross-match blood, you should start with type O, the universal donor. Specifically, O - in any female of childbearing age, and O+ in any male patient. As the resuscitation proceeds, you can get more type-specific blood. Using your institutions' massive transfusion protocol, especially in patients with end stage liver disease is key, so that you don't just limit transfusion to packed red blood cells, but plasma and platelets as well.

Bleeding disorders

In patients who have an underlying coagulopathy, it doesn't make sense just to simply replace their blood lost. They will continue to bleed until the underlying coagulopathy needs to be addressed. The key is to look for what their underlying etiology for coagulopathy is, and use this to direct treatment. Here are a few examples:

- **End stage liver disease:** patients are the classic example. These patients warrant IV vitamin K supplementation to help clot their blood.
- **Thrombocytopenia:** If they've got a known platelet problem and their platelets are low, it is important to transfuse platelets.
- **End stage kidney disease:** patients with renal failure will clot better when we give them the medication DDAVP.

Endpoints of resuscitation...how much is too much?

As we aggressively resuscitate these patients, some experts have argued that overzealous resuscitation may lead to worsening of the GI bleeding. The theory is that when we go above and beyond what they have lost, we continue to increase the blood pressure higher than baseline, and this makes the bleeding worse. Truth is, there is very little data to recommend a particular target BP in the setting of hemorrhagic shock from GIB. A reasonable goal for resuscitation is evidence of adequate perfusion of vital organs (e.g. mental status and urine output), rather than a particular blood pressure cutoff.

Complications:

We need to be mindful of any evidence of end organ dysfunction, which may result as a consequence of hemorrhagic shock and complicate management. Things to take into consideration along the resuscitation include:

- **Cardiac ischemia:** consider obtaining an ECG, due to increased cardiac demand.
- **Renal failure:** Decreasing urine output and elevated blood urea nitrogen and creatinine may indicate stress on the kidneys.
- **Acute Respiratory Distress Syndrome (ARDS):** can be triggered from either aspiration or simply due to massive transfusion. Decreasing oxygen saturation and changes in pressure on the ventilator are signs for this. Consider monitoring with chest Xray if you note increased oxygen demand.

Nasogastric (NG) Tube:

Over the years, the use of routine nasogastric tube in patients with GIB has come into question. Traditionally it was used as a diagnostic tool to help distinguish UGIB from LGIB. However we know that one major disadvantage of NGT use is patient discomfort, which is less of an issue when the patient is intubated and sedated. Major advantages of use of an NGT in intubated patients at risk for concomitant hepatic encephalopathy or hepatorenal syndrome is that aspirating blood decreases the adsorbed protein load that exacerbates these conditions. NGT aspiration in patients with severe ongoing losses can also serve as an additional "vital sign". It can provide foreshadowing changes in blood pressure, pulse and hemoglobin measurements. Thus, in the setting of an unstable patient with GIB, monitoring ongoing losses of blood via NGT suction can be one important predictor of impending circulatory collapse. Of note, you should keep in mind that a negative result of NG aspiration by no means, excludes the possibility of an ongoing upper source of bleeding.

Medications:

1. **Proton pump inhibitors (PPIs)** are recommended empirically in patients with suspected UGIB, as they have been particularly helpful names in variceal and peptic ulcer bleeding. There is no evidence that PPIs acutely stop bleeding but they may have a role in decreasing the incidence of rebleeding, lowering transfusion requirements, and reducing need for surgery and hospital length of stay. Because of their low side effect profile and the fact that it is difficult to distinguish between ulcerative versus variceal disease prior to endoscopy, some experts advocate for empiric PPIs in cases of undifferentiated UGIB.

2. **Somatostatin and its synthetic analogue (i.e.: octreotide)** work by decreasing splanchnic blood flow and have been used to slow variceal and nonvariceal bleeding, although admittedly, the evidence for its efficacy in the non-variceal group is weak.

3. **Vasopressin** and its analogue (i.e.: terlipressin) are vasoconstrictors. When used intravenously, serious side effects such as dysrhythmias and systemic vasoconstriction causing cerebral, cardiac, intestinal and extremity ischemia necessitate discontinuation of these agents in 10% of cases. Therefore vasopressin is typically limited for use when other medical treatments are exhausted and there is a high suspicion for variceal bleeding.

4. **Antibiotics** should be routinely administered in suspected variceal bleeding given the high incidence of comorbid infections such as (SBP) spontaneous bacterial peritonitis and as prophylaxis for invasive endoscopic procedures.

Interventions

While managing your patient medically as discussed above, it is equally critical to simultaneously mobilize your consultants for definitive interventional care. This subset of patients are highly unstable and despite aggressive resuscitation are still at high risk for exsanguination if definitive treatment is not obtained.

Endoscopy

Is a first line intervention for the definitive diagnosis and treatment of both variceal and nonvariceal UGIB [6,7,8]. It should be performed emergently in those with life-threatening GIB even if bleeding has been temporarily controlled by medical therapy to establish the diagnosis and prevent rebleeding. The preferred treatment for esophageal variceal bleeding is banding whereas the use of tissue adhesives (obturation) is favored for gastric varices. [9]. Treatment of non-variceal bleeding may include cautery, injection and coagulation. If upper GI tract endoscopy fails to identify a source of bleeding, sources of LGIB should be sought next. Although colonoscopy may be considered as a first-line for definitive diagnosis and treatment of LGIB, without bowel preparation it may be technically difficult [10].

Balloon Tamponade

In instances when there is a time delay to endoscopy (consultant is not in-house etc), yet your patient is literally exsanguinating before your eyes, you will need a temporizing measure to keep them alive. Balloon tamponade is this temporizing measure, which admittedly has its limitations, but can be life-saving nonetheless. Balloon tamponade works by physically filling up a space, and placing pressure to compress bleeding varices in the stomach and esophagus. Hemostatic pressure is applied to the fundus of the stomach (and by extension to the left gastric vein which feeds the esophageal venous plexus) by way of traction with a weight mechanism applied to the balloon.

In terms of the actual procedure, it depends on the type of tube that you are using in your particular ER and everyone should be familiar with what they have have available. Whether it be a Linton tube, or a Sengstaken-Blakemore tube, there are slight differences between them, but the net effect is the same. The goal is to tamponade the bleeding using a balloon.

When placing a balloon tamponade, remember these key pearls:

- Proper position of the gastric balloon must be confirmed, preferably with X-ray, to avoid inflation in the esophagus, which can cause a catastrophic rupture.
- Depending on the kit, inflation requires up to 400-700ml of air or contrast-water media in the gastric balloon.
- Utilization of a tube clamp, or other securing mechanism, may be necessary to prevent deflation as the filling syringe will have to be removed several times.
- Once filled, traction should be initiated with a 1 L saline bag and may need to be adjusted, based on the clinical results.
- Confirmation of tube placement should be checked initially if possible and intermittently thereafter with X-ray to ensure that migration has not occurred.
- Shears should be readily available to emergently decompress the tube in case of dislodgment.

Balloon tamponade has a reported success of 60 - 90% and a 50% rate of re-bleeding when used alone. Complications include displacement of the tube causing respiratory obstruction or esophageal, tracheal or duodenal rupture.

Other Treatment Modalities:

In cases of variceal bleeding with failure of endoscopic banding, an alternative procedure known as **transjugular intrahepatic portosystemic shunt (TIPS)** can be performed. TIPS essentially works by passing a catheter through the internal jugular vein to the hepatic vein, and then creating a shunt from the hepatic vein to the portal vein (through the liver parenchyma) thereby lowering portal pressure.

Angiographic techniques

Can also be used when endoscopy is unavailable, unsuccessful or contraindicated. Like endoscopy, angiography offers both diagnostic and therapeutic capability. It is also considered first line treatment in patients with suspected biliary or pancreatic bleeding. [8].Sclerotherapy can be performed during angiography using intra-arterial vasopressin or embolizing agents. Lesions particularly amenable to this treatment include gastric ulcers and Mallory-Weiss tears.

In cases of LGIB, angiography is typically considered a second line therapy but it is an important alternative in cases when colonoscopy is unavailable or fails. CT angiography is emerging as a potential modality in severe GIB and given its availability and ability to diagnose other surgical pathology, may have a role particularly when UGIB has been ruled out and another source is being sought [11,12].

Surgery

Historically, surgery played a major role in the management of GIB. Advances in endoscopic and angiographic techniques have reduced this role, but surgery continues to play a part in the definitive management of approximately of 10% of patients with GIB . In many cases of LGIB, surgery may nonetheless still be necessary as definitive therapy after bleeding is stopped with less invasive techniques.

Emergent surgical intervention for variceal hemorrhage is rare and occurs only after nonoperative techniques (ligation and TIPS) have failed. With respect to LGIB, surgery is typically reserved for cases in which a surgical lesion has been diagnosed.

Indications to obtain an emergent surgical consultant include:

- Any patient with peritonitis/surgical abdomen
- Failure of resuscitative measures and continuous massive bleeding
- Failure of medical/endoscopic/angiographic therapy.

Recognizing that managing an unstable patient with massive GI bleed requires a concerted effort by the treatment team for optimal outcome. We must work quickly, anticipate and troubleshoot complications and work with our GI and surgical colleagues to get these patients the definitive care that they need.

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