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Gastrointestinal Bleeding in Infants and Children

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Author Disclosure
Dr Boyle did not disclose any financial relationships relevant to this article.

Objectives   After completing this article, readers should be able to:

1. Develop a differential diagnosis based on the clinical presentation of gastrointestinal (GI) blood loss.
2. Discuss the age-related causes of upper and lower GI bleeding.
3. Delineate the sequence of evaluation and decision process in a child who has GI bleeding.
4. Describe new medical therapies and endoscopic maneuvers to control GI bleeding.

Case Study

A previously well 3-week-old female infant presented with a 2-day history of rectal bleeding. Her parents described three to five loose stools per day mixed with bright and dark red blood and mucus. Associated symptoms included episodic nonbilious, nonbloody emesis and an erythematous rash on her arms and legs. The infant was receiving standard cow milk formula. Her weight gain and linear growth were excellent. The abdominal examination revealed no tenderness or organomegaly, and there were no anal fissures. Stool was guaiac-positive. The complete blood count (CBC) revealed normal hematocrit, mean corpuscular volume (MCV), platelet count, and white blood cell count. The total eosinophil count was mildly increased at $0.55 \times 10^3/mm^3$. The stool culture was negative. Clostridium difficile toxin was present. Three days after having been switched to a protein hydrolysate formula, the infant's bowel frequency decreased to twice a day. Although the baby continued to appear well, with good weight gain and growth, her stools still contained gross strands of blood and mucus intermittently over the next 3 weeks. Flexible sigmoidoscopy at that time revealed moderate nodular lymphoid hyperplasia in the rectosigmoid region (Fig. 1). The colonic mucosa appeared normal otherwise. Biopsies from the sigmoid and rectum showed six eosinophils per high-power field, normal crypt architecture, and lymphoid nodules. The infant continued to receive the protein hydrolysate formula, and gross bleeding gradually resolved over the next 2 weeks.

Determining Severity of Gastrointestinal (GI) Bleeding

GI bleeding may present as bright red blood on toilet tissue after passage of a hard bowel movement, strands or small clots of blood mixed within emesis or normal stool, bloody diarrhea, vomiting of gross blood (hematemesis), grossly bright or dark red bloody stools (hematochezia), or tarry black stools (melena). In cases of occult bleeding, the clinical presentation may be unexplained fatigue, pallor, or iron deficiency anemia. The treatment sequence for a child who has GI bleeding is to assess (and stabilize if necessary) the hemodynamic status of the patient, establish the level of bleeding, and generate a list of likely diagnoses based on clinical presentation and age of the patient.

Severity of the acute presentation is determined by the physical appearance and hemodynamic status of the patient, the estimated volume of blood lost, and the color of the blood lost. Worrisome signs and symptoms include pallor, diaphoresis, restlessness, lethargy, and abdominal pain. The best indicator of significant blood loss is orthostatic changes in heart rate and blood pressure. Orthostatic change is defined as an increase in pulse rate by 20 beats/min or a decrease in systolic blood pressure of 10 mm Hg or more on moving the patient from the supine to the sitting position.

Fresh blood quickly changes color to brown in an acid environment. Intestinal bacteria

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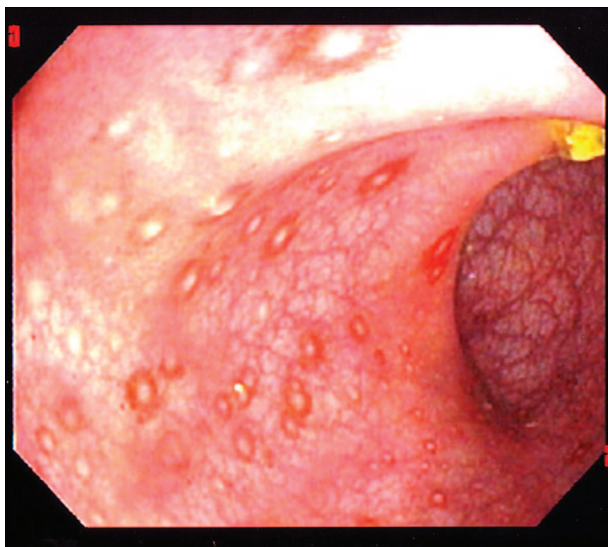
Figure 1. Nodular lymphoid hyperplasia: multiple submucosal nodules in the rectosigmoid area against a background of normal colonic mucosa.

oxidize hemoglobin to hematin, giving blood a tarry appearance. Coffee-ground emesis or melena suggests a lower rate of bleeding; bright red blood may indicate either a low or a very high rate of upper GI bleeding. The hematocrit is an unreliable index of the severity of acute GI bleeding because of the delay in compensatory hemodilution after acute blood loss. A low MCV of red cells on an automated CBC suggests a more chronic duration of bleeding, although the clinical presentation may appear as an acute GI hemorrhage.

Upper Versus Lower GI Bleeding

Upper GI bleeding refers to bleeding above the ligament of Treitz; lower GI bleeding is defined as bleeding distal to the ligament of Treitz. In most patients, the clinical presentation indicates the level of bleeding. Hematemesis is the classic presentation of upper GI bleeding. Bloody diarrhea and bright red blood mixed or coating normal stool are the classic presentations of lower GI bleeding. Hematochezia, melena, or occult GI blood loss could represent upper or lower GI bleeding. In cases of acute-onset hematochezia or melena, the level of bleeding can be confirmed by passage of a nasogastric (NG) tube. Not only is the presence of blood in the stomach diagnostic of upper GI bleeding (including significant duodenal hemorrhages that usually reflux into the stomach), but clearing of aspirated fluid during repeated NG lavage suggests that bleeding has stopped. Suspicion of bleeding esophageal varices is not a contra-

indication to passage of an NG tube. Persistent red or pink aspirate suggests ongoing bleeding and the need for more emergent diagnostic evaluation.

Is It Blood?

Chemical testing of the vomitus or stool is essential to verify the presence of blood. A number of substances may simulate bright red blood (food coloring, colored gelatin or children's drinks, red candy, beets, tomato skins, antibiotic syrups) or melena (bismuth or iron preparations, spinach, blueberries, grapes, licorice). The widely available guaiac test is the current recommended qualitative method for confirming the presence of gross or occult blood in vomitus or stool. Guaiac is a naturally occurring phenolic compound that can be oxidized to quinine by hydrogen peroxide in hemoglobin with detectable color change. Rarely, hemoglobin and myoglobin in meat or ascorbic acid in uncooked fruits and vegetables give false-positive test results. With newer guaiac test kits, exogenous iron preparations no longer give false-positive reactions. The newer method uses a buffered stabilized hydrogen peroxide solution to improve detection of blood in gastric aspirate or vomitus.

Immunochemical tests that detect only human blood have been proposed to improve sensitivity and specificity of detecting fecal occult blood in adults being screened for colon cancer. Immunochemical tests are the method of choice to confirm that red or tarry intestinal secretions are, indeed, human blood. However, the high sensitivity of these tests may be a problem in pediatric patients, in whom minute blood loss associated with passage of stool or perianal dermatitis may yield a positive test, leading to unnecessary diagnostic procedures. Additional studies are needed to determine the sensitivity and specificity of immunochemical tests compared with the guaiac-based tests as screens for occult blood loss in the evaluation of children who have chronic GI complaints such as chronic abdominal pain, vomiting, and failure to thrive.

Differential Diagnosis Based on Clinical Presentation

Most reviews of GI bleeding in children have focused on the differential diagnosis by age group, but causative disorders overlap considerably between age groups. This review focuses on the differential diagnosis based on clinical presentation (Table 1). Table 2 lists common causes of GI bleeding based on age group and clinical appearance of the child.

Table 1. **Differential Diagnosis of Gastrointestinal (GI) Bleeding Based on Clinical Presentation**

Hematemesis

- Swallowed blood
  Epistaxis, sore throat, breast feeding, dental work, or tonsillectomy
- Vitamin K deficiency in neonate
- Erosive esophagitis
- Mallory–Weiss tear
- Hemorrhagic gastritis
  Trauma, surgery, burns, or severe systemic stress (patients in intensive care units)
- Reactive gastritis
  Nonsteroidal anti-inflammatory drugs (NSAID gastropathy), alcoholic gastritis, cocaine ingestion, ingestion of caustic substances, stress, mechanical trauma, viral infection, Crohn disease, vasculitis (Henoch–Schönlein), radiation, bile reflux, bezoar, hiatal hernia, prolapse of the gastroesophageal junction, or congestive gastropathy (associated with portal hypertension)
- Peptic ulcer
- Variceal bleeding: associated with portal hypertension
- Submucosal masses
  Lipoma, stromal tumors, duplication
- Vascular malformation
  Angiodysplasia, hemangioma, Dieulafoy lesion
- Hemobilia

Hematochezia, Melena

- Intestinal ischemia
  Complicating intussusception, mid–gut volvulus, incarcerated hernia, or mesenteric thrombosis
- Meckel diverticulum
- Upper GI source: see hematemesis
- Vasculitis
  Henoch–Schönlein purpura
- Sloughed polyp
- Intestinal or colonic ulcer
  NSAID gastropathy, Crohn disease
- Ulcerative colitis
- Vascular malformation

Rectal Bleeding With Signs of Colitis (Bloody Diarrhea, Tenesmus, Nighttime Stooling)

- Infectious colitis
  Consider *Salmonella*, *Shigella*, *Yersinia enterocolitica*, *Campylobacter jejuni*, *Escherichia coli* O157:H7, *Aeromonas hydrophilia*, *Klebsiella oxytoca*, *Clostridium difficile*, *Neisseria gonorrhea*, cytomegalovirus, *Entamoeba histolytica*, *Trichuris trichiura*
- Hemolytic–uremic syndrome
- Necrotizing enterocolitis
- Eosinophilic proctocolitis
- Inflammatory bowel disease
  Ulcerative colitis, Crohn disease

Rectal Bleeding With Normal Stool Pattern

- Juvenile polyp
- Nodular lymphoid hyperplasia
- Eosinophilic colitis
- Inflammatory bowel disease
- Vascular malformation

(continued)

Table 1. Differential Diagnosis of Gastrointestinal (GI) Bleeding Based on Clinical Presentation—Continued

Bright Red Blood Coating Normal or Hard Stool

- Anal fissure
- Beta-hemolytic streptococcal cryptitis
- Ulcerative proctitis
- Rectal prolapse
- Solitary rectal ulcer
- Internal hemorrhoids

Occult GI Blood Loss

- Esophagitis
- Reactive gastritis
- Acid peptic disease
- Eosinophilic gastroenteritis, colitis
- Celiac disease
- Inflammatory bowel disease
- Polyposis
- Meckel diverticulum
- Vascular malformation

Hematemesis

CAUSES. Hematemesis (or acute hematochezia or melena with positive NG aspirate for blood) may result from swallowed blood, upper GI mucosal lesions, variceal bleeding, or rarely, hemobilia (hemorrhage into the biliary tract). Swallowed blood may be seen in conjunction with epistaxis, sore throat, or breastfeeding or may follow dental work or tonsillectomy. Mucosal lesions include esophagitis, Mallory-Weiss tear, reactive gastritis, stress ulcer, and peptic ulcer. A history of chronic heartburn, chest pain, epigastric abdominal pain, vomiting, oral regurgitation, or dysphagia suggests reflux esophagitis or peptic ulcer disease. Persistent vomiting, as seen in infants who have pyloric stenosis or older children who have cyclical vomiting, pancreatitis, or postviral gastroparesis, may result in acute erosive esophagitis. Infectious esophagitis, pill esophagitis, and eosinophilic esophagitis rarely present with GI bleeding.

A Mallory-Weiss tear is an acute mucosal laceration of the gastric cardia or the gastroesophageal junction. The classic presentation is hematemesis following repeated forceful retching, vomiting, or coughing. Abdominal pain is uncommon and, if present, more likely to be musculoskeletal in origin due to forceful emesis. Such vomiting episodes usually are linked to a concurrent viral illness.

Reactive gastritis may be diffuse or localized in the stomach. Significant hemorrhage may be seen with diffuse hemorrhagic stress gastritis associated with trauma, surgery, burns, or severe medical problems requiring hospitalization in an intensive care unit. Associated coagulopathy is not uncommon. Localized reactive gastritis is more common and may be associated with nonsteroidal anti-inflammatory drugs (NSAID gastropathy), alcoholic gastritis, cocaine ingestion, ingestion of caustic substances, *Helicobacter pylori* infection, viral infection, Crohn disease, vasculitis (Henoch-Schönlein purpura), radiation exposure, bile reflux, bezoar, hiatal hernia, prolapse of the gastroesophageal junction, or congestive gastropathy (associated with portal hypertension). Reactive gastritis may coexist with duodenal erosive lesions. Bleeding from localized gastritis usually manifests as coffee-ground emesis.

Peptic ulcers are rare in children, perhaps related to the current liberal use of acid reduction therapy in this population. *Helicobacter pylori* gastritis (Fig. 2) is an important cause of peptic ulcer in both children and adults, but bleeding from the gastritis alone is rare. Ulcers bleed when they erode into the lateral wall of a vessel. Foreign body ingestion is a rare cause of traumatic ulcer. A more common cause of gastric mucosal trauma is ulceration or erosions caused by tips of indwelling gastrostomy tubes or NG tubes.

Rare mucosal lesions that may present with hematemesis or melena include submucosal masses that extend into and erode the mucosal surface (lipoma, stromal tumors, gastroduodenal duplication), hemangioma, and

Table 2. Differential Diagnosis of Gastrointestinal Bleeding Based on Age, Appearance of Child, and Rate of Bleeding

	Ill-appearing Child	Well-appearing Child	
		High Rate of Bleeding	Low Rate of Bleeding

Infant	Upper Tract
	  Hemorrhagic gastritis
	  Stress ulcer
	
	Lower Tract
	  Infectious colitis
	  Necrotizing enterocolitis
	  Hirschsprung enterocolitis
	  Volvulus
			Reflux esophagitis
			Reactive gastritis
			Vitamin K deficiency
			
			Anal fissure
			Eosinophilic proctocolitis
			Infectious colitis
			Nodular lymphoid hyperplasia

2 to 5 years of age	Upper Tract
	  Esophageal varices (liver disease)
	  Hemorrhagic gastritis
	  Stress ulcer
	Lower Tract
	  Intussusception
	  Volvulus
	  Henoch-Schönlein purpura
	  Hemolytic-uremic syndrome
		Esophageal varices
		Gastric/Duodenal ulcer
		
		Meckel diverticulum
		Sloughed juvenile polyp
		Ulcerative colitis
			Mallory-Weiss tear
			Gastritis
			Reflux esophagitis
			
			Infectious colitis
			Juvenile polyp
			Nodular lymphoid hyperplasia
			Ulcerative colitis/Crohn disease
			Perianal streptococcal cellulitis
			Rectal prolapse/rectal ulcer

Older child	Upper Tract
	  Esophageal varices
	  Hemorrhagic gastritis
	
	Lower Tract
	  Infectious colitis
	  Ulcerative colitis
	  Henoch-Schönlein purpura
	  Intestinal ischemia
		Esophageal varices
		Bleeding ulcer
		Dieulafoy lesion
		Hemobilia
		
		Ulcerative colitis
		Meckel diverticulum
			Mallory-Weiss tear
			Reflux esophagitis
			Reactive gastritis
			
			Infectious colitis
			Ulcerative colitis/Crohn disease
			Juvenile polyp
			Hemorrhoid

Rare causes of bleeding: vascular malformation, hemobilia, intestinal duplication, submucosal mass, neutropenic colitis (typhlitis).

Dieulafoy lesion (a submucosal artery that aberrantly protrudes through a minute defect in the mucosa).

Gastrointestinal stromal tumors (GIST) are mesenchymal tumors arising from the GI wall, mesentery, omentum, or retroperitoneum. Most GIST tumors are found in the stomach (60% to 70%) and should be considered in a patient who has neurofibromatosis. Hemobilia is a rare complication of abdominal trauma, biliary tumor, or parasitic infection (*Ascaris*).

Upper gastrointestinal bleeding may be the initial presentation of esophageal varices. Variceal bleeding caused by portal hypertension should be considered in any child who has hepatomegaly, splenomegaly, ascites, jaundice, or scleral icterus. For the patient who has no previous history of liver disease, variceal bleeding is suggested by a past history of jaundice, hepatitis, blood transfusion, chronic right heart failure, or disorders associated with extrahepatic portal vein thrombosis (history of abdominal surgery or neonatal sepsis, shock, exchange transfusion, omphalitis, umbilical vein catheterization).

ASSESSMENT. Most previously well children who present with hematemesis are hemodynamically stable and usually describe hematemesis as coffee ground-like or bright red-tinged vomitus, again indicating a low rate of bleeding. Bleeding from mucosal lesions usually stops spontaneously. The initial laboratory evaluation reveals a normal hematocrit, MCV, platelet count, coagulation profile, total and direct bilirubin, liver enzymes, total protein, and albumin. Affected patients can be prescribed oral inhibitors of gastric acid secretion and followed as outpatients. A bleeding mucosal lesion can be diagnosed
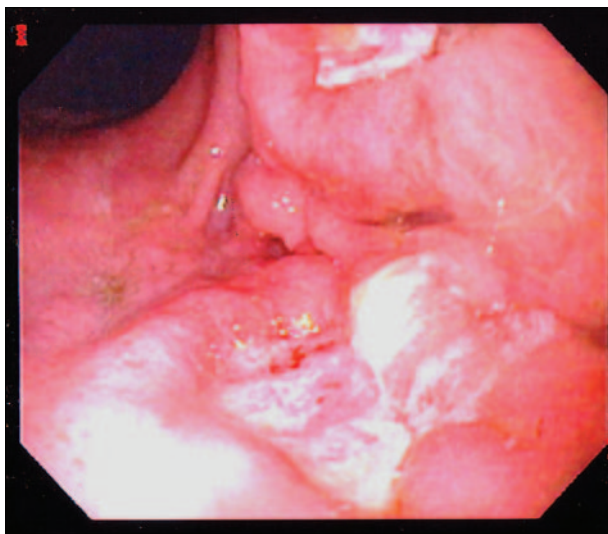
Figure 2. Multiple duodenal ulcers in a 12-year-old child who has *Helicobacter pylori* gastritis.

presumptively on the basis of the history and physical examination; stool guaiac test usually is negative.

Generally, infants younger than 1 year of age or any patient who has a history of significant upper GI blood loss, acute hematemesis associated with heme-positive stool, or physical or biochemical evidence of possible portal hypertension should be hospitalized for observation. If blood in the emesis of a well-appearing breastfed infant can be determined to be of maternal origin, admission is not indicated. The Apt-Downey test is based on an infant's blood containing more than 60% fetal hemoglobin that is alkali-resistant. Blood of maternal origin that may be swallowed during delivery or comes from a fissure in the mother's nipple contains adult hemoglobin, which converts to brownish-yellow alkaline hematin upon mixing with alkali. All neonates who have hematemesis should be screened for coagulopathy due to vitamin K deficiency from failure to administer prophylaxis postdelivery, maternal thrombocytopenic purpura, hemophilia, and von Willebrand disease. However, coagulopathy in the absence of mucosal lesions or varices is a rare cause of gross GI blood loss in older infants and children.

Upper endoscopy is the test of choice for evaluating hematemesis. The goals of endoscopy in upper GI bleeding are to identify the site of the bleeding, diagnose the specific cause of the bleeding, and initiate therapeutic interventions when indicated. Emergency endoscopy is necessary only when the patient continues to bleed at a rate considered to be life-threatening (ongoing hematemesis, hemodynamic instability, continuous transfusion

requirement). Most centers use general anesthesia and control of the airway in children who have active upper GI hemorrhage. Upper endoscopy during active bleeding usually can identify the site of bleeding, distinguish variceal from mucosal bleeding, and identify diffuse gastritis. Esophageal varices run upward from the gastroesophageal junction (Fig. 3). The surface tends to have a blue tint, and the outline usually is beaded. The greater their diameters, the more prominent they appear. A clot or cherry-red spot on a varix supports the occurrence of recent variceal bleeding.

The quality of the stomach examination during active bleeding, particularly the ability to see ulcers and vessels within the ulcers or to identify a Dieulafoy lesion, may be hampered by the presence of residual blood and clots in the upper GI tract. The characteristic endoscopic appearance of a Dieulafoy lesion is blood spurting from a pinpoint mucosal defect without surrounding exudates, inflammation, or ecchymosis. Gastric varices seldom occur in the absence of esophageal varices.

The combination of gastric lavage and intravenous erythromycin prior to endoscopy improves stomach cleansing. Erythromycin, a macrolide antibiotic, acts as a motilin receptor agonist that accelerates gastric emptying by inducing gastric contractions within a few minutes after infusion. For optimal diagnostic results, endoscopy should be performed soon after active bleeding has stopped. Elective upper endoscopy is indicated following significant hematemesis; for a patient who has recurrent hematemesis, unexplained biochemical evidence of iron deficiency, or presumed persistent peptic disease while
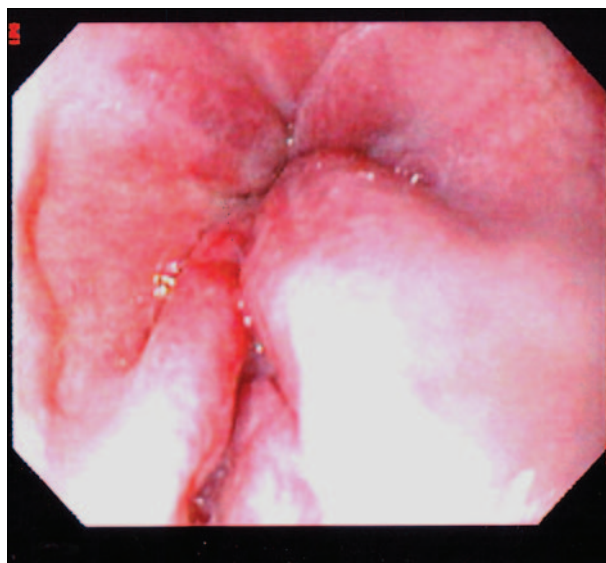

Figure 3. Esophageal varices in the distal esophagus.

receiving acid reduction therapy; and for any patient in whom portal hypertension is suspected, as indicated by a history of liver disease, jaundice, hepatomegaly, splenomegaly, elevated transaminases, hyperbilirubinemia, hypoalbuminemia, coagulopathy, or signs of hypersplenism, including thrombocytopenia and leukopenia.

Hematochezia

CAUSES. The differential diagnosis of hematochezia, the passage of gross blood or melena per rectum, depends on the clinical presentation. The color of the blood, the age of the patient, the presence of abdominal pain or tenderness, and a history of altered bowel pattern are important factors in assessing a child who has hematochezia. Although rare, blood from the upper GI tract may appear unchanged in the stool due to rapid intestinal transit. Approximately 10% to 15% of mucosal or variceal hemorrhages from the upper GI tract may present with melena alone, without hematemesis. In children, the acute passage of melena or dark blood usually indicates bleeding originating from the stomach, duodenum, small bowel, or proximal colon. In such cases, an NG tube should be passed to distinguish upper from lower GI bleeding, taking care not to mistake a small amount of fresh clotted blood caused by passing the NG tube for active upper GI hemorrhage.

Acute hematochezia in an ill-appearing child (either extreme irritability or lethargy) who has acute abdominal pain and tenderness suggests intestinal ischemia as a complication of intussusception, mid-gut volvulus (associated with malrotation, mesenteric cyst, intestinal duplication, or internal hernia), incarcerated hernia, or mesenteric thrombosis. Intestinal bleeding is a late sign of acute intestinal obstruction that subsequently led to venous congestion, ischemia, and hemorrhagic necrosis of the affected area of bowel. Idiopathic intussusception should be the working diagnosis for any child younger than 2 years of age in whom abdominal pain or tenderness is associated with lower GI blood loss. The sudden onset of colicky abdominal pain and vomiting in the setting of an antecedent viral illness followed by passage of "currant jelly" stool is an intussusception until proven otherwise.

Beyond age 2 years, intussusception is more likely to be associated with a lead point such as a Meckel diverticulum, polyp, nodular lymphoid hyperplasia, foreign body, intestinal duplication, intramural hematoma, lymphoma, or bowel wall edema in the presence of Henoch-Schönlein purpura. Obvious hematochezia or melena in association with abdominal pain and distention occurs in 15% to 25% of children who have Henoch-Schönlein purpura and may antedate skin lesions by up to 1 week. A more chronic history of abdominal pain antedating hematochezia raises the possibility of inflammatory bowel disease, Meckel diverticulum (with associated ulcer), or GI tuberculosis.

Painless passage of blood per rectum suggests a Meckel diverticulum, polyp, intestinal duplication, intestinal submucosal mass (GIST), angiodysplasia/vascular malformation, or superior mesenteric artery aneurysm. A Meckel diverticulum is a vestigial remnant of the omphalomesenteric duct located on the antimesenteric border in the distal ileum that occurs in 1.5% to 2.0% of the general population. A Meckel diverticulum that contains gastric mucosa may present as painless acute lower GI bleeding. Bleeding from a Meckel diverticulum, 50% of which occurs before the child is 2 years of age, sometimes is severe. The passage of a large amount of bright to dark red blood by a well child should be considered bleeding from a Meckel diverticulum until proven otherwise.

Anemia and severe bleeding rarely occur from a juvenile polyp. Autoamputation of a juvenile polyp that has outgrown its blood supply may cause significant hematochezia. Parents may observe tissue in the blood. Rarely, painless bleeding from deep ulceration of the terminal ileum or colon may be the initial presentation of Crohn disease. It also is important to remember that NSAIDs may cause ulcerations in the small bowel and colon in addition to the upper GI tract.

ASSESSMENT. All infants who experience acute hematochezia should undergo abdominal flat plate and either upright or cross-table lateral radiography to screen for intestinal obstruction or pneumatosis intestinalis (gas in the bowel wall, a radiologic finding in ischemic bowel disease). Several modalities are available to diagnose the patient who is suspected of having intussusception. With a high degree of suspicion in infants younger than 2 years of age, an air or water-soluble contrast enema is not only diagnostic, but also potentially therapeutic. The classic contrast enema finding is a "coiled spring," which results when contrast coats the crevices between crowded haustra. When a contrast enema is performed for a suspected intussusception, a pediatric surgeon should be available in case complications occur. In the older child, the differential diagnosis of intestinal ischemia may be broader. Therefore, abdominal computed tomography (CT) scan or abdominal ultrasonography may be the initial diagnostic choice after consultation with the pediatric surgical staff.

After excluding intestinal ischemia due to intussus-

ception and other causes, the next step in the evaluation of hematochezia is to perform a Meckel scan (99Tc-pertectnetate nuclear scan) to look for a Meckel diverticulum. The radionuclide binds rapidly to gastric mucosa within the diverticulum, resulting in a well-demarcated focus, usually in the right lower quadrant. The radionuclide also may be taken up by gastric heterotopia in the small bowel mucosa or enteric duplications. Some have advocated pretreatment with a histamine$_2$-receptor antagonist prior to the Meckel scan to stimulate technetium uptake by gastric mucosa.

After excluding obstruction or Meckel diverticulum, the algorithm to investigate hematochezia and melena can be exhaustive and can include upper endoscopy, colonoscopy, nuclear medicine scans, contrast enteroclysis (radiologic procedure that uses modified contrast agents to enhance visibility of the small bowel mucosa), capsule endoscopy, push enteroscopy, angiography, laparoscopy, and intraoperative enteroscopy.

Upper endoscopy and colonoscopy should be performed at the same time. The diagnostic role of upper endoscopy has been discussed. Colonoscopy can detect polyps, angiodysplasia, and ulcers in the terminal ileum, colon, and ileocolonic anastomotic site in patients who have had previous surgical ileocolostomy as well as vasculitis and inflammatory bowel disease. Endoscopic biopsy from areas of bleeding or from ulcerations may be diagnostic of vasculitis associated with Henoch-Schönlein purpura. The colon must be inspected for vascular lesions during insertion of the endoscope because endoscopic manipulation often causes petechial hemorrhage, which can be mistaken for angiodysplasias on withdrawal.

When bleeding persists and endoscopy fails to identify a bleeding site, radioisotope-tagged red blood cell scans using technetium 99m-sulfur colloid may be capable of detecting the location of bleeding if the rate exceeds 0.1 mL/min. Unfortunately, this modality has significant false localization and false-negative rates. Angiography is technically difficult in children but can be useful when there is 1 to 2 mL/min of active bleeding.

If bleeding has stopped, complete radiologic evaluation of the small bowel with barium contrast or CT enteroclysis may detect small structural mucosal, but not vascular, lesions. Wireless capsule endoscopy has revolutionized evaluation of the GI tract and now is being applied in pediatrics. Adult studies have described this technique as providing the highest diagnostic yield in ongoing, overt, small bowel bleeding of obscure origin (~ 90%), with a lesser diagnostic yield in patients who have heme-positive stools and anemia (~ 40%). Capsule

retention is the most serious complication. To prevent this complication, patients routinely should undergo a contrast small bowel enteroclysis prior to the capsule study to rule out mass lesions or intestinal stricture.

When no exact bleeding source can be identified and melena or hematochezia continue, laparoscopy may be useful in ruling out missed Meckel diverticulum, intestinal duplication, or an abnormal-appearing gallbladder suggesting possible hemobilia. Before proceeding with laparoscopy in a patient who has obscure GI bleeding, repeat upper endoscopy and colonoscopy should be considered. In adults, approximately 30% of upper lesions and 3% of colonic lesions are missed during initial endoscopy.

Rectal Bleeding With Signs of Colitis

CAUSES. Symptoms of colitis include bloody diarrhea, tenesmus (urgency to defecate), nighttime stooling, and abdominal pain. Acute onset of bloody diarrhea suggests an infectious colitis. In a well infant younger than 6 months of age, the cause of acutely bloody stools most likely is infectious colitis or eosinophilic proctocolitis. Late-onset necrotizing enterocolitis or Hirschsprung disease with enterocolitis also must be considered in an ill-appearing infant. The latter consideration is especially important if there has been a preceding history of constipation dating to early infancy.

Beyond infancy, the two common causes of bloody diarrhea are infectious colitis, which can be associated with hemolytic-uremic syndrome in the case of *Escherichia coli* and *Shigella* infections, and inflammatory bowel disease. In 70% to 80% of children who have hemolytic-uremic syndrome, bloody diarrhea precedes the recognition of hemolytic anemia, thrombocytopenia, and renal insufficiency by 3 to 16 days. Because most bacterial colitis is self-limiting and resolves spontaneously within 2 weeks, any patient who has a history of bloody diarrhea for more than 2 weeks should be referred to a pediatric gastroenterologist for evaluation of inflammatory bowel disease. The presence of fever, fatigue, weight loss, arthralgia, or arthritis supports the diagnosis of inflammatory bowel disease.

Ischemic colitis or vasculitis should be considered in patients who have collagen vascular disease, a history of recent anesthesia, cardiac failure, uremia, or a history of taking birth control medications or digitalis. Radiation enterocolitis also must be considered in selected oncology patients. Typhlitis is an acute inflammation or necrosis of the cecum, appendix, and terminal ileum associated with profound neutropenia and is seen most commonly in children who have leukemia being treated with cyto-

toxic drugs, although it also is associated with aplastic anemia, lymphoma, acquired immunodeficiency syndrome, and immunosuppression following transplantation.

ASSESSMENT. Stool studies should include smear for polymorphonuclear leukocytes; bacterial culture for *Salmonella*, *Shigella*, *Yersinia enterocolitica*, *Campylobacter jejuni*, *Escherichia coli* O157:H7, *Aeromonas hydrophilia*, and *Klebsiella oxytoca*; and toxin assay for *Clostridium difficile* (both toxin A and B). In an adolescent, a perianal culture for *Neisseria gonorrhea* should be obtained. Cytomegalovirus (CMV) colitis can present with bloody diarrhea and should be considered in an immunocompromised patient. CMV can be cultured from the stool. Rotavirus rarely is associated with blood-tinged diarrhea. Enzyme immunoassay for rotavirus is indicated only in the clinical context of acute watery diarrhea, which may be blood-tinged in a child 6 months to 3 years of age who has the associated symptoms of vomiting, colicky abdominal pain, and low grade fever.

If indicated by geography or recent travel, stool samples for *Entamoeba histolytica* and *Trichuris trichiura* should be obtained. It is not unreasonable to obtain a CBC with platelet count, blood urea nitrogen (BUN) measurement, creatinine assessment, and urinalysis for all patients presenting with acute bloody diarrhea to screen for hemolytic-uremic syndrome. Because bloody diarrhea may precede renal manifestations of hemolytic-uremic syndrome by 3 to 16 days, repeat CBC with platelet count, BUN and creatinine measurements, and urinalysis should be considered 14 days from the onset of GI symptoms for patients who have culture-proven bacterial colitis.

Colonoscopy is indicated for patients who show evidence of significant inflammation (greater than five grossly bloody stools per day, nighttime stooling, anemia, tachycardia, hypoalbuminemia) or well-appearing patients who have persistent bloody diarrhea in excess of 2 weeks. Colonoscopy allows collection of colonic secretions for culture and assay that are not contaminated with urine, which might affect test results. Characteristic pseudomembranes may be seen with *C difficile* and *Shigella* infection. The goals for colonoscopy in patients who have inflammatory bowel disease are to define the extent of the inflammation, obtain biopsies to try to distinguish Crohn disease from ulcerative colitis, and subjectively aid in planning initial therapy. Colonoscopy is contraindicated if a child appears toxic, has signs of peritonitis, has toxic megacolon (a life-threatening condition characterized by a dilated colon, abdominal dis-

tention, abdominal pain, and sometimes fever or shock), or might have a condition requiring surgery or a surgical complication.

Rectal Bleeding in Which Blood is Mixed With Normal-appearing Stool

CAUSES. Many times a parent or child reports blood mixed within normal stool with or without mucus. It is important to realize that colitis does not always present with diarrhea. In a well infant younger than 6 months of age, blood mixed within stool may be a sign of eosinophilic proctocolitis or nodular lymphoid hyperplasia. Between 2 and 6 years of age, a well child who passes small amounts of bright-to-dark red blood mixed within a stool or coated on the outside of a stool most likely has a juvenile polyp. Juvenile polyps, which account for more than 95% of all polyps found in children, are inflammatory hamartomas that carry a very low, if any, malignant potential. Seventy percent of juvenile polyps occur in the left side of the colon and are solitary. Multiple polyps are associated with Peutz-Jeghers syndrome and multiple juvenile polyposis coli. Peutz-Jeghers syndrome should be suspected when mucocutaneous pigmentation is noted during physical examination. Adenomatous polyposis syndromes are less likely to present with rectal bleeding in the pediatric age range.

Painless rectal bleeding in young children also can be caused by nodular lymphoid hyperplasia of the colon. In most children, such nodules are self-limiting and associated with preceding viral infection or eosinophilic proctocolitis, but they may be associated with immunodeficiency (selective immunoglobulin A deficiency or hypogammaglobulinemia). The mechanism for the bleeding is believed to be thinning of the surface of the mucosa over the enlarged hyperplastic submucosal lymphatic tissue, with subsequent small mucosal ulceration and bleeding. Bleeding is most common when nodules are present in the sigmoid and rectum, suggesting friability of the stretched mucosa unmasked by passage of a bowel movement.

ASSESSMENT. Colonoscopy is indicated for any child who has unexplained rectal bleeding that is documented either visually or by chemical testing. Juvenile polyps occur most commonly in the left colon on a stalk and may be removed by snare and cautery. Endoscopic polypectomy of large colonic polyps (>2 cm) increases the risk of perforation because thermal energy delivered for polyp removal can traverse the thin muscular layer of the colon, resulting in tissue necrosis. Nodular lymphoid hyperplasia has the gross appearance of multiple 1- to

4-mm umbilicated submucosal nodules that may be hard to distinguish from aphthous ulcers.

Bright Red Blood Coating a Normal-appearing Stool or Associated With Constipation

CAUSES. Bright red blood coating a normal-appearing stool is suggestive of a perianal disorder, most commonly an anal fissure, cryptitis, or proctitis. Anal fissures typically occur before age 1 year and usually are associated with a history of constipation or recent acute diarrhea. The blood almost always is of small amount and red and appears most often as a strip on the outside of the stool. The fissure usually starts when passage of hard stool tears the sensitive squamous lining of the anal canal. In a patient who has perianal erythema and an anal fissure, beta-hemolytic streptococcal cellulitis should be considered and the anal canal cultured before applying bacteriostatic lubricant to perform a rectal examination.

Beyond infancy, perianal disease or recurrent anal fissures should raise suspicion of inflammatory bowel disease or sexual abuse. A foreign body inserted into the rectum also may traumatize the rectal mucosa and produce bleeding. Rectal prolapse, most often due to constipation and excessive straining, forces the anterior rectal mucosa into the anal canal, causing congestion, edema, and occasionally, ulceration. A solitary rectal ulcer is rare in childhood but can be a complication of mucosal congestion and edema. Symptoms include dyschezia (difficult defecation), tenesmus, discharge of mucus, and rectal bleeding. External hemorrhoids are associated with recurrent anal fissures and proctitis, but rarely are a cause of bleeding unless irritated by excessive cleaning after bowel movements. Internal hemorrhoids are rare in children and adolescents.

ASSESSMENT. In most cases, anal fissure can be diagnosed by careful examination of the perianal area. All patients who have perianal excoriation, multiple anal fissures, or fissure resistant to conservative management should have perianal culture for beta-hemolytic *Streptococcus*. Anal trauma, internal hemorrhoids, proctitis, and solitary rectal ulcer may be diagnosed by proctosigmoidoscopy with retroflexion in the rectum.

Occult GI Blood Loss

CAUSES. Occult blood in the stool is detected most commonly by chemical testing during the evaluation of chronic GI symptoms such as abdominal pain, vomiting, diarrhea, and constipation; unexplained systemic symptoms (weight loss, growth retardation, arthralgia, fever); or unexplained iron deficiency anemia. The causes of occult bleeding in children are similar to those of clinically apparent GI bleeding discussed previously. The most common causes are inflammatory disorders (including esophagitis), acid peptic disease, reactive gastritis, eosinophilic gastroenteritis, celiac disease, Henoch-Schönlein purpura, Crohn disease, ulcerative colitis, polyps, and Meckel diverticulum. Rare causes of occult bleeding are vascular anomalies, infection, and neoplasia. Infectious causes of occult GI blood loss include hookworm, ascariasis, amoebic infection, *Strongyloides* infection, and tuberculosis.

ASSESSMENT. A patient who has occult GI blood loss should undergo investigations directed toward identifying pathologic processes that can explain both the symptoms and the blood loss. For example, it is reasonable to perform upper endoscopy alone in a 14-year-old patient who has chronic epigastric abdominal pain, episodic vomiting, occult positive stool, and positive *H pylori* serology. However, if symptoms suggest the possibility of Crohn disease (growth deceleration, diarrhea, arthralgia or arthritis, perianal skin tags or fistula) or if a patient has no symptoms other than occult-positive stool and iron deficiency anemia, it is reasonable to perform both upper endoscopy and colonoscopy.

Therapeutic Considerations
Supportive Measures

Supportive measures include stabilization of hemodynamic status, correction of any coagulation or platelet abnormalities, blood transfusion if necessary, and iron supplementation. Because both intravascular and extravascular volumes are reduced in acute GI bleeding, crystalloid (normal saline, Ringer lactate) is the solution of choice for initial intravenous resuscitation. Colloid solutions or blood are used only when blood loss is massive, in which case the patient is at risk for developing respiratory insufficiency or shock lung because of a significant decrease in plasma oncotic pressure. Blood transfusion is the only method of restoring oxygen-carrying capacity during active GI bleeding.

Intravenous acid suppression has been shown to improve ulcer healing in adults. In children, pharmacokinetic studies have been performed with intravenous histmine$_2$-receptor antagonists (Table 3). Tachyphylaxis to intravenous ranitidine is a significant problem if more than 2 weeks of continuous therapy is required. Recent studies in adults have demonstrated improved outcomes after peptic ulcer bleeding by using an intravenous pro-

Table 3. Pharmacologic Therapy of Gastrointestinal Bleeding

Indication	Category of Drug	Dosage*
Active Bleeding		
Intravenous Inhibitors of Gastric Acid Secretion		
Ranitidine	Histamine₂ antagonist	Continuous infusion, 1 mg/kg followed by infusion of 2 to 4 mg/kg per day
		Bolus infusions, 3 to 5 mg/kg per day divided every 8 hours
Pantoprazole	Proton pump inhibitor	Children <40 kg: 0.5 to 1 mg/kg per day IV once daily
		Children >40 kg: 20 to 40 mg once daily (maximum, 40 mg/d)
Intravenous Vasoactive Agents		
Octreotide	Somatostatin analog	1 mcg/kg IV bolus (maximum, 50 mcg) followed by 1 mcg/kg per hour
		May increase infusion rate every 8 hours to 4 mcg/kg per hour (maximum, 250 mcg per 8 hours)
		When bleeding is controlled, taper 50% every 12 hours
		May stop when at 25% of starting dose
Vasopressin	Antidiuretic hormone	0.002 to 0.005 units/kg per minute×12 hours, then taper over 24 to 48 hours (maximum, 0.2 units/min)
Prevention of Rebleeding		
Oral Inhibitors of Gastric Acid Secretion		
Ranitidine	Histamine₂ antagonist	2 to 3 mg/kg per dose twice or three times a day (maximum, 300 mg/d)
Famotidine	Histamine₂ antagonist	0.5 mg/kg per dose twice daily (maximum, 40 mg/d)
Lansoprazole	Proton pump inhibitor	1 to 1.5 mg/kg per day once to twice daily (maximum, 30 mg twice daily)
Omeprazole	Proton pump inhibitor	1 to 1.5 mg/kg per day once to twice daily (maximum, 20 mg twice daily)
Oral Adhesive Protection of Ulcerated Mucosa		
Sucralfate	Local adhesive paste	40 to 80 mg/kg per day in 4 divided doses (maximum, 1,000 mg/dose in 4 divided doses)
Oral Prevention of Variceal Rebleeding		
Propranolol	Reduced mesenteric blood flow (beta-adrenergic blocker)	1 mg/kg per day in 2 to 4 divided doses
		May increase every 3 to 7 days to maximum of 8 mg/kg per day to achieve a 25% reduction from baseline pulse rate

*Evidence-based standard of care pediatric dosages for these medications are not well established. Dosages listed are taken from *Pediatric Lexi Drug* online formulary. They do not necessarily apply to neonates or infants younger than 3 months of age. Higher doses may be used by individual pediatric gastroenterologists based on peer-reviewed published case series and personal experience. Major adverse effects are listed in the text. Ranitidine and famotidine dosages must be adjusted downward for patients who have renal impairment.

ton pump inhibitor (PPI). Dosing in children has been extrapolated from adult literature, although available data suggest faster drug clearance and significant interindividual variability in pediatric patients (Table 3).

Control of Active Upper GI Bleeding

There is no evidence that gastric lavage has any therapeutic role in controlling hemorrhage, and there is no benefit to continuous lavage beyond 10 minutes if the NG

return is not clearing. In fact, continued NG lavage in the face of active bleeding might be deleterious by not allowing fibrin clots to form at the site of hemorrhage. Vasoactive agents, including octreotide and vasopressin, are used for both mucosal and variceal bleeding, usually as adjunctive therapy to endoscopic hemostasis. Published experience in pediatric patients is limited to case reports and small case series. These drugs decrease portal pressure by decreasing splanchnic blood flow. Octreotide is a synthetic octapeptide that has pharmacologic actions similar to those of the endogenous hormone somatostatin. Octreotide has fewer hemodynamic adverse effects than vasopressin and is the drug of choice. Vasopressin can cause disturbing peripheral vasoconstriction and may trigger renal failure. The major adverse effect of octreotide is hyperglycemia. Octreotide is initiated as a bolus injection of 1 mcg/kg (up to a maximum of 50 mcg) followed immediately by continuous infusion of 1 mcg/kg per hour, which may be increased hourly by 1 mcg/kg per hour up to 4 mcg/kg per hour (Table 3).

Endoscopic hemostasis of mucosal lesions includes injection and thermal methods. Among the mucosal lesions amenable to endoscopic therapy are ulcers with active bleeding, oozing from a clot overlying an ulcer, or an ulcer that has a visible vessel at its base. The injection method used most commonly in pediatric patients is injection of 1:10,000 epinephrine in normal saline into and near the periphery of an oozing lesion. Injection therapy may slow or stop active bleeding, but it should be followed by contact thermal coagulation. Contact thermal methods achieve hemostasis by local tamponade and coaptive coagulation, which involves fusing the walls of blood vessels up to 2 mm in size. Common contact thermal methods are heater probe, bipolar probes, and BICAP cautery. The heater probe allows tamponade with firm direct pressure on a bleeding site, followed by delivery of two to four pulses of 15 to 30 J to coagulate the lesion. In adults, perforation has been reported in approximately 1% of patients and rebleeding in 18% of patients after thermal methods. Endoscopic clip placement is a newer technique to capture and compress the tissue surrounding a bleeding vessel.

The endoscopic therapies for acute variceal bleeding include injection sclerotherapy and variceal band ligation therapy. Sclerotherapy in pediatrics employs 25-gauge needles to inject volumes of sclerosant based on patient weight. The most common significant complication of injection sclerotherapy is esophageal ulceration leading to stricture formation, which occurs in 15% of all children treated. Several controlled studies in adults have shown that band ligation has a higher efficacy in preventing rebleeding and

has fewer complications, lower costs, and higher rates of survival. With the development of multiband ligation devices, which allow application of up to six bands per session, pediatric experience with this technique is encouraging. The inability to pass the band ligation apparatus in infants and small children is the only limiting factor. When a child is bleeding and hemodynamically unstable, both sclerotherapy and band ligation can be technically difficult. In such cases, it is best to monitor the child in an intensive care unit, protect the airway with an endotracheal tube, sedate the patient, and temporarily control the bleeding by passing a Sengstaken-Blakemore tube or Linton tube. These tubes allow mechanical balloon tamponade at the gastroesophageal junction to stop bleeding before proceeding with therapeutic endoscopy.

Angiography is employed most often when an additional therapeutic component is needed, such as placement of a transjugular portosystemic shunt, selective infusion of a vasoactive agent into a bleeding vessel, or embolization of a bleeding vessel with gel foam or coils.

Control of Active Lower GI Bleeding

Lower GI bleeding rarely is life-threatening. Meckel diverticulum is treated by surgical resection. Endoscopy can treat colonic lesions such as polyps, bleeding ulcers, telangiectasias, or small hemangiomas. Juvenile polyps are removed by snare polypectomy. As with upper GI bleeding, endoscopic hemostasis of mucosal lesions includes injection and thermal methods. Because of the thin wall of the colon, the total number of joules applied to a bleeding colonic lesion should be lower than that used in the stomach.

Prevention of Rebleeding

For conditions that have a significant rate of rebleeding (variceal bleeding, chronic NSAID therapy, angiodysplasia), the goal is to decrease the rebleeding rate. Medical therapy includes acid suppression with antacids, histamine$_2$-receptor antagonists, or PPIs. In addition, binding agents such as sucralfate have been shown to increase ulcer healing. Sucralfate is particularly effective for esophageal bleeding due to caustic or mechanical forms of mucosal damage (Table 3).

Secondary Prophylaxis in Variceal Bleeding

The risk of rebleeding following the initial episode of hemorrhage due to the rupture of esophageal varices is 80%. Such rebleeding occurs most commonly within the first 6 weeks after initial bleeding. Secondary prophylaxis to prevent variceal rebleeding is indicated for patients who have cirrhosis or cavernous transformation of the portal vein. Patients who have portal hypertension due to

cavernous transformation of the portal vein have relatively normal liver parenchyma and function and tend to develop spontaneous portosystemic shunts over time. Thus, secondary prophylaxis bridges the time from presentation until spontaneous shunts form or until the patient's age and radiographic evaluation predict success from shunt surgery.

Secondary prophylaxis combines endoscopic and pharmacologic modalities. The endoscopic options include injection sclerotherapy and variceal band ligation. Successful obliteration of esophageal varices has been reported in 75% to 90% of pediatric patients following multiple sessions of sclerotherapy. Several controlled studies in adults have shown that band ligation has a higher efficacy in preventing rebleeding, fewer complications, lower costs, and higher rates of survival. Pediatric experience with this technique is encouraging. Case series in children have reported ablation of esophageal varices in fewer sessions compared with injection sclerotherapy. Medical therapy for secondary prevention of variceal rebleeding includes nonselective beta blockers to reduce cardiac output and splanchnic and portal blood flow, leading to reduced portal pressure. Several studies in adults show benefits of combined endoscopic and medical therapies in patients who have cirrhosis without increasing the risk of ascites or impaired renal function. Prophylactic use of beta blockers has not been studied rigorously in children.

Case Study Summary

The presented case illustrates the evaluation and management of rectal bleeding in a healthy infant who has altered bowel histology. In the absence of an anal fissure, significant skin hemangioma, and evidence of pathogens in the stool, the conventional approach to this infant is to make a presumptive diagnosis of allergic colitis and initiate therapy with a hypoallergenic formula (or eliminate all cow milk protein from a breastfeeding mother's diet). The presence of *C difficile* toxin can be a confounding variable; *C difficile* toxin has been found in the stool in 10% of healthy neonates. Most infants who have *C difficile* toxin in the stool are healthy, indicating the coexistence of some protective antitoxic substance or lack of appropriate toxin receptors in young infants. When a physician orders a test, he or she must have a clear idea of what to do with the information. In this case, the infant was healthy-appearing and feeding well, and screening blood study results were normal. Patients who have pseudomembranous colitis associated with *C difficile* appear ill and frequently present with high fever, leukocytosis, and hypoalbuminemia.

In this case, the patient was treated for a working diagnosis of allergic colitis. For patients who have allergic colitis, evidence of gross bleeding should resolve in 3 weeks, although heme-positive stools may persist for 6 to 12 weeks. Sigmoidoscopy is indicated if gross bleeding does not resolve in 3 weeks or occult bleeding does not resolve by 12 weeks. Nodular lymphoid hyperplasia is the most common cause of persistent visible blood in the stool of an infant who has allergic colitis.

Summary

GI bleeding can occur in any area of the GI tract, from the mouth to the anus. For severe bleeding, the sequence of management is first to stabilize the patient (including establishing intravenous access), followed by identifying the source of bleeding. The differential diagnosis is prioritized by addressing the clinical presentation of the bleeding and the age of the patient. Best outcomes are achieved by a timely multidisciplinary approach, using the combined skills of a pediatric gastroenterologist, radiologist, and surgeon. With the availability of a broad array of endoscopic and radiologic techniques for accurate diagnosis and the advent of innovative methods for controlling GI bleeding, the major challenge in the coming years will be to determine the optimal approach to the individual patient based on assessment of risk status. As described in the case study, management based on empiric diagnosis remains an acceptable approach for many of the conditions that cause GI bleeding in children.

Suggested Reading

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PIR Quiz
Quiz also available online at www.pedsinreview.org.

1. A previously healthy 2-year-old girl has been retching and vomiting for the past 12 hours. On the last occasion, streaks of bright red blood were noted in the pale yellow emesis. Findings on her examination are unremarkable. The *most* likely explanation of her hematemesis is:

   A. Esophageal varices.
   B. Hemorrhagic stress gastritis.
   C. Mallory-Weiss tear.
   D. Peptic ulcer.
   E. Vitamin K deficiency.

2. A previously healthy 5-year-old boy presents with the acute onset of maroon-colored hematochezia. Physical examination reveals a pale child who exhibits tachycardia. His mother reports that he has had occasional unexplained abdominal discomfort in the past that did not affect his activity. The *most* likely explanation of his symptoms is:

   A. Henoch-Schönlein purpura.
   B. Infectious colitis.
   C. Juvenile polyp.
   D. Meckel diverticulum.
   E. Superior mesenteric aneurysm.

3. A previously well 8-year-old boy has had diarrhea for the past 5 weeks, with occasional bright red and dark red blood mixed with the stool. Associated symptoms include episodic vomiting, decreased appetite, and a 4-lb weight loss. He has not taken any antibiotics in the past 6 months and has had no recent travel. Findings on physical examination include mild pallor and a small effusion in his right knee joint. His hemoglobin is 9.2 g/dL (92 g/L) and mean corpuscular volume is 72 fL (normal, 78 to 102 fL). Of the following, the *most* likely diagnosis is:

   A. Allergic colitis.
   B. Bacterial infectious colitis.
   C. Cytomegalovirus colitis.
   D. Pseudomembranous colitis.
   E. Ulcerative colitis.

4. A previously healthy 6-year-old girl has had small amounts of bright red blood mixed with her otherwise normal stools for the past 3 weeks. She generally has one soft stool each day. She has had no other symptoms. She has no known allergies to medications. You have seen her twice. On both occasions, findings on her examination have been normal. Her hemoglobin is 11.3 g/dL (113 g/L). Effective treatment *most* likely will involve:

   A. A 10-day course of oral penicillin.
   B. An oral proton pump inhibitor.
   C. Removal of milk from her diet.
   D. Resection of a Meckel diverticulum.
   E. Snare polypectomy.

5. A previously healthy 9-year-boy's conjunctivae appear pale during a health supervision visit. His mother reports that he has had episodic blood in his stool over the past 2 to 3 months, which she assumed had been caused by hard stool associated with constipation. He has no other symptoms. Physical examination reveals brown-black 1- to 2-mm macules on his lips. His hemoglobin is 9.1 g/dL (91 g/L). Indices are consistent with iron deficiency anemia. A normal-appearing stool is guaiac-positive. The *most* likely cause of the gastrointestinal blood loss is:

   A. Colon polyp.
   B. Hemangioma.
   C. Intestinal neurofibroma.
   D. Rectal prolapse.
   E. Solitary rectal ulcer.

Gastrointestinal Bleeding in Infants and Children
John T. Boyle

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