Lower GI bleeding (LGIB) afflicts 20 to 27 of every 100,000 persons annually in the United States. The rate of LGIB increases more than 200-fold from the third to the ninth decade of life, with 25% to 35% of all cases occurring in elderly patients. It is one of the common medical emergencies that can become life-threatening in elderly patients.

Risk stratification for LGIB by Strate et al. has identified seven predictors of severe bleeding: heart rate higher than 100 beats/min, systolic blood pressure lower than 115 mm Hg, syncope, nontender abdominal examination, gross rectal bleeding, aspirin use, and more than two comorbid conditions. Patients with more than three risk factors have an 84% risk for severe bleeding, defined as transfusion of more than 2 units of red blood cells.

Pediatric GI bleeding is fairly common worldwide; however, the incidence of severe GI bleeding in U.S. children is very low. LGIB is a more common complaint in the practice of general pediatrics, and it accounts for 10% to 15% of referrals to a pediatric gastroenterologist.

In most children, bleeding is not life-threatening and ceases spontaneously, with only supportive care being required. The age of the child guides the clinician toward specific diagnoses.

### PATHOPHYSIOLOGY

#### UPPER GASTROINTESTINAL BLEEDING

Upper GI bleeding (UGIB) is defined as bleeding from a source proximal to the ligament of Treitz, which is located at the junction of the duodenum and jejunum. UGIB accounts for three quarters of cases of GI tract hemorrhage, with duodenal and gastric ulcers being the specific sources in more than half of patients with an upper tract cause.

Hematochezia is generally a symptom of LGIB but may be associated with brisk upper tract hemorrhage. UGIB sources are identified in 11% of patients in whom LGIB was initially suspected. Melena most commonly results from bleeding proximal to the jejunum and should be considered a marker of UGIB.

Variceal hemorrhage is the most serious complication of portal hypertension and occurs in one third of patients with esophageal varices. It is more common in patients with Child B and C cirrhosis. The extent of severe bleeding depends on portal pressure, variceal size, and variceal wall thickness. Esophageal varices should be suspected in any alcoholic with unexplained anemia or obvious GI bleeding.

One study noted a decline in the frequency of peptic ulcer disease in patients with UGIB and reported that the...
Patients with upper gastrointestinal bleeding should be instructed to avoid nonsteroidal antiinflammatory drugs (NSAIDs). Studies have shown that the risk for recurrent bleeding is significantly higher in long-term users of NSAIDs or regular-dose aspirin, especially if patients are elderly. For short-term users of NSAIDs or aspirin, cotreatment with proton pump inhibitors (but not with histamine H₂ blockers) may reduce the risk for bleeding to less than the risk in nonusers.

A thorough history and complete physical examination are important for evaluation of a child with GI bleeding. Bright

either upper or lower tract bleeding. Specifically, use of over-the-counter NSAIDs may represent an important cause of peptic ulcer disease and ulcer-related hemorrhage in those with UGIB.

Although most causes of LGIB in children are self-limited and benign, it is imperative to consider Meckel diverticulum, midgut volvulus, and intussusception in appropriate age groups.

**CLINICAL PRESENTATION**

Patients with GI bleeding can be rapidly assessed by their reported volume of blood loss and initial hemodynamic status. Massive hemorrhage is associated with signs or symptoms of hemodynamic instability, including tachycardia (heart rate greater than 100 to 120 beats/min), systolic blood pressure less than 90 to 100 mm Hg, symptomatic orthostasis, syncope, ongoing bright red or maroon hematemesis, transfusion requirements in the first 24 hours, and inability to stabilize the patient.

Vital signs and postural changes should be assessed in patients who appear sufficiently stable. An increase of 20 beats/min or more in pulse or a decrease of 20 mm Hg in systolic blood pressure between the supine and upright positions indicates loss of more than 20% of blood volume in normal adult patients.

Tachycardia, low blood pressure, reduced urine output, and conjunctival pallor in patients with GI bleeding are signs that mandate immediate volume replacement. Hypovolemic shock implies at least a 40% loss of blood volume. Note that abnormalities in vital signs, especially postural vital sign, are unreliable in pediatric and elderly patients.

The history should focus on the quantity, frequency, and duration of bleeding (differentiating between melena and hematochezia) to characterize the nature of the GI bleeding. Comorbid status, including other GI disorders, anticoagulant use, syncope, weight loss, alcohol intake, and cardiovascular disease, should be assessed.

In addition to continuous monitoring of vital signs, physical examination should include assessment of mental status, skin (for jaundice or pallor), and pulmonary and cardiovascular compromise (especially in the elderly because of ischemia from blood loss), as well as a thorough abdominal examination for distension, tenderness, or masses. Digital rectal examination and testing of stool for gross or occult blood should be performed in patients with suspected GI bleeding.

Complaints associated with LGIB include hematochezia or melena, although patients may have additional findings, such as anemia, light-headedness, hypovolemia, weakness, malaise, chest pain, and dyspnea. It is important to note that patients with LGIB may be asymptomatic and have complaints seemingly unrelated to intestinal bleeding (e.g., fatigue, weight loss); dramatic findings consisting of massive rectal bleeding in acutely ill and unstable patients are less common.

Delayed black tarry stools may occur from a source of bleeding in the small bowel or ascending colon and may not be noted by the patient until several days after the bleeding has stopped.

A thorough history and complete physical examination are important for evaluation of a child with GI bleeding. Bright
red blood that coats but is not mixed with stool suggests an anorectal source. Hematochezia indicates bleeding from the distal part of the small bowel or proximal part of the colon. Bloody diarrhea usually suggests colonic bleeding. Currant-jelly stools are indicative of the vascular congestion and hyperemia seen with intussusception.

Food allergy may lead to GI bleeding from food-induced colitis and could result in dehydration in infants younger than 3 months. Anal fissures are common in infants and produce red streaks or spots of blood in the diaper. Other causes of dark stool are iron, charcoal, flavored gelatin, red fruits, bismuth, and food dyes. Maternal blood swallowed by neonates during delivery may be diagnosed with the Apt test.

DIFFERENTIAL DIAGNOSIS

The most common causes of UGIB in adults are listed in Box 33.1, and causes of LGIB in adolescents and adults are listed in Box 33.2.

The exact cause of the GI bleeding is less important to the emergency physician (EP) than differentiation between upper and lower tract sources.

An aortoenteric fistula may have developed in a patient with massive LGIB and recent surgery.

DIFFERENTIAL DIAGNOSIS FOR PEDIATRIC GASTROINTESTINAL BLEEDING

Table 33.1 lists the differential diagnosis for UGIB and LGIB according to patient age. Ingestion of maternal blood is the most common cause of suspected GI bleeding in neonates; blood is swallowed during either delivery or breastfeeding (from a fissure in the mother’s breast). Other causes of GI bleeding in neonates include bacterial enteritis, milk protein allergies, intussusception, anal fissures, lymphonodular hyperplasia, and erosions of the esophageal, gastric, and duodenal mucosa.

Mucosal injuries presumably result from a dramatic rise in gastric acid secretion and laxity of the gastric sphincters in infants. Maternal stress in the third trimester has also been proposed to increase maternal gastrin secretion and enhance infantile peptic ulcer formation.

Some drugs have been implicated in neonatal GI bleeding, including NSAIDs, heparin, and tolazoline, which are used for persistent fetal circulation. Indomethacin, administered to

<table>
<thead>
<tr>
<th>AGE GROUP</th>
<th>CAUSES OF UPPER GI BLEEDING</th>
<th>CAUSES OF LOWER GI BLEEDING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>Hemorrhagic disease of the newborn</td>
<td>Anal fissure</td>
</tr>
<tr>
<td></td>
<td>Swallowed maternal blood</td>
<td>Necrotizing enterocolitis</td>
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<td></td>
<td>Stress gastritis</td>
<td>Malrotation with volvulus</td>
</tr>
<tr>
<td>Infants (1 mo-1 yr)</td>
<td>Esophagitis, Gastritis</td>
<td>Anal fissure</td>
</tr>
<tr>
<td></td>
<td>Intussusception</td>
<td>Gangrenous bowel</td>
</tr>
<tr>
<td></td>
<td>Gastritis</td>
<td>Milk protein allergy</td>
</tr>
<tr>
<td>Infants (1-2 yr)</td>
<td>Peptic ulcer disease, Gastritis</td>
<td>Polyps</td>
</tr>
<tr>
<td></td>
<td>Meckel diverticulum</td>
<td></td>
</tr>
<tr>
<td>Children (2-12 yr)</td>
<td>Esophageal varices, Gastric varices</td>
<td>Polyps</td>
</tr>
<tr>
<td></td>
<td>Infectious diarrhea</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td></td>
<td>Vascular lesions</td>
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</table>

maintain a patent ductus arteriosus in neonates, may cause GI bleeding through intestinal vasoconstriction and platelet dysfunction. Maternal medications can also cross the placenta and incite GI problems in the developing fetus and neonate on delivery. Aspirin, cephalothin, and phenobarbital are well-known causes of coagulation abnormalities in neonates. Stress ulcers in newborns are associated with dexamethasone, which is used for fetal lung maturation.

Rarer causes of GI bleeding in a neonate are volvulus, coagulopathies, arteriovenous malformations, necrotizing enterocolitis (especially in preterm infants), Hirschsprung enterocolitis, and Meckel diverticulitis.14

In infants, GI mucosal lesions and irritations are the most common causes of bleeding and include esophagitis, gastritis, duodenitis, ulcers, colonic polyps, and anorectal disorders. Intussusception is a common and important cause of GI bleeding in this age group. The incidence of intussusception is greatest in infants aged 3 months to 1 year, but it can occur in children up to 5 years of age. Approximately 80% of all cases of intussusception occur in infants younger than 2 years.

Other causes of infantile GI bleeding are infectious diarrhea, midgut volvulus, Meckel diverticulum, arteriovenous malformation, and GI duplication. Rare causes include foreign body ingestion, varicose disease, irritable bowel disease, and acquired thrombocytopenia.

Older children may have any of the preceding conditions, but duodenal ulcer, Mallory-Weiss tear, and nasopharyngeal bleeding are important causes of bleeding in this age group. Less common causes are gastritis or ulcers induced by salicylates or NSAIDs, Henoch-Schönlein purpura, ingestion of caustic substances, hemolytic-uremic syndrome, inflammatory bowel disease, and vasculitis. In adolescents older than 12 years, the most common causes of UGIB are duodenal ulcers, esophagitis, gastritis, and Mallory-Weiss tears.14

**DIAGNOSTIC TESTING**

**NASOGASTRIC ASPIRATION**

Historically, nasogastric aspiration has been used to determine whether the bleeding originated from the upper GI tract in patients with melena—a bloody aspirate confirmed an upper tract source, whereas an aspirate testing negative for blood represented either resolved bleeding or a more distal site of hemorrhage. In some studies, however, nasogastric aspiration was noted to be insensitive for detection of UGIB in patients without active hematemesis, and a negative result provided little information about the cause of the bleeding.10,31 The routine use of gastric aspiration and lavage in patients arriving at the ED with GI bleeding is not supported.32 Aspirates testing positive for blood confirm only that the bleeding is proximal to the pylorus, and patients must undergo endoscopy for further differentiation.

A nasogastric aspirate containing more than 1 L of fresh blood or inability to obtain a clear aspirate through lavage with more than 1500 mL of saline should alert the physician to massive UGIB that requires immediate gastroenterologic or surgical intervention. In patients with frank hematemesis and brisk persistent hemaanemia, the information provided by nasogastric aspiration may be lifesaving.33

**UPPER ENDOSCOPY**

Endoscopy is now the diagnostic test of choice for establishing the source of UGIB. The overwhelming majority of existing data suggest that early endoscopy is a safe and effective procedure in all risk groups.34,35 Patients without active hematemesis may benefit from immediate upper endoscopy by a gastroenterologist to confirm the site of bleeding rather than undergoing the potential additional discomfort and morbidity associated with placement of a nasogastric tube. Endoscopy is both diagnostic and therapeutic in many cases.36 One study noted that live-view video capsule endoscopy (VCE) accurately identifies high- and low-risk patients in the ED with UGIB. The use of VCE to risk-stratify these patients significantly reduced time to performance of emergency EGD and therapeutic intervention.37

**TAGGED RED BLOOD CELL STUDIES**

An advanced modality for detecting of the source of GI bleeding is radionuclide imaging, such as radioisotopic imaging with technetium Tc 99m sulfur colloid—or technetium pertechnetate–labeled red blood cells. Technetium Tc 99m red blood cell imaging is a useful test in the management of acute GI bleeding, particularly if the bleeding has been occurring for more than 3 hours and other modalities have failed to identify a source. A limitations of this test is poor detection of bleeding in the foregut, with the highest sensitivity noted for bleeding in the colon.38 A technetium Tc 99m sulfur colloid–labeled red blood cell study requires active bleeding at a rate of more than 0.1 mL/min for visualization. Radionuclide imaging has not been widely tested in the ED setting and is still reserved for inpatient use at most institutions.

**ARTERIOGRAPHY**

Angiography is appropriate for initial testing of patients with massive bleeding.39 When the bleeding cannot be identified and controlled by endoscopy, intraoperative enteroscopy or arteriography may help localize the bleeding source and facilitate segmental resection of the bowel.40 Mesenteric angiography can detect bleeding at a rate of 0.5 mL/min or greater.41 Either angiography or angiographic computed tomography may be used to identify aortoenteric fistulas.

Intraarterial injection of vasopressin or other vasoconstrictors at the site of bleeding can control hemorrhage; embolization is an option when immediate upper endoscopy by a gastroenterologist to confirm the site of bleeding rather than undergoing the potential additional discomfort and morbidity associated with placement of a nasogastric tube. Endoscopy is both diagnostic and therapeutic in many cases.36 One study noted that live-view video capsule endoscopy (VCE) accurately identifies high- and low-risk patients in the ED with UGIB. The use of VCE to risk-stratify these patients significantly reduced time to performance of emergency EGD and therapeutic intervention.37

**DISTAL COLONIC IMAGING**

Colonoscopy has high diagnostic yield and a low rate of perforation in patients with LGIB. It is best performed after colonic cleansing and in patients with slow bleeding. Proctosigmoidoscopy is used in patients with mild rectal bleeding to determine whether stool above the rectum contains blood. Barium enema is not useful in the acute setting but can be ordered after an acute bleeding episode has resolved.44

The optimal timing for colonoscopic intervention for LGIB is still unclear.9 More recent literature defines urgent colonoscopy as taking place within 12 hours.45 Evidence suggests that earlier colonoscopy leads to more diagnostic and therapeutic opportunities45 and reduces hospital length of stay.10,12,45
PROCEDURES

Proper technique for the safe placement and use of nasogastric tubes and gastroesophageal balloon tamponade tubes (e.g., Blakemore-Sengstaken tube) is discussed in Chapter 46.

TREATMENT

Figure 33.1 presents an algorithm for the treatment of GI bleeding. Insert two 18-gauge or larger intravenous lines and administer 0.9% normal saline or lactated Ringer solution on arrival of the patient. Quickly evaluate the patient’s hemodynamic status and determine the extent of blood or fluid resuscitation necessary. Standard resuscitative measures for the management of shock should precede or occur in parallel with definitive diagnostic testing. Management should otherwise be directed toward the underlying source of bleeding. Note that in 80% of cases, LGIB spontaneously stops.

If the patient is hemorrhaging, consult a gastroenterologist and surgeon. Upper endoscopy is the diagnostic and therapeutic procedure of choice for acute UGIB. Surgery is indicated for patients with active bleeding when medical therapy proves
Ineffective and continued hemorrhage requires more than 5 units of blood within the first 4 to 6 hours. Bowel resection may be required for pronounced LGIB.

In the absence of consultants, massive esophageal hemorrhage as a result of variceal bleeding may be temporarily treated with the placement of a gastroesophageal balloon tamponade device (Blakemore-Sengstaken tube). Although this is an uncommon procedure, EPs in remote practice locations should be familiar with the indications for and proper use of such potentially lifesaving devices.

Octrerotide acetate is a synthetic analogue of somatostatin that should be administered intravenously to all patients with suspected UGIB from esophageal varices to induce splanchnic vasoconstriction and a reduction in portal hypertension. The loading dose is 50 mcg intravenously, followed by infusion of 25 to 50 mcg/hr for 5 days.

Comorbid condition, such as coagulopathies, hypokalemia, and cardiac ischemia, should be identified and treated. The EP should consider the administration of fresh frozen plasma, platelets, recombinant factor VIIa (NovoSeven), or desmopressin (DDAVP) as appropriate.

**PATIENT TEACHING TIPS**

Patients should stop drinking alcohol and seek an alcohol cessation program immediately if needed.

Patients who take nonsteroidal antiinflammatory agents should receive concomitant therapy with a proton pump inhibitor.

Patients with gastric ulcers should be reexamined 6 to 8 weeks after the initial bleeding episode.

Patients should maintain daily intake of synthetic bulk-forming agents.

Surveillance colonoscopy should be performed every 3 years in patients with colon polyps and adenomatous changes or every 5 years in those with hyperplastic polyps.


**SUGGESTED READINGS**


**REFERENCES**

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES