Consultation with the Specialist: Upper Gastrointestinal Hemorrhage

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Upper Gastrointestinal Hemorrhage

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The new onset of gastrointestinal hemorrhage in a child is an extremely frightening event for both the patient and his or her parents. These patients usually are brought to the physician promptly upon the onset of bleeding because many parents have had experiences with adult relatives or friends who have bled from gastrointestinal malignancies. In addition to assessing the patient's condition rapidly and inquiring about past medical history, the clinician must be prepared to answer honestly the many questions the parents will raise and to address their fears directly. Three basic questions must be answered initially, as soon as the patient's clinical condition is stabilized:

1) Is this material, in fact, blood and, if it is, is it the patient's blood?
2) Does this represent an upper or lower gastrointestinal hemorrhage?
3) What is the specific diagnosis and site of hemorrhage?

In this review, we focus on upper gastrointestinal hemorrhage, usually defined as bleeding from a site proximal to the ligament of Treitz. Approximately 20% of all gastrointestinal bleeding in children arises at these sites. Affected patients usually present with either hematemesis or melena. Occasionally, upper gastrointestinal hemorrhage in the infant, who has rapid gastrointestinal motility, may present as passage of bright red blood from the rectum.

Editorial Board.

Evaluation

The primary focus of the initial evaluation of a patient who has upper gastrointestinal bleeding is resuscitation and stabilization of the child. Patients who have had a significant episode of bleeding should have a large-bore intravenous line established and receive 10 to 40 mL/kg of Ringer lactate solution, depending on initial vital signs and clinical condition. As much as 15% of the child's circulating blood volume may be lost without any hemodynamic changes. When more than 15% of the volume is lost, the first compensatory mechanism is tachycardia. Not until more than 30% of volume has been lost does systemic hypotension become evident.

Blood should be drawn when the intravenous catheter is placed and sent for typing and cross-matching as well as measurement of hemoglobin level, hematocrit, platelet count, serum electrolyte concentrations, and blood urea nitrogen/creatinine and a coagulation profile. Obtaining a thorough history of the bleeding episode, including an estimate of the amount of blood lost, can help establish the cause and source of the hemorrhage. Minor episodes of hematemesis that involve little or no pain may reflect gastritis due to ingestion of medication or esophagitis from gastroesophageal reflux. More substantial hematemesis may be the result of bleeding from esophageal varices or peptic ulcer disease. The patient's age at initial presentation often helps to define the etiology of the bleeding (Table 1).

A careful physical examination may provide further clues to the diagnosis. Examination of the mucous membranes of the nose and throat will rule out a nasopharyngeal source of the blood. The presence of hepatosplenomegaly may indicate portal hypertension and bleeding from esophageal varices, while epigastric tenderness might suggest peptic ulcer disease. Because many causes of upper gastrointestinal hemorrhage may present with either melena or passage of bright red blood from the rectum, examination of a nasogastric aspirate is essential to establish an upper intestinal origin of the rectal bleeding. Children who have upper gastrointestinal hemorrhage and present with passage of bright red blood from the rectum usually have a major bleeding episode. They must be treated aggressively with intravenous fluids and prepared for possible blood transfusion. Patients who have bright red blood in the nasogastric aspirate should receive gastric lavage with saline to evacuate blood clots from the stomach and to allow gastric contraction. The use of iced saline offers no advantage in controlling bleeding and may cause profound hypothermia in young children.

The first consideration in evaluating children who have new-onset "bleeding" is to determine whether actual blood is in the material passed. Several foods ingested by infants and children may give the appearance of blood in the emesis or stool. Certain food-coloring agents such as those found in many popular gelatins and fruit juices appear

TABLE 1. Upper Gastrointestinal Hemorrhage: Age and Diagnosis

Neonate
- Maternal blood
- Gastritis
  - — Stress
  - — Sepsis
  - — Cow milk intolerance
  - — Trauma from nasogastric tube insertion
- Necrotizing enterocolitis
- Coagulation disorders

1 Month to 1 Year
- Substantial Hemorrhage
  - — Peptic ulceration
  - — Curling ulcer
  - — Duplication cyst
  - — Foreign body
- Mild Hemorrhage
  - — Reflux esophagitis
  - — Gastritis
    - Stress
    - Medication—acetylsalicylic acid (ASA), nonsteroidal anti-inflammatory agents (NSAIDS)
    - Caustic ingestion

3 to 5 Years
- Peptic ulceration
- Gastritis—ASA, NSAIDs
- Varices
- Epistaxis
- Mallory-Weiss tear

>5 Years
- Varices
- Peptic ulceration
- Coagulation disorders
  - — Immune thrombocytopenic purpura
  - — Chemotherapy

bright red when vomited. Ingestion of iron supplements or bismuth may cause dark stools or "melena". Use of the Hemoccult® test on stool is accurate in detecting blood, but this test is inactivated by acid and is not as effective for detecting blood in emesis or nasogastric aspirate. For this assay, the Gastroccult® test is more accurate.

If blood is identified, it must be determined whether it is the patient's blood. Hematemesis in the newborn simply may represent regurgitation of maternal blood swallowed during delivery. Breast-fed infants may ingest small amounts of maternal blood from irritated nipples, which may present as hematemesis or melena. In these clinical situations, it is important to differentiate between adult and fetal hemoglobin. Fetal hemoglobin is resistant to denaturization by alkali. The Apt-Downey Test is used to differentiate between these two forms of hemoglobin and to confirm swallowed maternal blood in these patients (Table 2).

If the material in the emesis or stool is, in fact, the patient's own blood, the clinician must establish that the blood is from the gastrointestinal tract. Older children who have epistaxis may present with hematemesis due to ingested blood from the nose or pharynx. A careful history and physical examination usually can make this differential diagnosis.

Diagnostic Tests

The single most important diagnostic test for children who have upper gastrointestinal hemorrhage is esophagogastroduodenoscopy. Prior to the introduction of flexible endoscopy in the 1970s, most cases of upper intestinal bleeding in children went undiagnosed. In the hands of a skilled endoscopist, this procedure now can diagnose the cause of upper gastrointestinal hemorrhage correctly in more than 90% of affected patients. Complementary use of upper gastrointestinal radiography allows determination of a specific diagnosis in virtually all affected children.

Most patients who have an episode of significant upper intestinal bleeding that involves changes in vital signs indicating significant volume loss should undergo flexible endoscopy as soon as the bleeding is controlled and before the use of contrast radiography is considered. The presence of contrast material in the stomach and duodenum at the time of endoscopy often obscures the source of bleeding. Endoscopy usually is more sensitive at diagnosing bleeding from superficial sources, such as esophagitis and gastritis. Contrast radiography, which is less invasive and expensive than endoscopy, can detect major sources of bleeding, such as duodenal ulcers or esophageal varices. The use of double-contrast radiography may enhance the diagnostic yield of this procedure.

Occasionally, children who have massive upper gastrointestinal hemorrhage require angiography either for definitive diagnosis or for therapy. Arteriography performed through the celiac trunk and superior

TABLE 2. Apt-Downey Test

1. Mix stool or emesis with water (1:5)
2. Centrifuge mixture
3. Add 1 mL 0.25N sodium hydroxide to 5 mL supernatant and wait 5 min
4. Brown-yellow color indicates adult hemoglobin; pink color indicates fetal hemoglobin

mesenteric artery visualizes the vessels supplying the intestinal tract from the gastroesophageal junction to beyond the ligament of Treitz. These studies may detect bleeding hemangiomas and arteriovenous malformations of the intestine, which often are difficult to diagnose by endoscopy or contrast radiography. A rate of bleeding of 0.5 to 1.0 mL/min is required to visualize the site of bleeding by arteriography. The venous phase of arterial injections demonstrates the anatomy of the portal vein and may visualize gastric and esophageal varices. Selective embolization of bleeding vessels at the time of arteriography or the intra-arterial infusion of vasopressin (0.005 U/kg per minute) may control bleeding in these children. Nuclear medicine studies, using radiolabeled red blood cells, have little role in the evaluation of upper gastrointestinal bleeding in children.

Etiology and Management

NEONATE

The etiology of gastrointestinal bleeding in children and its presentation are definitely age-related (Table 1). Although upper gastrointestinal bleeding usually presents with hematemesis, the gastrointestinal motility in neonates and small infants is sufficiently rapid that such bleeding may present as passage of bright red blood from the rectum. In these patients, examination of the gastric aspirate may provide the evidence of an upper gastrointestinal source. Once ingestion of maternal blood has been eliminated, most bleeding in the neonate can be traced to superficial gastroduodenal ulceration induced by sepsis or stress. This diagnosis often is made on the basis of the history and physical examination; endoscopy is not needed for confirmation.

Infants who have simple stress-related gastritis may be treated with oral or intravenous histamine-2 (H-2) receptor antagonists, such as ranitidine (6.0 mg/kg per day BID). Patients who have active bleeding require a continuous infusion of ranitidine (0.1 to 0.25 mg/kg per hour). Preterm neonates who have necrotizing enterocolitis often present with bright red blood in the nasogastric aspirate. The bleeding rarely is severe, and the etiology usually is evident from a careful history and the presence of abdominal distention and a rapidly deteriorating clinical course. The diagnosis of necrotizing enterocolitis is confirmed by the detection of pneumatosis intestinalis on plain abdominal radiographs. Further diagnostic studies frequently are unnecessary. Treatment involves nasogastric decompression and aggressive fluid management in conjunction with the use of broad-spectrum antibiotics. Surgery may be necessary for infants who have transmural necrosis of the intestine. Rarely, intestinal duplications of the proximal gastrointestinal tract may present with hemorrhage. Usually, these lesions are suspected by the presence of signs of gastrointestinal tract obstruction and an abdominal mass. The diagnosis is confirmed by upper gastrointestinal contrast radiography or computed tomography of the abdomen.

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AGES 1 TO 3 YEARS

Upper gastrointestinal tract bleeding in children between the ages of 1 and 3 years of age often is caused by peptic ulceration of the esophagus, stomach, or duodenum. The use of certain medications in children of this age, such as aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), or steroids, may precipitate gastric ulceration. Bleeding usually is self-limited and responds to gastric lavage, discontinuation of the offending medications, and the use of H-2 receptor antagonists.

The source of bleeding in these patients usually can be identified by flexible endoscopy, although drug-induced ulceration generally is evident from findings on the history and does not require endoscopic confirmation. Active bleeding from gastric or duodenal ulcers visualized at the time of endoscopy can be controlled with thermal probes or laser coagulation. Biopsies of the antrum and body of the stomach should be obtained to examine for evidence of *Helicobacter pylori* infection.

Treatment of children in the 1- to 3-year-old age group consists of H-2 receptor antagonists. If *H pylori* is identified, bismuth and clarithromycin (15 mg/kg per day) also should be administered. Occasionally, repeated vomiting from a gastrointestinal viral infection may induce Mallory-Weiss tears of the esophagus or esophagogastric junction. This bleeding generally is controlled with antiemetics and H-2 receptor antagonists.

AGE MORE THAN 3 YEARS

Upper gastrointestinal bleeding in children older than the age of 3 years may be more difficult to manage. These patients may experience bleeding from esophageal varices or chronic duodenal ulcers. In addition, children who have leukemia or idiopathic thrombocytopenic purpura may present with bleeding from a severe coagulopathy.

The patient should be evaluated endoscopically as soon as he or she is stabilized and the coagulopathy, if present, corrected. Esophageal varices in these patients may be caused by extrahepatic portal vein thrombosis or by cirrhosis of the liver due to biliary atresia or cystic fibrosis. Bleeding from esophageal varices is rare in the first year of life. The onset of bleeding depends on the progression of portal hypertension. Most affected patients begin bleeding by 10 years of age. Variceal bleeding may be precipitated by a generalized viral syndrome or the ingestion of aspirin. Endoscopy is especially important in these children because 50% of patients who have known varices will be bleeding from a source other than the varices, usually a peptic ulcer.

Variceal bleeding often can be controlled by the use of sclerotherapy performed at the time of diagnostic endoscopy. If the bleeding is too rapid to allow sclerotherapy,

intravenous or intra-arterial vasopressin may be administered. Recently, the use of intravenous somatostatin has been shown to be effective in controlling variceal bleeding in approximately 80% of patients. A loading dose of 1 to 2 mcg/kg is administered intravenously over 2 to 5 minutes, followed by a continuous infusion of 1 to 2 mcg/kg per hour. This infusion may be started prior to sclerotherapy if the diagnosis is confirmed and endoscopy is not anticipated. The use of a Sengstaken-Blakemore tube no longer is recommended for children, except in desperate cases, because of complications associated with its use. Because prolonged use of vasopressin may cause significant retention of fluid, central venous pressure and urine output must be monitored carefully. The use of somatostatin appears to be associated with fewer hemodynamic alterations.

Children within this age range in whom the bleeding is from peptic ulcer disease should be treated with prolonged H-2 antagonist therapy. Occasionally, children of this age will experience "hematemesis" due to swallowed blood from a nosebleed or bleeding from the tonsillar area. A careful history and physical examination usually reveals the true source of this bleeding.

Conclusion

The clinician presented with a child who has new onset of upper gastrointestinal hemorrhage must approach resuscitation and evaluation in an organized manner. A relatively small number of diagnostic tests are required, with emphasis on the use of upper gastrointestinal flexible endoscopy. Knowledge of the age-dependent diagnoses in pediatric patients is helpful in establishing a specific source of bleeding. Once the diagnosis is confirmed, most affected children can be treated successfully with relatively simple maneuvers.

SUGGESTED READING

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