

Pediatric Gastroenterology-Chronic Vomiting in Infants from 0-12 Months of Age

Joan S. Di Palma, MD

Educational Goals

1. Identify differences in approach between pediatric and adult gastroenterology
2. Develop a differential diagnosis for vomiting in infants
3. Initiate an evaluation for a vomiting infant

The approach to the pediatric patient with a gastrointestinal problem differs to the approach to an adult patient. First, the infant is not speaking for him/her self. The history is always provided by another individual (usually a parent). Therefore, objective measurements and observations become very important. Second, infants are more likely to present with congenital anomalies, so these entities need to be elevated nearer to the top of the differential diagnosis. Third, the incidence of gastrointestinal cancer (esophageal, gastric, colon) is rare. These entities need to be moved to the bottom of the differential. Forth, infections and systemic conditions such as acidosis and other metabolic derangements are more likely to present as gastrointestinal symptoms, such as vomiting. Fifth, young infants are more susceptible to dietary protein intolerance, which can present as gastrointestinal symptoms. Finally invasive procedures in an infant are often more difficult from a technical, physical, and emotional standpoint. Therefore, the use of these procedures is approached judiciously.

The differential diagnosis of chronic vomiting in an infant is broad. Chronicity refers to greater than three weeks. Therefore, an acute gastroenteritis would be a less likely cause of chronic emesis. However, as mentioned above, chronic infections, such as otitis media, urinary tract infections, etc. can present this way. Metabolic diseases, especially ones that result in persistent acidosis, frequently are associated with vomiting. Examples are renal tubular acidosis, Methyl malonic acidemia and propionic acidemia. Increased intracranial pressure can result in emesis. Examples would be hydrocephalus, meningitis, and brain tumors.

From the gastrointestinal standpoint, one of the commonest causes of chronic emesis is **gastroesophageal reflux (GER)**. GER is the flow of gastric contents back up into the esophagus. The etiology is not fully defined, but it is thought to be due mostly from inappropriate relaxation of the lower esophageal sphincter. GER is present in up to 80% of normal full term infants. Approximately 20% of these require medical attention for symptoms. Symptoms can consist of emesis (non-bilious, non-projectile), gastrointestinal bleeding, poor growth and irritability. A number of respiratory symptoms have been related to GER, including apparent life threatening events (ALTE), chronic cough, recurrent pneumonias, and wheezing. The diagnosis of GER can be made in a variety of ways. Many infants can be diagnosed by history alone. Other modalities used in the diagnosis of GER are the upper GI series (UGI), nuclear medicine scintiscan or "milk scan", 24-hour pH probe and endoscopy. Treatment of GER spans from "conservative" therapy (positioning and feeding alterations), to medications, and finally to surgery. The natural history of reflux in healthy full-term infants is that it resolves in many cases by two years of age.

The entity of "**allergic gastroenteritis**", including eosinophilic esophagitis and eosinophilic gastritis has received a great deal of attention in the past 5-10 years. This entity has been associated with chronic vomiting, failure to thrive, and feeding disorders. Cow's milk and soymilk protein are usually the implicated proteins, as they comprise most of the protein available to a young infant. Most of the infants who present with allergic gastroenteritis come from "atopic" families-other family members have a history of food allergies, eczema, or asthma. Diagnosis of allergic gastroenteritis can be made clinically, or with the aid of allergy testing and endoscopy. Treatment is usually dietary, although medications are sometimes beneficial.

As mentioned above, congenital anomalies of the gastrointestinal tract need to be excluded, particularly in the young infant (<6 months). The more proximal in the GI tract the anomaly is, the earlier the defect presents.

Esophageal atresia occurs in ~1/3000-5000 births. It is usually associated with a tracheo-esophageal fistula. The most common defect is a blind proximal esophageal pouch with a distal tracheo-esophageal fistula. These defects occur as a failure of the foregut to separate completely into respiratory and esophageal components during the first few weeks of fetal development. 50% of infants present with polyhydramnios. Most other infants present in the first few hours of life with emesis of swallowed secretions and aspiration. However, some variants present later in infancy with vomiting and respiratory symptoms. Diagnosis of esophageal atresia is made clinically and via radiologic studies. Treatment is almost always surgical. Infants with esophageal atresia have a higher incidence of other congenital anomalies.

Pyloric stenosis is a narrowing of the pyloric outlet secondary to idiopathic hypertrophy of the pyloric muscle. It occurs in ~1/1000 births. The male:female ratio is 5:1. Infants usually present with vomiting at 3-6 weeks of life. The emesis is non-bilious and can be "projectile". If the vomiting is prolonged, the infants can develop poor weight gain and dehydration (hypochloremic alkalosis). Diagnosis can be made clinically by history and physical exam. The hypertrophied pylorus can sometimes be palpated on abdominal exam ("olive"). Pyloric sonograms and UGI series can also be helpful in making the diagnosis. Treatment is almost always surgical.

Duodenal atresia, duodenal stenosis and annular pancreas occur in ~1/10,000 infants. 50% of the infants are premature and 20-30% have Down's syndrome or another chromosomal anomaly. Infants present with emesis within the first 2 days of life. If the stenosis is above the ampulla of Vater, the emesis will be non-bilious (~30%). Otherwise, the emesis is bilious. These infants will understandably tolerate feedings poorly. Many of them will have abdominal distension. Diagnosis is made via an abdominal flat plate demonstrating the "double bubble" sign—a dilated stomach and dilated duodenal bulb. An UGI series is sometimes necessary as well. Treatment is almost always surgical.

Anomalies of intestinal rotation and fixation, sometimes referred to as "**malrotation**" occur in ~0.2% of the population. They stem from a defect in intestinal lengthening and rotation during the 8th week of gestation. There are many forms of malrotation. One of the most common is "non-rotation"—the entire small intestine is on the right and the colon is on the left. Anomalies of intestinal rotation and fixation result in a narrowed base of attachment for the small bowel mesentery. This predisposes the bowel to twist (volvulus) resulting in bilious emesis and pain. **Volvulus** can result in bowel ischemia and even bowel necrosis. The results can be dire. Infants with malrotation can present with vomiting and distention due to volvulus and/or obstruction from bowel compression due to misplaced connective tissue (Ladd's bands). Presentation is usually in the first few months of life. Diagnosis is usually by radiological studies (UGI series and/or barium enema). Treatment is almost always surgical.

Intestinal atresias suggest a complete obstruction of the lumen of the bowel. Stenosis is an incomplete obstruction. Bowel atresias are more common in the jejunum and ileum and occur in ~1/2700 births. Atresias are idiopathic, but they may stem from ischemic events to the bowel in utero. Atresias and bowel stenosis can present as bilious emesis, growth failure, and abdominal distension at any time in infancy. At times, a keen clinical suspicion is necessary. Diagnosis is via radiological studies and/or exploratory laparotomy. Treatment is almost always surgical.

From the above differential, one can devise a diagnostic approach to chronic emesis in infants. First, the history is crucial. The nature of the emesis (projectile, bilious, blood content, quantity and frequency) is very important. The infant's feeding pattern is imperative. The presence of a normal bowel pattern speaks against chronic bowel obstruction. Non-bilious, non-bloody, non-projectile emesis in an infant feeding and developing well rarely portends abdominal obstruction. The physical exam is frequently helpful—especially vital signs, growth parameters, and abdominal exam (distension, masses, bowel sounds), and an observation of the stools for occult blood. If abdominal obstruction is

suspected, one of the most helpful initial evaluations is an abdominal obstructive series. A surgical consultation is required to pursue the diagnosis of abdominal obstruction.

If the infant has persistent emesis accompanied by poor feeding, failure to thrive and a normal abdominal exam, other “non GI” entities might be considered. A careful physical exam will reveal signs of infection (otitis) or increased intracranial pressure (increased head circumference and/or bulging fontanelles). In a newborn, blood cultures and a urine culture might be helpful. Metabolic and genetic studies might be pursued. Metabolic tests would include the newborn screen, serum lactate, pyruvate and ammonia, as well as serum electrolytes and liver function tests.

Most chronic emesis in infants can be diagnosed utilizing a careful history and physical exam. Diagnostic tests can provide insight when carefully utilized.

Reference

Wyllie R and Hyams JS, eds. Pediatric Gastrointestinal Disease, Pathophysiology, Diagnosis and Management. W.B. Saunders Company (Philadelphia, PA), 1999:pp14-32,149,207,505.

Appendix: Differential Diagnosis of Chronic Vomiting in Infancy

Infections

Otitis Media
Meningitis
Urinary tract infection

Anatomic Abnormalities

Tracheo-esophageal fistula
Pyloric stenosis
Duodenal atresia/stenosis
Malrotation with volvulus
Inguinal hernia
Intestinal atresia/stenosis

Mucosal Injury

GER/esophagitis
Peptic ulcer/duodenitis
Allergic gastroenteritis

GI Motility Disorders

Hirschsprung's disease
Pseudoobstruction

Visceral GI Disorders

Choledochal cyst

Endocrine/Metabolic Disorders

Adrenal hyperplasia
Organic acidemias
Disorders of fatty acid oxidation
Amino acidemias
Urea cycle defects
Mitochondriopathies

Neurologic Disorders

Hydrocephalus
Subdural hematoma
Intracranial neoplasm

Other

“Overfeeding”
Toxic ingestion
Rumination
Munchausen by proxy