Approach to upper gastrointestinal bleeding in children

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INTRODUCTION

— Upper gastrointestinal (UGI) bleeding (arising proximal to the ligament of Treitz in the distal duodenum) commonly presents with hematemesis (vomiting of red blood or coffee ground-like material) and/or melena (black, tarry stools). In comparison, hematochezia (bright red or maroon-colored blood or fresh clots per rectum) is usually a sign of a lower gastrointestinal (LGI) source (defined as distal to the ligament of Treitz).

The initial approach to a child with suspected UGI bleeding is discussed in this topic review. The approach to LGI bleeding in children, or to UGI bleeding in adults, is reviewed separately. (See "Lower gastrointestinal bleeding in children: Causes and diagnostic approach" and "Approach to acute upper gastrointestinal bleeding in adults".)

EPIDEMIOLOGY

— The incidence of UGI bleeding is not well established in children. As much as 20 percent of all episodes of gastrointestinal (GI) bleeding in children come from a UGI source [1]. A population-based study from France estimated that UGI bleeding occurred in 1 to 2 per 10,000 children per year (77 percent of whom required hospitalization) and that exposure to nonsteroidal antiinflammatory drugs (NSAIDs) played a role in 36 percent of these cases [2].

The most detailed studies have been in the critical care setting [3-5]. In one of the largest prospective studies, UGI bleeding (defined for this study as hematemesis or any amount of blood from the nasogastric tube) was observed in 63 of 984 (6.4 percent) pediatric intensive care unit admissions [4]. Independent risk factors for bleeding included a high Pediatric Risk Mortality score, coagulopathy, pneumonia, and multiple trauma. Higher rates of bleeding (up to 25 percent) were observed in two other series of critically ill pediatric patients who were not receiving prophylactic therapy to prevent UGI bleeding [3,5].

ETIOLOGY

— The most common causes of UGI bleeding in children vary depending upon age and the geographic setting. In Western countries, the most common causes are gastric and duodenal ulcers, esophagitis, gastritis, and varices [6-8]. In India and some other parts of the world, variceal bleeding predominates [9,10]. These observations may reflect regional differences in indications for endoscopy, in addition to differences in predisposing conditions. Conditions associated with structural abnormalities of blood vessels (such as hereditary hemorrhagic telangiectasia and Ehlers-Danlos syndrome) and congenital or acquired coagulopathies can produce bleeding at any time of life [11].

Neonates

— UGI bleeding is rare in the first month of life but may occur for a variety of reasons (table 1) [12-25]. Important considerations include:

- True UGI bleeding in a neonate must be distinguished from swallowed maternal blood. (See 'Differential diagnosis' below.)
- Vitamin K-deficient bleeding (hemorrhagic disease of the newborn) should be considered in neonates who were not given vitamin K prophylaxis at birth. (See "Overview of vitamin K", section on 'Vitamin K deficient bleeding in newborns and young infants'.)
- Stress gastritis or ulcers are associated with critical illness but also may occur spontaneously even in the first few days of life [16], in rare instances.
- Congenital anomalies including intestinal duplications or vascular anomalies may present with gastrointestinal (GI) hemorrhage [19,23,26].
- Coagulopathy in a neonate may also be caused by infection, liver failure, or a congenital coagulation factor deficiency. Several types of coagulopathies can present during the newborn period. Most present with other bleeding symptoms, such as a large cephalohematoma after vaginal delivery, oozing from the umbilical stump, prolonged bleeding after circumcision or blood sampling, or intracranial hemorrhage in a term infant [27]. Occasionally, these coagulopathies come to medical attention because of GI bleeding, but this rarely occurs during the neonatal period.
- Milk protein intolerance may present with UGI bleeding, although lower gastrointestinal (LGI) bleeding is much more common. Care must be taken to exclude other causes of UGI bleeding, as outlined below, because UGI bleeding or passage of blood per rectum alone is

not diagnostic of milk protein intolerance. (See "Food protein-induced allergic proctocolitis of infancy".)

Infants, children, and adolescents

— The spectrum of causes of UGI bleeding in children is similar to that in adults, although some risk factors vary with age (table 1 and table 2) [12,25]. Particular considerations include:

- Mallory-Weiss syndrome – Mallory-Weiss syndrome is characterized by longitudinal mucosal lacerations in the distal esophagus, usually developing after forceful retching. The bleeding is usually small and self-limited but occasionally is severe. (See "Mallory-Weiss syndrome".)

- Esophageal or GI foreign body – A foreign body can cause GI bleeding if it is sharp, caustic, and/or lodged in the esophagus. Clinical clues to this possibility include a history of a choking episode, even if it was transient or occurred days or even weeks before the bleeding episode. Rarely, ingestion of a button battery has led to severe UGI hemorrhage due to aortoesophageal fistula, which can be fatal [28]. (See "Foreign bodies of the esophagus and gastrointestinal tract in children" and "Button and cylindrical battery ingestion: Clinical features, diagnosis, and initial management".)

- Esophagitis – Esophagitis in this age group usually is caused by gastroesophageal reflux disease or eosinophilic esophagitis and occasionally by caustic ingestion. Peptic esophagitis also may develop after recurrent vomiting from other causes. (See "Clinical manifestations and diagnosis of gastroesophageal reflux disease in children and adolescents" and "Caustic esophageal injury in children" and "Clinical manifestations and diagnosis of eosinophilic esophagitis".)

- Peptic ulcers and gastritis – Gastritis and ulcers occasionally occur in all age groups, typically in the setting of critical illness or use of nonsteroidal antiinflammatory drugs (NSAIDs). Young children are particularly susceptible to developing UGI bleeding after NSAID use [2]. Binge drinking of alcohol is an important cause of gastritis in adolescents. Gastritis or peptic ulcers also may be related to *Helicobacter pylori* infection or, occasionally, to a viral infection including cytomegalovirus [25]. In these disorders, epigastric discomfort and/or vomiting typically precede the hematemesis, which usually is low grade. (See "Causes of acute abdominal pain in children and adolescents", section on 'Peptic ulcer disease' and "Chronic abdominal pain in children and adolescents: Approach to the evaluation".)

- Pill esophagitis – Pill esophagitis is caused by direct injury to the esophageal mucosa from prolonged contact with certain drugs, including tetracyclines (doxycycline and minocycline) that are commonly used for treatment of acne, or by NSAIDs including aspirin, or with the use of bisphosphonates. The condition presents with pain with swallowing (odynophagia)

and may progress to hematemesis. Similar symptoms may be caused by infectious esophagitis (due to *Candida*, cytomegalovirus, or herpes simplex). (See "Medication-induced esophagitis" and "Clinical manifestations and diagnosis of gastroesophageal reflux disease in children and adolescents", section on 'Dysphagia or odynophagia'.)

- Bleeding from esophageal varices – Variceal bleeding is the most common cause of **severe** acute UGI bleeding in children. Esophageal varices are caused by portal vein hypertension. Clues to portal hypertension include splenomegaly and/or a history of thrombocytopenia, even in a patient without a history of liver disease. Causes of portal hypertension include:
  - Cirrhosis due to chronic liver disease (eg, cystic fibrosis-related liver disease, biliary atresia, or intestinal failure-associated liver disease). (See "Cystic fibrosis: Hepatobiliary disease" and "Biliary atresia" and "Intestinal failure-associated liver disease in infants".)
  - Portal vein thrombosis is most commonly associated with a history of umbilical vein catheterization or sepsis during the neonatal period. In some patients, the disorder first presents as an acute variceal bleed, which can be severe [29]. (See "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management".)
  - Hepatic vein obstruction (Budd-Chiari syndrome). (See "Budd-Chiari syndrome: Epidemiology, clinical manifestations, and diagnosis".)
- Arterial bleeding – Rarely, severe acute UGI bleeding is from an artery, either from an overlying peptic ulcer or a Dieulafoy lesion [30,31]. (See "Causes of upper gastrointestinal bleeding in adults", section on 'Vascular lesions'.)

Unusual causes of UGI bleeding have been described in case reports, including hemangiomas [23,32], aortoesophageal fistulas [28,33], hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome) [34], Kasabach-Merritt syndrome [35], duplication cysts [26,36], parasites [37], vasculitis [38], gastric polyps, annular pancreas [39], antral or duodenal web [40,41], and systemic mastocytosis [12,25,42].

Differential diagnosis

— The differential diagnosis of UGI bleeding includes vomited blood originating from structures or organs other than the GI tract and ingested blood or blood-like substances:

- Swallowed maternal blood – Neonates and infants may swallow maternal blood during delivery or while nursing, and this can mimic UGI bleeding [43]. One method with which to distinguish maternal blood is the Apt-Downey test. Fetal hemoglobin is resistant to denaturation in an alkaline solution and remains red or pink, while adult hemoglobin discolors to a brownish yellow. The Apt-Downey test is valid for only the first few months of

life because the percentage of fetal hemoglobin decreases sharply during this period. (See "Lower gastrointestinal bleeding in children: Causes and diagnostic approach", section on 'Swallowed maternal blood'.)

- Epistaxis – Swallowed blood from the patient's nasopharynx or respiratory tract may be very difficult to distinguish from UGI bleeding. This source should be considered in a child with a history of recent or recurrent epistaxis. To evaluate this possibility, the physical examination should include inspection of the nares for evidence of venous injury of the anterior medial septums. Some patients may require endoscopic evaluation to adequately assess for lesions in the nasopharynx, larynx, or respiratory tract. (See "Evaluation of epistaxis in children".)

- Substances that resemble blood – Red food colorings and dyes (eg, red-colored drinks or liquid medications) also may be confused with blood, particularly after vomiting. While this can frequently be suspected from this history, bedside tests for occult blood also can be helpful. These tests rely on color changes to detect the presence of hemoglobin. However, some of the commercial tests (such as Hemoccult) may produce false-negative results in the presence of acid. It is preferable to use a kit designed specifically to detect blood in gastric secretions (eg, Gastroccult), which incorporate additional alkali to neutralize the gastric acid present in emesis [44].

- Medical child abuse – Factitious illness (Munchausen syndrome by proxy), caused by surreptitious administration of blood or blood-like substance to simulate UGI bleeding, should be considered in patients with unexplained GI bleeding [45]. The patient or siblings may have a history of frequent recurrent illnesses without a clear etiology. (See "Medical child abuse (Munchausen syndrome by proxy)".)

CLINICAL PRESENTATION

— UGI bleeding typically presents with hematemesis (vomiting of blood or coffee ground-like material) and/or melena (black, tarry stools).

- Hematemesis – Bright red blood usually indicates brisk or very fresh bleeding. Coffee ground-like material generally indicates a slower rate of bleeding because this appearance is caused by the effect of gastric acid on blood. This effect can be altered with acid suppression therapy.

- Stool – UGI bleeding tends to be associated with melena (dark red or black and sticky stools), while lower gastrointestinal (LGI) bleeding tends to be associated with hematochezia (bright red in color). However, these distinctions in stool color are not absolute, because melena can be seen with proximal LGI bleeding and hematochezia can be seen with massive UGI bleeding. Because of short intestinal transit time, neonates and infants with UGI bleeding are

more likely than adults to present with hematochezia. (See "Lower gastrointestinal bleeding in children: Causes and diagnostic approach", section on 'Diagnostic approach'.)

CLINICAL ASSESSMENT

— The initial evaluation of the patient with UGI bleeding should always start with an assessment of hemodynamic stability and resuscitation, if indicated. Rapid assessment and early resuscitation are particularly important in children. Diagnostic studies follow, with the goal of diagnosing the cause of the bleed. Endoscopy usually is indicated if the bleeding is brisk or unexplained after a thorough history and physical examination, or if there are associated signs of shock. Brisk bleeding is suggested by repeated episodes of grossly bloody emesis or large volumes of blood aspirated through a nasogastric tube, or a persistent drop in hemoglobin. In some cases, the source of the bleeding can be treated through the endoscopic procedure. Endoscopic evaluation and treatment should generally be performed after the patient is stabilized and within 24 to 48 hours of presentation of the gastrointestinal (GI) bleed.

Initial assessment and resuscitation

— Vital signs, including the heart rate, blood pressure, presence of orthostatic changes, and capillary refill, are used to assess and monitor the hemodynamic state of the patient. Patients with hemodynamic instability (shock, orthostatic hypotension) should be admitted to an intensive care unit for resuscitation and close observation. Such patients should be stabilized prior to endoscopy. Both a gastroenterologist and a surgeon should be notified promptly of all patients with severe acute UGI bleeding.

Clinical features suggesting a severe UGI bleed are [46,47]:

- Melena or hematochezia
- Heart rate >20 beats per minute above the mean heart rate for age
- Prolonged capillary refill time
- Decrease in hemoglobin of more than 2 g/dL
- Need for fluid bolus
- Need for blood transfusion (given if hemoglobin <8 g/dL)

The principles involved in resuscitation of children with hemorrhagic shock including UGI bleeding are discussed separately. (See "Hypovolemic shock in children: Initial evaluation and management", section on 'Fluid resuscitation'.)

Obtaining intravenous (IV) access can be difficult, especially in severely volume-depleted patients. Intraosseous fluids or central venous access may be required in this setting. (See

"Intraosseous infusion".)

History

— The clinical history should include information concerning the time course of the bleeding episode, estimated blood loss, and any associated symptoms (table 3). The presence of hematemesis, melena, or hematochezia should be documented; these characteristics provide clues about the source and rate of bleeding (see 'Clinical presentation' above). Particular attention should be given to GI symptoms including dyspepsia, heartburn, abdominal pain, dysphagia, and weight loss. In infants, these features may be reflected in poor feeding and irritability.

The history should also include information about the following symptoms or signs, which may provide clues to an underlying disorder:

- Recent onset of jaundice, easy bruising, or change in stool color, which may suggest underlying liver disease
- Recent or recurrent epistaxis, to investigate the possibility of a nasopharyngeal source of bleeding
- History of easy bruising or bleeding, which suggests a disorder of coagulation, platelet dysfunction, or thrombocytopenia
- Personal or family history of liver, kidney, or heart disease, or coagulation disorders

A drug history is important to assess potential contributions from medications that may induce ulceration (such as nonsteroidal antiinflammatory drugs [NSAIDs] and corticosteroids); even short-term use of ibuprofen can cause gastric ulcers and hematemesis [48]. Tetracyclines, which are commonly used to treat acne, may cause a pill esophagitis (see "Medication-induced esophagitis"). Some medications, such as NSAIDs, also affect coagulation and can exacerbate bleeding from another cause. Alcohol ingestion (in particular, binge drinking) and tobacco use can contribute to peptic ulcer disease, and large intakes of caffeine (eg, from coffee or caffeinated sodas) can promote acid secretion and dyspepsia. (See "Peptic ulcer disease: Genetic, environmental, and psychological risk factors and pathogenesis", section on 'Factors that influence the course of peptic ulcer'.)

In addition, it is important to know if the patient has been taking drugs or has a cardiac condition that affects homeostatic responses (such as beta-adrenergic antagonists) because these may mask tachycardia associated with life-threatening hypovolemia and shock.

Physical examination

— The rapid assessment of hemodynamic status is described above. The remainder of the physical examination should include the following elements, which suggest possible sources for

the bleeding (table 4):

- Examination of the skin and mucus membranes for bruising, petechiae, or mucosal bleeding. Depending on the presentation and pattern, these findings may suggest a bleeding disorder (eg, immune thrombocytopenia [ITP]), trauma, or liver disease.
- Examination of the skin for cutaneous signs of generalized vascular malformations/disorders. The presence of cutaneous hemangiomas (especially five or more) suggests the possibility of GI hemangiomatosis. However, up to 50 percent of infants with visceral hemangiomas do not have cutaneous hemangiomas [32]. Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome) is characterized by mucocutaneous telangiectasia (picture 1) and commonly presents with recurrent epistaxis and/or GI bleeding. (See "Infantile hemangiomas: Epidemiology, pathogenesis, clinical features, and complications" and "Clinical manifestations and diagnosis of hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)".)
- Abdominal examination for evidence of portal hypertension, such as splenomegaly or prominent cutaneous abdominal and hemorrhoidal vessels and a protruding abdomen. Portal hypertension often leads to esophageal varices, which may hemorrhage.
- Inspection of the nasopharynx for evidence of disrupted mucosa or inflamed tonsils suggesting swallowed blood, and inspection of the anterior nares for evidence of venous injury of the anterior medial septum. If present, these findings suggest the possibility of a nasopharyngeal rather than UGI bleed. (See 'Differential diagnosis' above and "Evaluation of epistaxis in children".)

In patients presenting with unexplained GI bleeding that is clinically significant, nasogastric or orogastric tube lavage is often used to confirm the diagnosis and to determine if the bleeding is ongoing. (See 'Nasogastric tube' below.)

Laboratory evaluation

— The laboratory assessment depends on the clinical scenario and magnitude of the blood loss. In most cases, it should include a complete blood count, coagulation studies, tests evaluating liver function, blood urea nitrogen (BUN), and serum creatinine. For patients with epigastric abdominal pain, pancreatitis also should be ruled out with screening amylase and lipase; pancreatitis occasionally is associated with gastritis, duodenitis, and peptic ulcer disease. For patients with clinically significant bleeding or known varices, a specimen should be drawn to type and cross-match blood in case transfusion is required. Less extensive laboratory evaluation may be appropriate for patients with small amounts of blood in the vomitus and a likely explanation.

The BUN result can be helpful for confirming the source of bleeding. An increase in BUN in the absence of renal disease is consistent with a UGI (rather than a lower gastrointestinal [LGI]) source of blood loss because blood in the proximal GI tract has relatively more time to be absorbed, leading to an increase in the BUN. However, a normal or low BUN does not rule out a UGI bleed.

Imaging

— Imaging tests can be helpful in specific clinical settings:

- Plain radiographs may be helpful to identify a foreign body if this is suspected by the clinical history. They are also helpful to evaluate for bowel obstruction or perforation in patients with significant abdominal pain, distension, or tenderness.
- Abdominal ultrasound can be used to evaluate splenomegaly and portal hypertension and should be performed in patients with severe acute UGI bleed suggestive of variceal bleeding, known or suspected liver disease, or those with signs of portal hypertension on examination (eg, splenomegaly, prominent abdominal wall vessels).
- UGI barium studies should **not** be performed in the setting of UGI bleeding, because the contrast will interfere with subsequent endoscopy, angiography, or surgery.

Endoscopy

— Endoscopy is often required in patients with UGI bleeding for both diagnostic and therapeutic purposes, as discussed below. (See 'Endoscopy' below.)

Other diagnostic tests

— Rarely, angiography or radionucleotide imaging may be needed to identify a source of bleeding.

- Angiography may be useful in patients with rapid bleeding in whom endoscopy is unsuccessful in finding a source. For diagnostic purposes, magnetic resonance angiography (MRA) or computed tomographic angiography (CTA) may be used [49,50]; the preferred method may be specific to a particular institution. For therapeutic purposes, standard angiography may be useful in treating some patients with vascular anomalies, hemobilia, or some ulcers that are not amenable to other types of treatment [51]. (See "Angiographic control of nonvariceal gastrointestinal bleeding in adults".)
- Radionucleotide imaging (a tagged red blood cell scan) also can be used to detect an obscure bleeding source for patients with very brisk bleeding. However, this is rarely helpful in

patients with UGI bleeding because endoscopy is far more sensitive for detecting bleeding above the ligament of Treitz. (See "Evaluation of occult gastrointestinal bleeding".)

MANAGEMENT

— For hemodynamically stable patients with small amounts of blood in the vomitus with a likely explanation (eg, Mallory-Weiss tear), supportive care with observation generally is sufficient, usually with acid suppression to treat any peptic component and reduce the risk for rebleeding. For patients with larger amounts of blood in vomitus or hemodynamic instability, the following interventions may be used:

Nasogastric tube

— In patients presenting with unexplained gastrointestinal (GI) bleeding that is clinically significant (eg, more than a teaspoon estimated blood loss), nasogastric or orogastric tube lavage is often used to confirm the diagnosis and to determine if the bleeding is ongoing. This approach is particularly helpful if the bleeding is suspected to be a vascular bleed (eg, variceal). The lavage will also remove particulate matter, fresh blood, and clots to facilitate endoscopy and decrease the risk of aspiration. The lavage may be performed with either water or normal saline, at room temperature. Ice water lavage is **not** recommended; this older practice does not slow bleeding [52] and may induce iatrogenic hypothermia, particularly in infants and small children.

If the lavage returns fresh blood or blood with the appearance of coffee grounds, this helps to confirm a UGI (or nasopharyngeal) source of bleeding. However, lavage may not be positive if the bleeding has ceased or arises beyond a closed pylorus. The presence of bilious fluid suggests that the pylorus is open and if lavage is negative, that there is no active UGI bleeding.

Pharmacologic options

— Acid suppression usually is appropriate for children with clinically significant UGI bleeding to treat or prevent any peptic component of the underlying disorder. Vasoactive agents may be helpful for selected cases of vascular bleeding (eg, from esophageal varices). The most commonly used drugs and doses are summarized in the table (table 5).

Acid suppression

— We suggest acid suppression for children with UGI bleeding. For hemodynamically unstable children or those with large-volume bleeding, we suggest an intravenous (IV) preparation of a proton pump inhibitor (PPI; esomeprazole or pantoprazole) or histamine 2 receptor antagonist (eg, ranitidine, famotidine, or cimetidine). For hemodynamically stable children with mild bleeding, we suggest oral administration of a PPI such as omeprazole or esomeprazole.

This suggestion for acid suppression is primarily based on studies in adults, which have examined the effect of acid suppression given before or after endoscopy (with or without therapeutic intervention). In the setting of active UGI bleeding due to peptic ulcers, high-dose antisecretory therapy with an IV infusion of PPI significantly reduced the rate of rebleeding compared with standard treatment in patients with bleeding ulcers. Oral and IV PPI therapy also decrease the hospital stay, rebleeding rate, and the need for blood transfusion in adult patients with high-risk ulcers treated with endoscopic therapy. (See "Approach to acute upper gastrointestinal bleeding in adults", section on 'Acid suppression'.)

Somatostatin and octreotide

— Octreotide (an analog of somatostatin) may help in reducing or temporizing GI bleeding in selected cases of variceal bleeding that are difficult to control as adjunctive therapy to help control bleeding before endoscopy or when endoscopy is unsuccessful, contraindicated, or unavailable [53,54]. Octreotide reduces portal venous inflow and intravariceal pressure; these medications reduce the risk of rebleeding in adult patients with variceal hemorrhage. In addition, these medications may reduce the risk of bleeding due to nonvariceal causes [55]. (See "Overview of the treatment of bleeding peptic ulcers" and "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

Guidelines for the use of octreotide in the pediatric age group have not been developed, but octreotide is commonly administered as an initial bolus of 1 to 2 microgram/kg body weight (maximum 100 micrograms), followed by 1 to 2 microgram/kg/hour as a continuous IV infusion [54]. The infusion rate is titrated to the response. The optimal duration of therapy is unclear. Adverse effects include bradycardia and hyperglycemia. If bleeding stops, octreotide doses are typically tapered gradually over approximately 24 hours. Discovering and treating the cause of the GI bleed should remain the principal goal.

Experience with octreotide in children is limited [53]. Some providers prefer octreotide over vasopressin for the treatment of acute variceal hemorrhage because it appears to have better efficacy and fewer side effects, but data on either of these drugs in children are limited. (See "Methods to achieve hemostasis in patients with acute variceal hemorrhage", section on 'Pharmacologic therapy'.)

Endoscopy

— Guidelines recommend that endoscopy be performed within 24 to 48 hours for infants and children presenting with UGI bleeding that is acute and severe, particularly if transfusions are required [56]. Earlier endoscopy may be needed if bleeding cannot be controlled. Hemodynamically unstable patients should be stabilized prior to endoscopy, including transfusion

and correction of coagulopathy if present. Endoscopy is also appropriate in children with low-grade bleeding that is unexplained and persistent or recurrent [12].

Upper endoscopy permits identification of the bleeding source, allows for risk stratification regarding the likelihood of continued bleeding, and permits therapeutic intervention. Multiple reports have attested to the safety of upper endoscopy in children [7,8,57-59]. The techniques involved in performing an endoscopy and therapeutic intervention are similar in children and adults and include thermal coagulation or clips for bleeding ulcers and ligation (banding) or sclerotherapy for variceal bleeding. (See "Overview of the treatment of bleeding peptic ulcers" and "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

However, there are several considerations pertinent to children:

- Children often require deep sedation or general anesthesia for upper endoscopy. For patients with active or severe bleeding, or if endoscopic therapy is anticipated, general anesthesia with endotracheal intubation is appropriate. This approach optimizes the examination and minimizes the risk of aspiration of the blood. Moreover, in some series, use of general anesthesia, administered by a dedicated anesthesiologist, was associated with a lower rate of complications than use of deep sedation [60,61]. Children undergoing endoscopy for a GI bleed should be carefully monitored during the procedure; sedation in these cases should be guided by a clinician experienced in anesthesiology or intensive care.

- Smaller-caliber endoscopes limit the size of catheters that can be passed through the working channel.

- Band ligation of esophageal varices is often possible, but use of this technique in small children is limited by the small diameter of the esophagus [62-65]. In small children, an overtube should probably not be used [12]. (See "Endoscopic variceal ligation".)

Surgery or angiography is reserved for the uncommon patients in whom endoscopy fails to control bleeding or in whom an anatomic abnormality exists that requires surgery, and if the patient cannot be fully stabilized despite resuscitative measures.

SOCIETY GUIDELINE LINKS

— Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Gastrointestinal bleeding in children" and "Society guideline links: Hemophilia" and "Society guideline links: von Willebrand disease" and "Society guideline links: Acquired bleeding disorders" and "Society guideline links: Rare inherited bleeding disorders" and "Society guideline links: Peptic ulcer disease".)

SUMMARY AND RECOMMENDATIONS

- Upper gastrointestinal (UGI) bleeding can present with hematemesis (vomiting of blood or coffee ground-like material) and/or melena (black, tarry stools). The appearance of the stool is not a reliable indicator of the source of the bleeding. Although melena suggests UGI bleeding, it may also occur in patients with a proximal lower gastrointestinal (LGI) source. Conversely, patients with brisk UGI bleeding and rapid intestinal transit time may present with hematochezia, particularly if they are infants or toddlers. (See 'Clinical presentation' above.)

- The initial evaluation of the patient with UGI bleeding involves an assessment of hemodynamic stability and resuscitation, if necessary. (See 'Initial assessment and resuscitation' above.)

- The most common causes of UGI bleeding in children vary depending upon age and the geographic setting. In Western countries, the most common causes are Mallory-Weiss tears, gastric and duodenal ulcers, esophagitis, gastritis, and varices (table 1). Very rapid UGI bleeding is characteristic of variceal hemorrhage and may be the presenting symptom of portal hypertension. (See 'Etiology' above.)

- The pattern of vomiting, associated symptoms, and underlying diseases help to identify the likely cause of the bleeding, as summarized in the table (table 3). Foreign body ingestion should be considered in a patient with a history of a choking episode, even if transient. Pill esophagitis is suggested by odynophagia (pain while swallowing) and use of certain medications including tetracyclines. Important clues on the physical examination include bruises, petechiae, or mucosal bleeding, suggesting a bleeding disorder or trauma, and splenomegaly, suggesting portal hypertension (table 4).

- Swallowed blood from the nasopharynx may also present with hematemesis or melena and may be difficult to distinguish from UGI bleeding. The clinician should evaluate this possibility by inquiring about a history of recent epistaxis and inspecting the nose and oropharynx carefully. (See 'Differential diagnosis' above and 'Physical examination' above.)

- Nasogastric or orogastric lavage may be performed in patients with clinically significant UGI bleeding to confirm the location and to remove fresh blood or particulate matter from the stomach to facilitate endoscopy. Lavage using ice water is **not** recommended, and frequent lavage can lead to electrolyte imbalances. (See 'Nasogastric tube' above.)

- We suggest acid suppression for children with UGI bleeding (**Grade 2B**). For hemodynamically stable children with mild bleeding, we suggest oral administration of a proton pump inhibitor (PPI) such as omeprazole or esomeprazole. For hemodynamically

unstable children or those with large-volume bleeding, we suggest an intravenous (IV) preparation of a PPI (esomeprazole or pantoprazole) or histamine 2 receptor antagonist (eg, cimetidine or ranitidine) (table 5).

- Upper endoscopy should be performed in patients with acute severe hemorrhage, or low-grade persistent or recurrent hemorrhage. Endoscopy usually permits identification of the bleeding source, allows for risk stratification regarding the likelihood of continued bleeding, and in some cases, permits therapeutic intervention. (See 'Endoscopy' above.)

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