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Vomiting in Children: Reassurance, Red Flag, or Referral?

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Author Disclosure
Drs Chandran and
Chitkara have
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Objectives After completing this article, readers should be able to:

1. Discuss the most common causes of vomiting in children of different age groups.
2. Understand the physiology behind the process of vomiting.
3. Recognize common causes of vomiting based on the pattern and nature of emesis.
4. Be familiar with the basic diagnostic evaluation and treatment strategies for different causes of vomiting.

Case Study

A 1-month-old boy who has had postprandial vomiting for 1 week is admitted from the emergency department. He was born at term with no complications and had regained his birthweight by the second week after birth, feeding on a milk protein formula. He has been vomiting curdled milk intermittently for the past week, and on the day of admission was noted by his pediatrician to have lost 4 oz in weight since his last check-up.

On physical examination, the infant is slightly lethargic and has a sunken fontanelle. The rest of his physical examination findings, including evaluation of his abdomen, are normal. Abdominal ultrasonography shows normal width and length of the pylorus. However, no food movement past the pylorus is observed. An echogenic density in the prepyloric area is noted. An upper gastrointestinal (GI) radiographic series and endoscopy reveal an antral web, which is excised surgically.

Physiology of Vomiting

Vomiting involves the forceful expulsion of the contents of the stomach and is a highly coordinated, reflexive process. It is a feature of many acute and chronic disorders, including those causing increased intracranial pressure, metabolic diseases, and anatomic and mucosal GI abnormalities. Descent of the diaphragm and constriction of the abdominal musculature on relaxation of the gastric cardia force gastric contents back up the esophagus. The process is coordinated by the “vomiting center” in the central nervous system. The vomiting center receives sensory input from the vestibular nucleus (cranial nerve VIII), the GI tract via vagal afferents (cranial nerve X), and the bloodstream via the area postrema, also known as the chemoreceptor (or chemoreceptive) trigger zone. The stereotypic behaviors associated with emesis are a result of output from the vomiting center through vagal, phrenic, and sympathetic nerves.

Types of Vomiting

Vomiting can be classified according to its nature and cause as well as by the character of the vomitus. The nature of the vomiting may be projectile or nonprojectile. Projectile vomiting refers to forceful vomiting and may indicate increased intracranial pressure, especially if it occurs early in the morning. Projectile vomiting also is a classic feature of pyloric stenosis. Nonprojectile vomiting is seen more commonly in gastroesophageal reflux. These somewhat arbitrary descriptions are not definitive in establishing a diagnosis.

Emesis often is classified based on its quality. The vomitus may be bilious, bloody, or nonbloody and nonbilious. Emesis originating from the stomach usually is characterized as being clear or yellow and often contains remnants of previously ingested food. Emesis that

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is dark green is referred to as bilious because it indicates the presence of bile. Bilious vomiting frequently is pathologic because it may be a sign of an underlying abdominal problem such as intestinal obstruction beyond the duodenal ampulla of Vater, where the common bile duct empties. The presence of blood in the emesis, also known as hematemesis, indicates acute bleeding from the upper portion of the GI tract, as can occur with gastritis, Mallory-Weiss tears, or peptic ulcer disease. Coffee ground-like material often is representative of an old GI hemorrhage because blood darkens to a black or dark-brown color when exposed to the acidity of the gastric secretions. The more massive or proximal the bleeding, the more likely it is to be bright red.

Differential Diagnosis

A variety of organic and nonorganic disorders can be associated with vomiting. Organic causes are those related to specific medical conditions. The primary care practitioner needs to remember that vomiting does not localize the problem to the GI system in young infants but can be a nonspecific manifestation of an underlying systemic illness such as a urinary tract infection, sepsis, or an inborn error of metabolism. Nonorganic causes are much more difficult to identify and often are viewed as diagnoses of exclusion. Examples of nonorganic causes of vomiting are psychogenic vomiting, cyclic vomiting syndrome, abdominal migraine, and bulimia. Table 1 lists the differential diagnosis of vomiting based on organ systems. However, from a clinical perspective, it often is useful to consider causes from an age-related perspective.

Vomiting in Infancy

Table 2 details the age-related differential diagnosis of vomiting in infants. Vomiting in the first few days after birth may be a sign of serious pathology. Bilious emesis is suggestive of congenital obstructive GI malformations, such as duodenal/jejunal atresias, malrotation with midgut volvulus, meconium ileus or plugs, and Hirschsprung disease. Published reports of neonates evaluated in neonatal intensive care units with a principal diagnosis of bilious emesis revealed that 38% to 69% had an intestinal obstruction. (1) Nonsurgical causes of bilious emesis include necrotizing enterocolitis and gastroesophageal reflux (GER).

When caring for a neonate who has persistent bilious vomiting, the clinician should place a nasogastric or orogastric catheter to decompress the stomach and prevent any additional vomiting or aspiration before initiating any diagnostic or therapeutic maneuvers. Plain radiographs of the abdomen can demonstrate dilated bowel

loops and air-fluid levels, which strongly suggest bowel obstruction. Contrast imaging studies are more specific and can help pinpoint a precise diagnosis. Surgical and neonatal consultations should be obtained urgently when the diagnosis of bowel obstruction is considered.

Intestinal Atresias

Intestinal atresias are surgical emergencies and typically present within a few hours after birth. Duodenal atresia is a congenital obstruction of the second portion of the duodenum that occurs in 1 per 5,000 to 10,000 live births and is associated with trisomy 21 in approximately 25% of cases. It is believed to be due to a failure of recanalization of the bowel during early gestation. Infants present with clinical features of failure to tolerate feedings and bilious emesis shortly after birth. Due to the proximal nature of the obstruction, abdominal distention usually is not present. Plain abdominal radiographs may show a “double bubble” sign, which represents air in the stomach and proximal duodenum (Fig. 1).

More distal obstructions, such as jejunoileal atresias, typically present with bilious vomiting along with abdominal distention within the first 24 hours after birth. The cause of these atresias is believed to be a mesenteric vascular accident at some point during the course of gestation. The frequency of their occurrence is approximately 1 per 3,000 live births. Anatomically, jejunoileal atresias can be classified into four types: membranous, interrupted, apple-peel, and multiple. Abdominal radiography may show dilated loops of small bowel with air-fluid levels (Fig. 2). Urgent surgical correction is necessary for all types of intestinal atresias.

Malrotation With Midgut Volvulus

Understanding malrotation requires a review of the organogenesis of the gut. During the third week of fetal development, the primitive gut is divided into three regions: the foregut, midgut, and hindgut, based on vascular supply. The first stage of intestinal development involves rapid growth of the midgut outside the abdominal cavity through a herniation of the umbilical orifice. During the second stage, the midgut returns to the abdominal cavity, rotating 180 degrees and pushing the hindgut to the left. The last stage of intestinal development involves the retroperitonealization of portions of the right colon, left colon, duodenum, and intestinal mesentery, helping them serve as anchors for the bowel. Disruption of this process during the second or third stage can result in an aberrant return or anchoring of the midgut within the abdominal cavity.

Table 1. Differential Diagnosis of Vomiting by Systems

Gastrointestinal	Neurologic	Endocrine
<ul style="list-style-type: none"> • Esophagus: Stricture, web, ring, atresia, tracheoesophageal fistula, achalasia, foreign body • Stomach: pyloric stenosis, web, duplication, peptic ulcer, gastroesophageal reflux • Intestine: duodenal atresia, malrotation, duplication, intussusception, volvulus, foreign body, bezoar, pseudo-obstruction, necrotizing enterocolitis • Colon: Hirschsprung disease, imperforate anus, foreign body, bezoar • Acute gastroenteritis • <i>Helicobacter pylori</i> infection • Parasitic infections: ascariasis, giardiasis • Appendicitis • Celiac disease • Milk/soy protein allergy • Inflammatory bowel disease • Pancreatitis • Cholecystitis or cholelithiasis • Infectious and noninfectious hepatitis • Peritonitis • Trauma: Duodenal hematoma 	<ul style="list-style-type: none"> • Tumor • Cyst • Hematoma • Cerebral edema • Hydrocephalus • Pseudotumor cerebri • Migraine headache • Abdominal migraine • Seizure • Meningitis <p>Renal</p> <ul style="list-style-type: none"> • Obstructive uropathy: Ureteropelvic junction obstruction, hydronephrosis, nephrolithiasis • Renal insufficiency • Glomerulonephritis • Urinary tract infection • Renal tubular acidosis <p>Metabolic</p> <ul style="list-style-type: none"> • Galactosemia • Hereditary fructosemia • Amino acidopathy • Organic acidopathy • Urea cycle defects • Fatty acid oxidation disorders • Lactic acidosis • Lysosomal storage disorders • Peroxisomal disorders 	<ul style="list-style-type: none"> • Diabetic ketoacidosis • Adrenal insufficiency <p>Respiratory</p> <ul style="list-style-type: none"> • Pneumonia • Sinusitis • Pharyngitis <p>Miscellaneous</p> <ul style="list-style-type: none"> • Sepsis syndromes • Pregnancy • Rumination • Bulimia • Psychogenic • Cyclic vomiting syndrome • Overfeeding • Medications/vitamin/drug toxicity • Superior mesenteric artery syndrome • Child abuse

Although most infants who have intestinal malrotation present within the first week after birth due to the accompanying volvulus, the malrotation itself does not cause any notable symptoms and may be undetected for years. Bowel strangulation can occur at any age and any time because affected patients are at increased risk of volvulus due to a lack of proper mesenteric anchoring to the retroperitoneum. The midgut twists in a clockwise direction around the superior mesenteric vessels, leading to obstruction of vascular supply to most of the small and large intestine. Once bowel ischemia occurs, metabolic acidosis, unstable hemodynamics, and intestinal necrosis with perforation may ensue if the condition is not diagnosed and rapidly corrected surgically. A spiral configuration of the jejunum or demonstration of failure of contrast to pass beyond the second portion of the duodenum on upper GI radiographic series is diagnostic (Fig. 3). Abdominal ultrasonography also may reveal a

malposition of the superior mesenteric vessels. Timely surgical correction with the Ladd procedure is critical. If bowel ischemia is prolonged, loss of bowel and resultant short gut syndrome may occur.

Vomiting in Infancy Beyond the Neonatal Period

The differential diagnosis of vomiting in infants beyond the neonatal period is more extensive. Common causes are acute gastroenteritis, GER, and nutrient intolerances such as milk or soy protein allergies. Metabolic diseases and inborn errors of metabolism also should be considered for infants who have persistent progressive vomiting. Acquired or milder intestinal obstructive lesions, such as infantile hypertrophic pyloric stenosis (IHPS), also are possible and should be ruled out when clinically indicated.

Common entities such as GER, dietary protein intolerances

Table 2. Age-related Differential Diagnosis of Vomiting in Children Younger Than 12 Months of Age

Age	Common Causes	Type of Vomiting	Comment/Associated Features
Newborn	Intestinal atresia/webs	Bilious, depending on level of lesion	May occur at level of esophagus, duodenum, jejunum
	Meconium ileus	Bilious	Strongly associated with cystic fibrosis; genetic testing suggested
	Hirschsprung disease	Bilious or nonbilious	History of non-passage of stools in nursery suggestive; suction rectal biopsy may demonstrate lack of intestinal ganglion cells
	Necrotizing enterocolitis	Bilious or nonbilious	Plain films of abdomen may reveal intestinal pneumatosis
0 to 3 months	Inborn errors of metabolism	Bilious or nonbilious	May have acidosis or hypoglycemia
	Pyloric stenosis	Nonbilious	Hypochloremic metabolic alkalosis
	Malrotation with midgut volvulus	Bilious	Abdominal distention may be present; plain radiographs may show air-fluid levels and paucity of distal bowel gas; emergent surgical consultation necessary
	Inborn errors of metabolism	Bilious or nonbilious	Newborn metabolic screen may be abnormal; acidosis or hypoglycemia may be present
	Milk/soy protein allergy	Bilious or nonbilious; may have gross or occult blood	History of extreme fussiness may be present; fecal occult blood testing of stools may be positive
	Gastroesophageal reflux	Nonbilious; may have gross or occult blood	Emesis usually within 30 minutes of feeding; symptoms worse in supine flat position
3 to 12 months	Child abuse	Nonbilious	Anterior fontanelle fullness may be present; central nervous system (CNS) imaging studies may reveal acute or subacute bleeding
	Gastroenteritis	Nonbilious initially; may progress to bilious	Stool studies may help establish offending pathogen
	Intussusception	Bilious	Abdomen distention may be present; plain radiographs may show air-fluid levels and paucity of distal bowel gas; stools may be grossly bloody with "currant jelly" appearance; emergent surgical consultation indicated; may be reduced by contrast enema
	Child abuse	Nonbilious	Anterior fontanelle fullness may be present; CNS imaging studies may reveal acute or subacute bleeding
3 to 12 months	Intracranial mass lesion	Nonbilious	Anterior fontanelle fullness may be present; CNS imaging studies diagnostic



Figure 1. "Double bubble" sign on plain radiograph, which represents air in the stomach and proximal duodenum and indicates duodenal atresia.

erance, and IHPS are discussed in greater detail in this section. Intussusception is another important cause of vomiting in the young infant that is discussed in the next section because it also may present beyond infancy. The management of acute gastroenteritis is discussed in the section on management.

Gastroesophageal Reflux

GER is the most common cause of recurrent nonbilious emesis in infancy. It involves the retrograde movement of gastric contents into the esophagus as a result of an abnormally functioning lower esophageal sphincter (LES). Under normal circumstances, the LES relaxes after swallowing to allow passage of ingested food into the stomach. Patients who have GER have transient

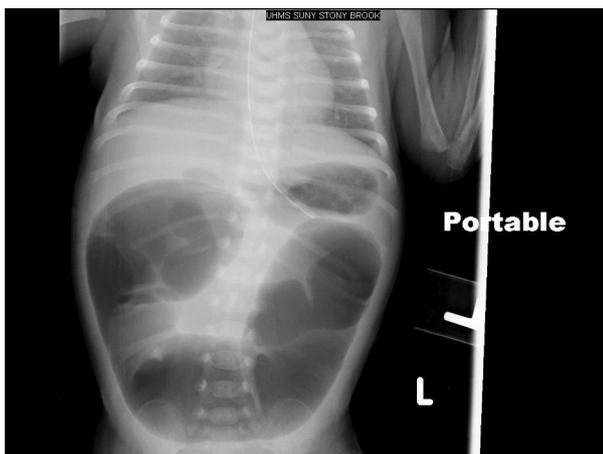


Figure 2. Dilated loops of small bowel with air-fluid levels, indicative of jejunoileal atresia.

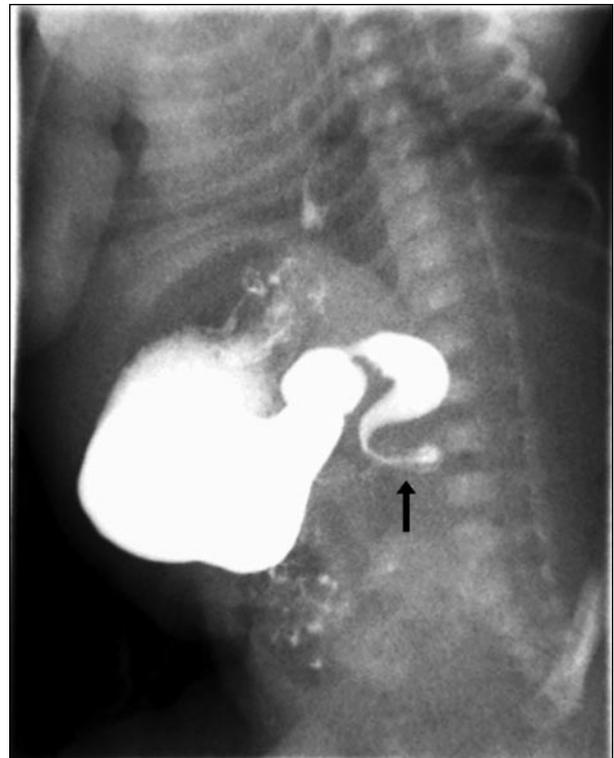


Figure 3. Failure of contrast to pass beyond the second portion of the duodenum, which is characteristic of malrotation with midgut volvulus.

relaxations of the LES that are not precipitated by a swallow, allowing gastric contents to move freely back into the esophagus from an area of higher to lower pressure. In young infants, such relaxation often results from developmental immaturity of the LES, which may improve over time. Infants who have GER present with recurrent postprandial regurgitation of ingested food or milk, most often within 30 minutes of a feeding. Affected children may appear irritable during or after feedings, and stereotypic opisthotonic movements with extension and stiffening of arms and legs and extension of the head (Sandifer syndrome) occasionally may be observed. Infants who have severe GER can have recurrent microaspiration into their lungs, resulting in chronic wheezing, respiratory symptoms, and even failure to thrive.

Infants who have the classic history of recurrent emesis but who are thriving and have normal physical examination findings do not need specific treatment. Thickening the formula or human milk by adding cereal may help reduce vomiting in such infants, but elevating the head in the supine position has no proven beneficial effect. (2) Infants who are irritable during feedings and those who have respiratory or growth problems may

need pharmacologic intervention. Acid blockade with histamine₂ receptor antagonists or proton-pump inhibitors may help lessen the burning sensation caused by the gastric refluxant. Prokinetic agents such as metoclopramide and erythromycin may help decrease the physical process of GER by targeting the LES.

A Cochrane meta-analysis reviewing seven randomized control trials showed that metoclopramide was superior to placebo in reducing daily symptoms of GER. (2) However, its use must be weighed against the potential adverse effects of extrapyramidal symptoms, headache, and drowsiness. Recent studies have suggested that baclofen, a GABA receptor agonist, may lessen the number of transient LES relaxations via vagal-mediated mechanisms and, thus, improve the pathophysiologic process associated with GER. (3) Additional investigation into this agent's overall efficacy for the treatment of GER is necessary.

Dietary Protein Intolerance

Dietary protein intolerance is a non-immunoglobulin E-mediated type of food hypersensitivity that typically presents in infants in the first postnatal year, shortly after exposure to the offending allergen. Commonly implicated proteins include cow milk protein, soy protein, and egg protein. Among the clinical symptoms are irritability, feeding intolerance, recurrent vomiting and diarrhea, and in severe cases, failure to thrive. Occasionally, patients may present with Heiner syndrome, manifesting as pulmonary hemosiderosis (due to recurrent microhemorrhages into the lungs), iron deficiency anemia, and failure to thrive. Examination of stools in patients who have protein intolerance may reveal occult blood, with polymorphonuclear cells, lymphocytes, and eosinophils. Stool-reducing substances may be positive due to carbohydrate malabsorption. Intestinal biopsies may reveal flattened villi and colitis with infiltration of lymphocytes, eosinophils, and mast cells.

Treatment of dietary protein intolerance involves removal of the allergen from the diet. In the case of cow milk protein allergy, 80% of patients respond to hydrolyzed casein formula; the remaining 20% require L-amino acid-based formulas or intravenous nutrition. (4) Once elimination has occurred, symptoms usually resolve in 3 to 10 days. The dietary protein intolerance typically subsides by 18 to 24 months of age. (4)

Infantile Hypertrophic Pyloric Stenosis

Infants who have pyloric stenosis typically present to medical attention with persistent projectile nonbilious emesis between 2 and 6 weeks of age. Males, especially

those who are firstborn, are affected approximately four times as often as females. The incidence is approximately 3 per 1,000 live births. The exact cause of pyloric stenosis remains unclear. The relaxation mechanism of the pyloric smooth muscle depends on nonadrenergic noncholinergic inhibitory innervation, mediated by vasoactive intestinal peptide and nitric oxide (NO). Deficiencies in neuropeptidergic innervation and NO have been implicated in cases of pyloric stenosis, but neither has been substantiated as etiologic. Very early exposure to erythromycin (within the first 2 weeks after birth) also has been associated with an eightfold increased risk of pyloric stenosis. (5) It is hypothesized that erythromycin interacts with intestinal motilin receptors, causing strong gastric and pyloric contractions and subsequent pyloric muscle hypertrophy.

Pyloric stenosis usually is diagnosed by a typical history and physical findings. Inspection of the abdomen shortly after an infant feeding may reveal a peristaltic wave because the stomach muscles contract in an attempt to pass ingested milk past the pylorus. A palpable "olive" in the mid-epigastric region represents the hypertrophic pyloric muscle and strongly supports the diagnosis of pyloric stenosis. Repeated episodes of vomiting of the gastric contents due to pyloric stenosis may result in characteristic electrolyte abnormalities, although serum electrolyte values may be normal if the patient is diagnosed in the early stages.

The classic electrolyte abnormality is a hypochloremic hypokalemic metabolic alkalosis. Normal acid production in the stomach is accompanied by the release of bicarbonate ions into the blood as a result of the action of carbonic anhydrase. Because of the loss of the hydrogen ions, this bicarbonate is unbuffered, resulting in an ensuing metabolic alkalosis. Under normal conditions, the excess bicarbonate is excreted in the urine. However, affected infants also lose significant amounts of fluid in addition to the electrolytes. The subsequent volume contraction triggers a renal response of enhanced proximal tubular reabsorption of bicarbonate and activation of the renin-angiotensin-aldosterone mechanisms. In addition, the lack of chloride ion in the proximal tubule results in increased local production and reabsorption of bicarbonate, thus worsening the existing metabolic alkalosis. Under the influence of high concentrations of aldosterone, the distal tubule excretes large amounts of potassium and hydrogen ions in exchange of sodium. Lack of hydrogen ions results in enhanced excretion of potassium, leading to significant hypokalemia.

When the diagnosis of pyloric stenosis is being considered, ultrasonography of the pyloric muscle can con-

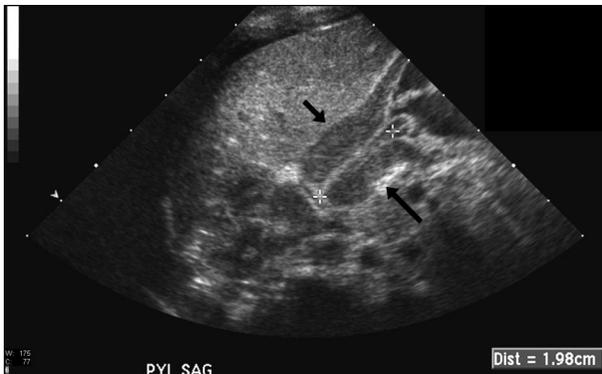


Figure 4. Thickened and lengthened pyloric muscle that is characteristic of pyloric stenosis.

firm the clinical suspicion, with sensitivity rates ranging from 85% to 100%. (6) Pyloric muscle thickness of 4 mm or more and muscle length of 14 mm or more are diagnostic of pyloric stenosis (Fig. 4). If ultrasonographic examination findings are normal, an upper GI radiographic series can be performed. The radiographic series has a slightly higher sensitivity for pyloric stenosis (89% to 100%) and can aid in the diagnosis of other causes of progressive emesis in this age group, such as antral web and other structural abnormalities. Surgical pyloromyotomy is the definitive treatment of pyloric stenosis and is being performed laparoscopically at many centers.

Vomiting in Older Children

Vomiting occurs most commonly in older children in the setting of an acute gastroenteritis accompanied by fever and diarrhea. Vomiting also can be a nonspecific manifestation of a systemic illness, although much less commonly than in the young infant. Both viral and bacterial meningitis can present with vomiting, usually accompanied by complaints of headache, fever, and neck stiffness. Elevation of intracranial pressure from entities such as a brain tumor or an intracranial hemorrhage also may present with a chief complaint of vomiting in association with a severe, progressive headache. Vomiting in such patients often occurs shortly after waking in the morning because of a gradual rise in intracranial pressure as the child sleeps in the supine position. Inadvertent toxic ingestions also should be considered, especially in toddlers.

Intussusception

Acquired bowel obstructions such as intussusception may present in the older infant and young child, with the peak incidence occurring between 3 months and 3 years

of age. Intussusception is the telescoping of one portion of the bowel into its distal segment. Most commonly, the terminal ileum invaginates into the cecum, often as a result of lymphatic hypertrophy in the Peyer patches from a recent viral infection. A history of intermittent episodes of severe and crampy abdominal pain with bilious emesis is classic. Parents often report that their child is lethargic in between episodes of pain and may describe blood-tinged, “currant jelly” stools. Physical examination may reveal intestinal obstruction with a sausage-shape mass palpable in the right lower quadrant. Rapid consultation with a pediatric surgeon is warranted. Contrast or air enemas can be diagnostic, with the contrast outlining the lead portion of the intussusception, giving the typical “coiled spring” appearance (Fig. 5). In addition, the hydrostatic pressure from the contrast enema may reduce telescoping of the intestine. Surgical reduction of the intussusception is indicated when the contrast enema is not successful.

Cyclic Vomiting Syndrome

Cyclic vomiting syndrome (CVS) is characterized by stereotypic recurrent episodes of nausea and vomiting without an identifiable organic cause. It is an idiopathic disorder that usually begins in early childhood; relatively little is known about its pathogenesis or cause. The diagnosis is based on several characteristic features: 1) three or more episodes of recurrent vomiting, 2) intervals of normal health between episodes, 3) episodes that are stereotypic with regard to symptom onset and duration, and 4) lack of laboratory or radiographic evidence to support an alternative diagnosis. Vomiting episodes are of rapid onset and persist for hours to days, separated by symptom-free intervals that can range from weeks to years. Treatment is supportive, focused on fluid management in cases where dehydration and electrolyte

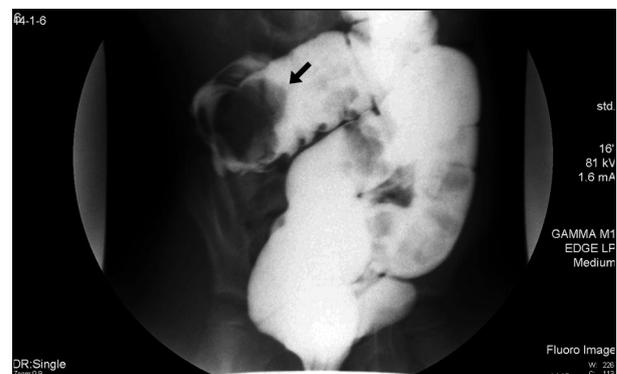


Figure 5. Contrast outlining the lead portion of the intussusception, giving the typical “coiled spring” appearance.

imbalance occur. Amitriptyline and propranolol have been described as effective for prophylactic therapy (antiemetics may be of benefit during an acute episode).

Abdominal Migraine

Abdominal migraines involve episodic attacks of epigastric or periumbilical abdominal pain and are believed to share pathophysiologic mechanisms with CVS. Abdominal migraines are more common in females than in males, with a ratio of 3:2, and the onset is primarily between 7 and 12 years. A family history of migraine headaches may be present. Episodes of abdominal pain are acute in onset and last for 1 hour or more. The pain is so intense that it interferes with the performance of normal activities and is associated with anorexia, nausea, vomiting, headache, photophobia, and pallor. Much like those who have CVS, patients who have abdominal migraines report intervals of completely normal health between the episodes of pain. Diagnostic evaluation looking for alternative organic conditions yields negative results. The diagnosis of abdominal migraine is supported by a favorable response to medications used for treatment of migraine headaches. Patients should be advised about trigger avoidance, specifically caffeine-containing foods, altered sleep patterns, prolonged fasting, emotional stress, and exposure to flickering lights.

Rumination

Rumination is the repeated and painless regurgitation of ingested food into the mouth beginning soon after food intake. The food is re-chewed and swallowed or spit out. Symptoms do not occur during sleep and do not respond to the standard treatment of GER. To qualify for the diagnosis, symptoms must be present for longer than 8 weeks. Rumination is not associated with retching and often is viewed as a behavioral entity, typically seen in mentally retarded children, neonates during prolonged hospitalization, and children and infants who have GER. Rumination also has been described in cases of child neglect and in older children and adolescents who have bulimia or are depressed. Most commonly, rumination is seen among female adolescents or male infants. One third of affected individuals have underlying psychological disturbances. The management of rumination involves a multidisciplinary approach, with a primary focus on behavioral therapy and biofeedback. Occasionally, tricyclic antidepressants and nutritional support may be necessary.

Superior Mesenteric Artery Syndrome

Superior mesenteric artery (SMA) syndrome, otherwise known as Wilkie syndrome or cast syndrome, is a functional upper intestinal obstructive condition. Normally, the SMA forms a 45-degree angle, with the abdominal aorta at its origin and the third portion of the duodenum crossing between the two structures. When the angle between the SMA and the aorta is narrowed to less than 25 degrees, the duodenum may become entrapped and compressed. This condition most commonly is described in patients who have experienced rapid weight loss, immobilization in a body cast, or surgical correction of spinal deformities.

SMA syndrome typically presents with epigastric abdominal pain, early satiety, nausea, and bilious vomiting. Patients experience worsening pain in the supine position, which may be relieved in the prone or knee-chest position. Diagnosis usually is confirmed by upper GI radiographic series (Fig. 6) or computed tomography scan (Fig. 7) with failure of contrast to pass beyond the third portion of the duodenum. Conservative initial management of SMA syndrome focuses on gastric decompression, followed by the establishment of adequate nutrition and proper positioning after meals. Placement of an enteral feeding tube distal to the obstruction or parenteral nutrition may be needed in severe cases. Surgical correction with a duodenojejunostomy is a last resort.

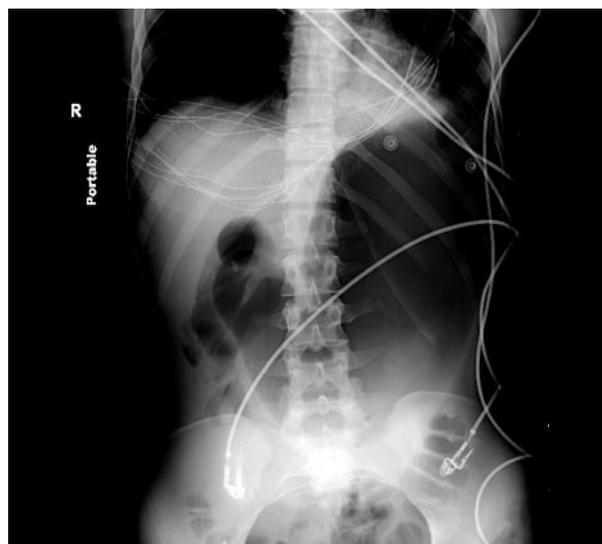


Figure 6. Severely dilated stomach and proximal duodenum indicative of high obstruction consistent with superior mesenteric artery syndrome.



Figure 7. Computed tomography scan of the abdomen reveals dilated stomach and duodenum with air–fluid levels. The duodenum tapers abruptly (arrow) as it crosses the midline between the aorta and the superior mesenteric artery.

General Principles in the Management of Vomiting

Therapy to alleviate vomiting should be directed at the specific cause, when possible. Gastrointestinal obstructions should be corrected, as deemed appropriate by the pediatric surgery team. Management of nonsurgical causes of vomiting include steps to correct fluid and electrolyte imbalances that result from prolonged or excessive vomiting and to identify and treat the underlying disorder causing the symptom. The 2003 Centers for Disease Control and Prevention practice guidelines for the management of acute gastroenteritis in children, endorsed by the American Academy of Pediatrics, recommend oral rehydration therapy (ORT) in cases of mild-to-moderate dehydration from acute gastroenteritis. (7) Using an appropriate glucose-electrolyte solution, 50 to 100 mL/kg of fluid should be administered to the child over the course of 4 hours, along with replacement of continuing losses from stool and emesis. This is most effective when the ORT is administered in small, 5-mL increments every 1 to 2 minutes. In cases of severe dehydration, ileus, or persistent vomiting despite adequate attempts at ORT, parenteral fluids must be administered.

Although the previously cited guidelines do not recommend the routine use of antiemetic drugs in the management of patients who have acute gastroenteritis, unique situations may warrant their use. If the cause of the vomiting is unclear, antiemetics are contraindicated. Phenothiazines such as prochlorperazine, promethazine, and chlorpromazine are antiemetics that act as D2-

receptor antagonists at the chemoreceptor trigger zone. Such drugs rarely are used in pediatric patients because of their extrapyramidal and sedative adverse effects. Antihistamines such as diphenhydramine, hydroxyzine, and dimenhydrinate also may help alleviate nausea and vomiting but have a sedative effect that makes clinical reevaluation difficult. A newer class of antiemetics is the 5HT₃-receptor antagonists, ondansetron and granisetron. The 5HT₃ blockade occurs both at the enteric level and at the chemoreceptor trigger zone. These drugs, unlike the phenothiazines and antihistamines, do not have central nervous system adverse effects, making them more attractive options. The 5HT₃-receptor antagonists have been approved for the management of chemotherapy-induced nausea and vomiting and for pregnancy-associated and postoperative vomiting in adults. For children, however, there is no substantive scientific evidence supporting their efficacy in treating acute gastroenteritis. Therefore, these agents have not been endorsed officially for routine use.

Conclusion

Vomiting is a nonspecific symptom that may accompany a wide variety of GI and extraintestinal disorders. Serious extraintestinal causes of vomiting include brain tumor and meningitis; congenital or acquired intestinal obstructive syndromes are the most serious intestinal causes. Associated fluid and electrolyte imbalances always must be considered when assessing a child who has a history of vomiting. Conditions such as mild GER may only necessitate reassurance, but symptoms of bilious vomiting should prompt immediate referral to a pediatric surgeon. Results of the history and physical examination, keeping in mind the nature of the vomiting and age of the child, may help the clinician determine the likely cause and the need for emergent treatment.

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PIR Quiz

Quiz also available online at www.pedsinreview.org.

Match the clinical finding with the most likely condition. Each answer may be used once, more than once, or not at all.

1. Flattened villi and colitis on biopsy.
2. Uncoordinated esophageal relaxation.
3. Hypochloremic hypokalemic metabolic alkalosis.
4. Recurrent microaspiration.
5. “Coiled spring” appearance on radiography.

- A. Abdominal migraine.
- B. Gastroesophageal reflux.
- C. Intussusception.
- D. Protein intolerance.
- E. Pyloric stenosis.

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