Abdominal Pain in Children

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Abdominal pain and gastrointestinal (GI) symptoms, such as vomiting or diarrhea, are common chief complaints in young children presenting in emergency departments (ED). It is the emergency physician’s role to differentiate between a self-limited process such as viral gastroenteritis or constipation and more life-threatening surgical emergencies. Extra-abdominal conditions such as pneumonia or pharyngitis caused by streptococcal infection also can present with abdominal pain and must be considered (Box 1). Considering the difficulties inherent in the pediatric examination, it is not surprising that the diagnoses of appendicitis, intussusception, or malrotation with volvulus continue to be among the most elusive diagnoses for the emergency physician (EP). This article reviews self-limited and more benign gastrointestinal conditions such as viral gastroenteritis or constipation and emergency surgical conditions that may present such as appendicitis or intussusception.

**General approach to the child who has abdominal pain**

Important information often can be elicited even before speaking to the parents or laying hands on a child. Infants and young toddlers are usually afraid of strangers. Older children may associate a clinic environment or a “man in a white coat” with immunizations and pain. The difficulty of physical examination increases when the physician enters the examination room and the child bursts...
Box 1. Extra-abdominal causes of gastrointestinal distress

Abdominal epilepsy
Abdominal migraine
Black widow spider bite
Hemolytic uremic syndrome
Henoch-Schönlein purpura
Ingestions (eg, iron)
Pharyngitis (especially induced by streptococcal infection)
Pneumonia
Sepsis

into tears. Observing the child’s behavior before any interaction may reveal the reassuring signs of a young child ambulating comfortably around the ED or of an older infant sitting up on a gurney, interested in the surroundings. An older child who walks slowly down a corridor in the ED holding his right lower quadrant similarly has given the examiner a great deal of information. Once the child is approached, use of a nonthreatening manner may pay dividends during the assessment; for example, a position sitting down or kneeling will bring the examiner closer to the child’s eye level and is less intimidating.

If a child is found to be poorly responsive or displays other signs of shock, the ongoing assessment of the abdomen will need to occur simultaneously with the immediate priorities of resuscitation. A patent and secure airway must be ensured. Ventilation should be assisted if necessary and supplemental oxygen delivered. Vascular access should be achieved using the intravenous or intraosseous routes, and fluid boluses of normal saline should be administered as necessary. The child should be placed on a cardiac monitor. Immediate bedside tests should include a blood glucose test and hemoglobin determination. The delivery of intravenous antibiotics should not be delayed if there is a reasonable suspicion of underlying sepsis.

Children and parents are often poor historians. Trying to elicit the chronology of symptoms with questions such as “did the pain start before the vomiting or visa versa?” may be difficult. Parents of young infants may only describe their child as irritable and not realize that the abdomen is the source of pain. Adolescents may be embarrassed to talk about bodily functions or sexual issues, especially with physicians of the opposite sex. It is important also to question adolescents about their medical history separately from their parents because they may be more forthcoming when assured of their privacy.

Attempting to bond with the child or using a toy as a distraction before auscultation or palpation can often improve the reliability of the abdominal examination. Infants may be distracted by a set of car keys. Hand and finger puppets also can be used for this purpose. Allowing the child to remain in a parent’s arms or lap for as long as possible is also helpful. For older children, examining the mother first may show the child that the examination is nothing
to be feared. An older child also can be allowed to place his or her hand on top of
the examiner’s and simultaneously apply pressure and can be questioned about
school or play activities.

Before touching the patient’s abdomen, the examiner should look for any
obvious abnormalities such as distension, masses, or peristaltic waves. If a child
is crying, it should be remembered that the abdomen is relatively soft during
the child’s’ inhalation. This may be the best time to detect masses. To elicit areas of
tenderness or peritoneal signs, a quieter, calm child is helpful. If the examiner
has difficulty, in some cases it may be possible to have the mother gently push on
different areas of the abdomen, while the examiner merely observes the child’s
response. Another technique is to have the mother hold the child over her shoul-
der with the child facing away. The examiner can then stand behind the child
and slip a hand between the mother and child to palpate the abdomen. Peritoneal
signs may also be elicited by having the mother bounce the child up and down on
her lap. Fussiness or crying while this maneuver is performed raises the suspicion
of peritonitis. Older children can be asked simply to jump up and down.

Rectal examinations are not imperative in a child presenting with abdominal
pain. In particular, rectal examinations have not been shown to be helpful in the
diagnosis of appendicitis. Rectal examinations, however, can aid in the diagnosis
of gastrointestinal bleeding, intussusception, rectal abscess, or impaction. If a
rectal examination is necessary, it can be performed by partially introducing a
small finger. Inspection of the genitalia may reveal a hair tourniquet, hernia, or
signs of testicular torsion and is an important part of the examination.

A thorough extra-abdominal examination is indicated in most children with
abdominal pain. For example, failure to examine the throat may lead to a missed
diagnosis of pharyngitis, which may be associated with abdominal pain. Lower
lobe pneumonias also can mimic an abdominal emergency. The general exami-
nation also includes an assessment of the child’s hydration status. Classic signs
and symptoms of dehydration in infants and young children are dry mucous
membranes, decreased tearing, sunken eyes and fontanelles, decreased skin tur-
gor, prolonged capillary refill, and decreased urine output. Interestingly, most of
these signs have not been well studied, and some may not be reliable.

For the surgical disease processes discussed in this article, pain is typically the
chief complaint. Management of the child’s pain during the evaluation is of paramount
importance. The use of pain medication in children with abdominal pain does not
appear to increase the risk of misdiagnosis [1]. In fact, often a better physical
examination can be accomplished once the patient’s pain has been addressed.

Gastroenteritis

Epidemiology

Acute gastroenteritis (AGE) is the most common gastrointestinal inflamma-
tory process in children. The cause is usually viral, and rotavirus is the most
common virus. In the United States, 200,000 children are hospitalized every year, and 300 to 400 deaths are caused by diarrheal disease. Costs to health care are estimated at $2 billion per year. Rotavirus is the most significant cause of severe diarrhea in childhood, with a peak incidence between 4 and 23 months of age. A rotavirus vaccine was to be part of the routine immunization schedule recommended by the American Academy of Pediatrics; however, the Centers for Disease Control and Prevention does not recommend the vaccine as of 1999 because of the significant number of bowel obstructions and intussusception cases that occurred after the first vaccines were administered. Further studies are underway and may be promising for a new vaccine. Norwalk virus is responsible for up to 40% of diarrheal disease in older children. Campylobacter is the leading cause of bacterial diarrhea in the United States.

Presentation

Vomiting usually precedes the diarrhea by as much as 12 to 24 hours. A low-grade fever may or may not be associated with AGE. When the parent states that the child is vomiting “everything,” clarify how much the child is taking in at one time (many times a child will drink too much at one time and then vomit). Children who are mildly dehydrated may not manifest clinical signs. Decreased urine output can be a late sign of dehydration. The children who are more at risk for dehydration include those who are younger than 12 months old; those with frequent stools (more than 8 per day); those with frequent vomiting (more than twice per day); and those who are severely undernourished. Examination of the abdomen usually reveals a nondistended soft abdomen with no localized tenderness (may be diffusely, mildly tender), and usually there is minimal to no guarding. AGE may cause an ileus in severe cases.

Viral diarrhea will target the small bowel, resulting in midabdominal cramping and large volumes of watery diarrhea. Bacterial diarrhea will target the large bowel, resulting in lower abdominal pain and smaller volumes of bloody mucoid diarrhea. A bacterial cause should be considered in any child who has a history of travel, has been exposed to an epidemic in daycare, or has higher fevers, bloody stools, or severe cramping. Other diagnoses to consider when a child presents with vomiting include urinary tract infection, appendicitis, inborn errors of metabolism, or volvulus, especially in very young infants, diabetic ketoacidosis, and hemolytic uremic syndrome (the appearance of illness in children usually is preceded by diarrhea).

The assessment of dehydration can be based on the known pre-illness weight in kilograms. The problem in the ED is that parents rarely know the exact weight of a child, especially in kilograms, and scales may vary slightly. If the pre-illness weight is known, then every kilogram of weight lost corresponds to a loss of 1 L of body fluid. The assessment of dehydration also can be based on clinical findings. The percentage of dehydration based on clinical findings such as dry mucous membranes or decreased urine output can vary slightly from reference
to reference. When clinical signs of dehydration are found in infants, the infants will have a higher percentage of dehydration than in older children (Table 1). The more clinical signs of dehydration the child has, the higher the percentage of dehydration will be [2,3].

**Laboratory and radiology findings**

A blood glucose check is recommended if the vomiting or abdominal pain is prolonged or associated with polyuria or polydipsia, to rule out diabetic ketoacidosis. Hypoglycemia in an alert child is generally not a concern [4]. Electrolytes generally do not need to be checked in well-appearing children who have signs of mild dehydration. The American Academy of Pediatrics (AAP) does not recommend electrolytes in every child; rather, the AAP recommends electrolytes in children who have acute gastroenteritis with, among other signs, an altered mental status, clinical signs of moderate to severe dehydration, clinical signs of hypernatremia or hypokalemia, prolonged, severe diarrhea (≥ 48 hours) as a risk for hypokalemia, infants who are ≤ 6 months old, and suspicious or unusual histories. The bicarbonate level has not been shown to be well correlated with the degree of dehydration.

A urinalysis to rule out infection is recommended, especially in young females who present only with abdominal pain and vomiting. A young infant who has vomiting and clinical signs of dehydration without ketones in the urine may have an inborn error of metabolism. Stool cultures generally are not necessary in most children who present with vomiting and diarrhea. Cultures should be considered in cases of admission, systemic illness, travel history, daycare exposure, a food- or water-borne source, recent antibiotics, bloody or mucoid stools, immunocompromised infants, or epidemics.
Management

Children with clinical signs of dehydration need rehydration. Rehydration can be administered either orally or through a nasogastric tube or an intravenous line. If a child has signs of severe dehydration, altered mental status, or evidence of an ileus, then rehydration should occur through an intravenous line.

An oral challenge is not oral rehydration. If a child is going to be orally rehydrated, then parents need to be instructed on the proper techniques of oral rehydration. Oral rehydration is very effective but is more labor intensive for children and parents [5]. However, when surveyed, parents actually prefer oral rehydration compared with intravenous rehydration. The commercially available rehydration solutions (eg, Pedialyte or Rehydralyte) are fairly close to delivering the optimal amount of sodium and glucose recommended by the World Health Organization (sodium plus 60–90 mEq and dextrose 2.0%). Many homemade recipes (eg, a water, salt, and sugar mixture) for oral rehydration solution can be found on the Internet.

The key to successful oral rehydration in children who present with vomiting is to offer small amounts at a time; for example, 5 cc (sips) for young children or 15 cc (tablespoon) for older children every 2 minutes. A syringe or a 5-F feeding tube attached to a syringe can be used to help facilitate the oral rehydration. The parent also can drip the solution slowly into the child’s mouth or through the nares into the posterior pharynx. If vomiting occurs, wait 10 to 15 minutes and try again. The child should receive either 50 cc/kg orally for mild to moderate dehydration or 100 cc/kg for moderate to severe dehydration, over 3 to 4 hours. Children who experience ongoing losses, such as by continued diarrhea, should receive an additional 10 cc/kg of rehydration.

Breast feeding infants can be rehydrated using more frequent, shorter feeds. Another option for oral rehydration is the use of frozen rehydration popsicles such as Revital-ICE. Placing a nasogastric tube is an alternative route for hydration in the child who refuses absolutely to take anything by mouth or in whom an intravenous line cannot be established but is clinically stable [6].

Intravenous rehydration should be used for any child who fails oral rehydration or has signs of severe dehydration, an ileus, or an altered mental status. Many experts recommend a minimum of 30 to 40 cc/kg in cases of mild to moderate dehydration, which will correct dehydration of 3% to 4%. Antiemetics and antidiarrheal medications are not recommended currently by the AAP because of limited literature to support their use [7]. Prochlorperazine, promethazine, and metoclopramide have been shown to be of some benefit but have some increased risk of sedation and an increased risk of dystonic reaction in children. However, many emergency and pediatric emergency physicians understand that is cruel to allow a child to remain nauseous and vomiting in the ED. Ondansetron (Zofran), a 5-hydroxytryptamine-3 receptor antagonist, which has been used for years as chemotherapy for pediatric nausea, has now been studied in emergency departments for children with acute gastroenteritis [8,9]. More literature is needed to support the use of antidiarrheal agents in
children, but many emergency physicians use these medications in otherwise healthy older children who are presumed to have viral diarrhea.

Live bacterial cultures, such as *Lactobacillus* in yogurt, have been shown to help treat infectious diarrhea and to help prevent diarrhea associated with antibiotics [10]. Antibiotics are not recommended for most children who are presumed to have viral AGE. In children with confirmed bacterial diarrhea, the role of antibiotics in treating infections by *Campylobacter jejuni*, *Escherichia coli*, and *Yersinia* is unclear. Nontyphoid *Salmonella* infection is self-limiting and may have prolonged excretion with antibiotic therapy. However, the treatment for *Salmonella* is indicated in infants less than 3 months of age, who have a history of immunodeficiency or hemoglobinopathy. Antibiotic therapy should not be initiated unless *E coli* 0157:H7 has been excluded because patients may develop hemolytic uremic syndrome from empiric antibiotic use. *Shigella* infection may be treated with trimethoprim-sulfamethoxazole, 8 mg/kg/d, divided twice per day, or erythromycin, 40 mg/kg/d, divided four times per day, for 5 to 7 days. Erythromycin is the drug of choice for treating *Campylobacter* infection. *Giardia* may be treated with metronidazole, 15 mg/kg/d, divided three times per day, for 7 days. *Clostridium difficile* infections may be treated with oral vancomycin, 50 mg/kg/d, divided four times per day, or metronidazole for 7 days.

Resuming formula feeding in young infants or solids in older children as soon as possible should also be encouraged. Transient lactose intolerance may develop, especially during AGE caused by rotavirus, but is transient. Most children can return to eating milk products or formula. Occasionally, lactose intolerance persists and may be a cause of post-AGE diarrhea. If persistent diarrhea occurs after the reintroduction of milk products or if the stool is acidic and contains more than 0.5% reducing substances, a lactose-free formula should be considered.

### Constipation

Parents often worry that their infant or child is constipated, particularly because it is common for infants to strain and turn red in the face during bowel movements. Unfortunately, a uniform definition of constipation has yet to be determined. The best way is to define constipation is not by the frequency of the stool but by the difficulty or painful passage of large or hard stools. Newborns typically have a meconium stool in the first 48 hours of life and then can range from zero to 12 stools per day for the first week of life. The stools of breast-fed infants are very soft and pale yellow and often occur after each feeding. However, bottle-fed infants tend to have firm, formed, yellow stools one to four times per day. When infants are 3 to 4 months of age, stool frequency decreases, with some bottle-fed infants passing one stool every other day. Most children develop the adult pattern of having a mean of 1.2 stools per day by 4 years of age.
Causes

The more common serious causes of constipation in the newborn and infant are imperforate anus, anal stenosis, meconium plug syndrome, meconium ileus, Hirschsprung’s disease, volvulus, anal fissure, infant botulism, hypocalcemia, hypercalcemia, and hypothyroidism. Constipation in the older infant or child is related commonly to changes in diet, especially from breast milk to formula or advancement to solid baby foods. Inadequate fluid intake is another common cause of constipation. The school-aged child may present with constipation caused by high carbohydrate diets and a hesitance to go to the bathroom at school. The child who has rectal retention and encopresis has fecal soiling of the underpants and may paradoxically complain of diarrhea. A lower abdominal mass may be found by palpation, and fecal impaction may be found on rectal examination. Older children may present with abdominal pain, which may be in the right lower quadrant and mimic appendicitis.

Presentation

Pertinent history that should be obtained from the caregiver includes the time after birth of the first bowel movement, frequency of bowel movements, consistency and size of stools, presence of pain with bowel movements, and associated systemic findings such as fever, weight loss, and vomiting. Dietary habits should be a particular focus, and a medication history should also be obtained. A complete physical examination should be performed, including an abdominal palpation for masses, inspection of the perineum and perianal area for fissures, and imperforate anus or stenosis. A plain abdominal radiograph is helpful in confirming the diagnosis when the history or physical examination is confusing or inconclusive.

Laboratory and radiologic findings

Laboratory tests and radiologic studies generally are unnecessary in the diagnosis and management of constipation in young children. An abdominal series or flat-plate radiograph of the abdomen can confirm that the colon has a significant amount of stool present.

Management

If fecal impaction is present, disimpaction is necessary. Oral medications include mineral oil, 1 to 4 mL/kg/dose, once or twice per day (contraindicated in infants and in children at risk for aspiration); lactulose, 1 to 2 mL/kg/dose, once or twice per day; milk of magnesia, 1 to 3 mL/kg/dose, once or twice per day, or with medications containing polyethylene glycol (PEG); or sorbitol, senna, or bisacodyl [11]. A tasteless, commercially available electrolyte-free PEG solution (MiraLax) can be mixed with any clear liquid beverage [12]. It is prepared by
dissolving 1 capful (17 g) of powder in 8 oz of liquid and giving the child 10 to 14 mL/kg/d in two divided doses. Rectal disimpaction also can be performed. However, hypertonic phosphate enemas have been associated with severe, acute hypocalcemia and cardiac arrest in infants [13]. Tap water enemas have been associated with acute hyponatremia, seizures, and death [14]. In the older infant and toddler, milk of magnesia, mineral oil, or lactulose can be used. Docusate (Colace), 5 to 10 mg/kg/d or senna extract (Senokot), 5 to 10 mL daily, can be safely used in older children.

Maintenance therapy of constipation is most appropriately managed by a primary care clinician. Dietary management includes increasing fluid intake and adding fiber and fruits such as prunes, pears, or plums to the diet. A barley extract (Maltsupex) or Karo syrup can be recommended safely for infants in a dosage of 1 to 2 teaspoons two to four times daily, added to formula, juice, or food. Behavioral modification for the older child includes regular toilet sitting, stool diaries, and reward systems. If an anal fissure is discovered, management includes frequent, gentle, thorough cleansing of the anus and liberal lubrication with petroleum jelly. A stool softener must be used, and a topical anesthetic ointment may be helpful to avoid a pattern of pain and stool retention.

Appendicitis

Causes

Appendicitis is the abdominal pain most commonly treated surgically in childhood, affecting four of every 1000 children. Appendicitis is the cause of pain in 2.3% of all the children with abdominal pain seen in ambulatory clinics or EDs. Of all the children admitted to the hospital with abdominal pain, 82% are diagnosed with appendicitis [15]. Because of the difficulty in evaluating young children with abdominal pain, perforation rates for appendicitis are higher than in the general adult population (30%–65%). Moreover, because the omentum is less developed in children, perforations are less likely to be “walled off” or localized, leading to generalized peritonitis.

Presentation

The classic presentation, consisting of generalized abdominal pain migrating to the right lower quadrant, associated with nausea, vomiting, and fever, is seen less often in the pediatric patient [16]. In addition, children often present earlier in their clinical course than adults do, when only mild or less specific symptoms are present. However, limited data appear to indicate that individual signs such as rebound tenderness and Rovsing’s sign have a high sensitivity and specificity in children [4].

The most common findings of appendicitis in children are right lower quadrant pain, abdominal tenderness, guarding, and vomiting [17]. If available, a hist-
tory of abdominal pain preceded by vomiting can be helpful in distinguishing appendicitis from acute gastroenteritis. Very young children commonly have diarrhea as the presenting symptom [18]. Bearing in mind the special techniques discussed above for eliciting peritoneal irritation, the EP should also remember that the position of the appendix can vary greatly, and tenderness may be found in locations other than the classic McBurney point. Although the rectal examination is not usually helpful in making a diagnosis of appendicitis [19], some authors advocate a rectal examination in infants, in whom there may be a palpable rectal mass in up to 30% of cases [20]. Changes in skin temperature over the area of the appendix have not been shown to be helpful in the diagnosis of appendicitis [21].

**Differential diagnosis**

Gastroenteritis is the most common diagnosis in cases of missed appendicitis. Although enteritis caused by *Y enterocolitica* and *Y pseudotuberculosis* has been termed the “great imitator” of appendicitis, in reality, the amount of diarrhea in gastroenteritis is usually more pronounced. Appendicitis is also frequently mistaken for a urinary tract infection (UTI), which may also present with abdominal pain and vomiting. A study reported by Reynolds [22] in 1993 showed that missed cases of appendicitis were more likely to have diarrhea, to not be anorexic, and to be afebrile.

**Laboratory evaluation**

No laboratory test is 100% sensitive and specific for appendicitis. The white blood cell count (WBC) can be helpful in the diagnosis, although, by itself, it is neither specific nor sensitive for appendicitis and therefore cannot be used alone to rule in or rule out the disease [23]. The WBC, however, can be used as an adjunct, after the clinical suspicion of appendicitis is estimated. If clinical suspicion is low before any laboratory or other investigations (for example, in a child who has vomiting and diarrhea but minimal abdominal tenderness) and the WBC is normal, the likelihood of appendicitis becomes very low. If the WBC is high, the likelihood of appendicitis is raised sufficiently to warrant further tests or observation.

A urinalysis should be performed; however, caution must be exercised in its interpretation, because mild pyuria, hematuria, and bacteriuria can all be present if an inflamed appendix is located adjacent to a ureter. The presence of C-reactive protein also has been studied as a marker for appendicitis [24–27], but it is not significantly more sensitive or specific than the WBC.

**Diagnostic radiology**

Plain film abdominal series typically have nonspecific findings and are of low yield in cases of appendicitis [15]. Appendicoliths are present only in approximately 10% of true appendicitis cases. Barium enemas have also been
used, with the principle that an inflamed appendix will fail to fill and will not be visualized. Unfortunately, 10% to 30% of normal appendices are not visualized with barium studies, creating a high number of false-positive results [28].

Ultrasoundography is considered by many experts to be the imaging test of choice in children. Ultrasonography is noninvasive, rapid, and can be performed at the bedside. It does not require oral contrast, which is an advantage for patients who may require surgery. It also spares the pediatric patient exposure to radiation. The normal appendix in pediatric patients is visualized readily by ultrasonography because there is usually less abdominal wall fat than in adults. Graded compression of the appendix is used to determine the presence or absence of inflammation. An inflamed appendix is usually aperistaltic, difficult to compress, and measures ≥6 mm in diameter. It is important for the ultrasonographer to visualize the entire appendix to avoid a false-negative reading because sometimes only the distal tip of the appendix is inflamed. The mucosal lining may be intact or poorly defined, and a fecolith may or may not be present. A periappendiceal fluid collection may indicate an early perforation but may result simply from inflammation. Experienced ultrasonographers can achieve sensitivities of 85% to 90% and specificities of 95% to 100% in acute appendicitis [29–37]. However, studies have not shown an improvement in outcome measures such as a decrease in negative laparotomies or time to the operating room [38,39]. Color flow Doppler ultrasonography is now being added to increase the accuracy of the sonographic examinations. Doppler measurement demonstrates an increase in blood flow to the area of an inflamed appendix [40].

In recent years, CT has become the test of choice for pediatric surgeons when ultrasonography fails to give a definitive diagnosis [41]. Every variation, from triple-contrast (intravenous, oral, and rectal) CT scanning to noncontrast, unenhanced CT, has been used [42,43]. CT offers the advantage of greater accuracy, the ability to identify alternative diagnoses, and in some studies, lower negative laparotomy rates [44]. Although CT appears to be better than ultrasonography in making the diagnosis of appendicitis in children [45], it is slower, requires oral contrast in most centers, and exposes the young child to significant radiation. If the child is vomiting, keeping the oral contrast in the gastrointestinal tract can be a challenge, and antiemetics may be required.

Leukocyte imaging studies [46] and technetium scans [47] have been used for equivocal cases of abdominal pain in children. The overall sensitivity, specificity, and accuracy, however, are lower than with CT. Magnetic resonance imaging is also superior in its ability to diagnose appendicitis in children [48], but it may not be available or practical. No study can be relied on for 100% accuracy. If clinical suspicion is high and imaging studies are negative, the child should be hospitalized for observation and serial examinations.

Management

When the clinical suspicion for appendicitis is high, consultation with a surgeon is warranted before any radiologic study. Nonetheless, many surgeons
will request a diagnostic study to decrease the likelihood of a negative laparotomy. When the diagnosis of appendicitis is made, then preparing the child for the operating room is essential. Usually the oral intake of these children has been limited during the day or days before presentation, and intravenous fluids are necessary. Electrolyte imbalances should also be addressed, although significant abnormalities are not common in children with appendicitis.

If there are clinical or radiologic signs of perforation, antibiotics with gram-negative and anaerobic coverage should be started in the ED [49]. A few studies have shown a benefit to antibiotic therapy in decreasing infectious complications in children with uncomplicated, nonperforated appendicitis as well [50]. Diagnosing appendicitis early is the key to a better outcome. Any child who is evaluated in the ED with a chief complaint of abdominal pain and who is considered well enough to go home but in whom the diagnosis of appendicitis has not been ruled out should be asked to return to the ED within 8 hours for another evaluation of the abdomen.

**Intussusception**

*Pathophysiolo*gy

Intussusception was first described over 300 years ago. It is the prolapse of one part of the intestine into the lumen of an immediately distal adjoining part. The most common type is ileocolic invagination. During the invagination, the mesentery is dragged along into the distal lumen, and venous return is obstructed. This leads to edema, bleeding of the mucosa, increased pressure in the area, and eventually obstruction to arterial flow. Gangrene and perforation result.

*Causes*

Intussusception is seen most frequently between the ages of 3 months and 5 years, with 60% of cases occurring in the first year and a peak incidence at 6 to 11 months of age. The disorder, which appears predominantly in males, was once believed to occur more often in the spring and autumn, although now it appears it has no seasonality [51,52]. Although it is usually idiopathic in the younger age groups, children older than 5 years often have a pathologic “lead point” for intussusception, such as polyps, lymphoma, Meckel’s diverticulum, or Henoch-Schöenlein purpura and require a work-up to determine the underlying cause.

*Presentation*

The classic triad of intermittent colicky abdominal pain, vomiting, and bloody mucous stools is encountered in only 20% to 40% of cases. At least two of these findings will be present in approximately 60% of patients. The vomiting is not
necessarily bilious because the level of obstruction is low in the ileocecal area. A palpable abdominal mass in the right upper or lower quadrant is an uncommon finding [53].

Abdominal pain associated with intussusception is colicky, lasts for approximately 1 to 5 minutes at a time, and then abates for 5 to 20 minutes. During episodes of pain, the child cries and may draw the knees upward toward the chest. Although the child often looks better between episodes, he or she still usually appears ill, quiet, or exhausted. Gradually, irritability increases and vomiting becomes more frequent and sometimes bilious. Fever may also develop at this point as the child deteriorates.

If a colicky episode is not witnessed by the ED staff, the EP should ask the parents to describe or demonstrate what the child was doing during the episodes. Most parents of a child who has gastroenteritis do not indicate that their child is in pain. Parents of a child who presents with intussusception usually believe that the child is in pain before or during episodes of vomiting. Intussusception also can present with lethargy, pallor, and unresponsiveness. It is important to keep this diagnosis in mind when dealing with an infant who has an altered mental status [54].

The abdomen may be distended and tender, but usually the pain appears to be out of proportion to the physical examination. There may be an elongated mass in the right upper or lower quadrants. Any type of blood in the stool may be caused by intussusception. Rectal examination may reveal either occult blood or frankly bloody, foul-smelling stool, classically described as “currant jelly” [55]. However, frank rectal bleeding is a late and unreliable sign; its absence should not deter the EP in the pursuit of the diagnosis. It should also be noted that what appears to be blood in a child’s stool may be something else, such as red fruit punch or Jell-O, therefore, guaiac testing may prevent this error when there is some question. A period of observation in the ED for the recurrence of a pain episode is helpful in equivocal cases. Specifically noting the absence of such episodes during ED observation is good practice and should be documented in the clinical record.

**Differential diagnosis**

Gastroenteritis presents typically with more diarrhea than intussusception, and the child usually has ill contacts. The presence of any degree of blood in the stool should also raise suspicion for a more serious condition. Bleeding from a Meckel’s diverticulum usually is painless, unless the diverticulum becomes inflamed.

An incarcerated hernia or testicular or ovarian torsion may also present with sudden abdominal pain and vomiting. Inspection of the genitalia, especially in males, is vital. With torsion, the rectal examination does not show occult or frank blood. Renal colic presenting with pain and vomiting generally is not seen in young children.
Laboratory tests

No laboratory test reliably rules in or out the diagnosis of intussusception. If the bowel has become ischemic or necrotic, acidosis may be present.

Diagnostic imaging

Unfortunately, plain abdominal films are neither sensitive nor specific for intussusception [56,57]. Plain films initially may appear normal. As the disease progresses, a variety of abnormalities may be seen, including a visible abdominal mass, abnormal distribution of gas and fecal contents, air fluid levels, and dilated loops of small intestine. A “target sign” on plain film consists of concentric circles of fat density, similar in appearance to a doughnut, visualized to the right of the spine. This sign is caused by layers of peritoneal fat surrounding and within the intussusception alternating with layers of mucosa and muscle. Less commonly, the soft tissue mass of the intussusception (leading edge) can be seen projecting into the colon. Large areas of gas with the head of the intussuscepted bowel may take the shape of a crescent, although other patterns may be seen.

Ultrasonography is used in some institutions to diagnose intussusception and to confirm reduction after treatment [58]. Sonographic findings in intussusception include the target sign, a single hypoechoic ring with a hyperechoic center and the “pseudokidney” sign, superimposed hypo- and hyperechoic areas representing the edematous walls of the intussusceptum and layers of compressed mucosa. Doppler flow may be used to identify bowel ischemia. If signs of intussusception are not identified by ultrasonography in cases in which the diagnosis is suspected clinically, proceeding with a barium or air enema should still be considered.

Management

The main focus in the management of a child who has intussusception is emergent reduction of the obstructed bowel. Classically, this reduction is accomplished by a barium enema, which acts as both a diagnostic and therapeutic radiologic study. The barium enema has been the gold standard for both the diagnosis and treatment of intussusception for decades [59]. Saline enemas have also been used successfully [60,61], and newer modalities such as air enemas and ultrasonographically guided enemas have emerged.

Many centers in the United States are now moving toward air enemas [62–67]. This modality was first introduced to the Western world at the American Pediatric Surgical Association meeting in 1985, with the presentation of a series of 6396 successfully treated patients [68]. Air enemas offer several advantages over barium enemas. They are easier to administer, and in most studies, they have a higher rate of successful reduction. Air enemas using fluoroscopic guidance deliver much less radiation than barium studies, and if ultrasonography guidance is used, there is no exposure. Limiting radiation exposure is important to consider
when dealing with infants and their susceptible reproductive organs; and if a perforation occurs during these investigations, air is much less dangerous to the peritoneum and abdominal contents than barium is.

Visualization of the entire colon to the terminal ileum is mandatory to rule out ileocolic intussusception. Ileo–ileo intussusception can be much harder to diagnose and much harder to reduce. Spontaneous reduction of intussuscepted bowel has been reported, although in a patient with significant symptoms, therapeutic intervention should not be delayed [69].

Not every child who has intussusception should undergo bowel reduction by enema. Clinical signs of peritonitis, perforation, or hypovolemic shock are clear contraindications to enemas. These signs mandate surgical exploration. Relative contraindications to enemas include prolonged symptoms (≥ 24 hours), evidence of obstruction such as air fluid levels on plain abdominal films, and ultrasonography findings of intestinal ischemia or trapped fluid.

Even in well-selected patients, enemas may cause the reduction of necrotic bowel, perforation, and sepsis. After a successful reduction, the child should be admitted for observation. A small percentage of patients (0.5%–15%) will have a recurrence of the intussusception, usually within 24 hours but sometimes after days or weeks. Even after reduction by laparotomy, the recurrence rate is 2% to 5% [52].

**Small bowel obstruction**

*Pathophysiology*

Small bowel obstruction may result from intrinsic, extrinsic, or intraluminal disease. Although the most common causes of small bowel obstruction are adhesions from previous abdominal surgery and incarceration of a hernia [70], intussusception, appendicitis, Meckel’s diverticulum, malrotation with midgut volvulus, and tumors also should be considered as possible causes. In addition to inguinal hernias, umbilical, obturator, and femoral canal hernias may also lead to small bowel obstruction [56].

*Presentation*

As obstruction develops, decreased oral intake occurs and vomiting ensues, often becoming bilious in nature. This is followed by obstipation. Abdominal distension and tenderness occur, and the abdomen may be tympanic to percussion. If the small bowel obstruction is caused by mechanical compression, high-pitched bowel sounds with “rushes” may be heard. When intraluminal pressure becomes higher than the venous and arterial pressures, ischemia develops in the bowel, and hematochezia may be seen. As with most abdominal emergencies in children, hematochezia is a late finding. Sepsis is another late finding because bacteria from the ischemic bowel enter the blood.
Differential diagnosis

Abdominal pain and vomiting also can be seen with other processes such as appendicitis. As time passes, a bowel obstruction will develop more abdominal distension than is seen typically in other processes. The lack of stool or gas passage points toward bowel obstruction. It is important to remember that the underlying cause of the obstruction may be as important to recognize as the obstruction itself.

Laboratory tests

No laboratory test is diagnostic of a bowel obstruction. Elevated levels of blood urea nitrogen, creatinine, and hematocrit may signify dehydration.

Diagnostic radiology

Plain abdominal films should be obtained when obstruction is suspected. A paucity of air in the abdomen is the most common finding in young children with bowel obstruction. Distended loops of bowel may be seen; however, smooth bowel walls are more common than distended bowel in small children. Multiple air–fluid levels also are seen commonly with small bowel obstruction. In later presentations, the bowel may resemble a tangle of hoses or sausages. An upright or lateral decubitus film will help to determine whether free air is present, caused by perforation. Further study with ultrasonography, CT, an upper-GI series, or an enema should be performed when there is suspicion of underlying pathologies such as appendicitis, midgut volvulus, and intussusception.

Management

Immediate surgical consultation is indicated when a bowel obstruction is seen on plain radiographs. Morbidity and mortality are increased if the obstruction is not treated within 24 hours [71]. The patient should be aggressively hydrated with normal saline boluses, and a nasogastric tube should be placed for gastric decompression. Broad-spectrum antibiotics are indicated, particularly if peritonitis is suspected.

Incarcerated hernia

Causes

Inguinal hernias occur in 1% to 4% of the population, more often in males (6:1), and more often on the right side (2:1). Premature infants are at a higher risk for hernias (30%), and 60% of incarcerated inguinal hernias occur during the first
year of life. Umbilical hernias are also commonly seen in the infant population. Unlike inguinal hernias, umbilical hernias will rarely become incarcerated and usually will close without surgery by 1 year of age. Other disorders place patients at an increased risk for abdominal hernias, including ventriculoperitoneal shunts, peritoneal dialysis, Marfan’s syndrome, cystic fibrosis, mucopolysaccharidoses, and hypospadias.

**Presentation**

Hernias usually present with an asymptomatic bulge in the groin or umbilical area, made more prominent with crying, straining, or laughing. The first sign of incarceration of an inguinal hernia is an abrupt onset of irritability in the young infant. Refusal to eat soon follows, followed by vomiting, which may become bilious and sometimes feculent.

Inguinal hernias may be palpated as smooth, firm, sausage-shaped, mildly tender masses in the groin; the hernia originates proximal to the inguinal ring and can extend into the scrotum. The “silk glove sign” occurs when the index finger rubs over the proximal spermatic cord and sometimes two layers of hernial sac can be felt rubbing together. If the child appears to be well, without vomiting, fever, or redness to the inguinal area, then the hernia is not likely incarcerated.

**Differential diagnosis**

There are many conditions that may mimic an inguinal hernia, but the most common condition is a hydrocele. A hydrocele is the result of incomplete obliteration of the process vaginalis, which allows an out-pocketing of peritoneum to appear in the scrotum. This fluid-filled sac can appear anywhere from the spermatic cord to the testicle, and if it is large, it can be transilluminated. Hydroceles can be palpated separately from the testes and are freely movable. A hydrocele usually appears in the first few months of life and disappears by 1 year of age.

**Diagnostic imaging**

If the diagnosis is uncertain, a scrotal ultrasonogram can differentiate an inguinal hernia from a hydrocele.

**Management**

If signs of incarceration are not present, a reduction should be attempted in the ED. Reduction of the hernia can be accomplished usually by placing the child in Trendelenburg position with ice packs to the area and the administration of pain medication. If reduction is not possible or if the hernia appears incarcerated or ischemic, emergent surgical consultation is required. Umbilical hernias rarely will become incarcerated and often will close without surgery.
Meckel’s diverticulum

Pathophysiology and causes

Meckel’s diverticulum is the most common congenital abnormality of the small intestine. Meckel’s diverticulum is a remnant of the omphalomesenteric (vitelline) duct that disappears normally by the seventh week of gestation. It is a true diverticulum, containing all layers of the bowel wall. Up to 60% of these diverticuli containing heterotopic gastric tissue and heterotopic pancreatic, endometrial, and duodenal mucosa have also been reported [72,73]. The features of Meckel’s diverticulum are commonly described by “the rule of 2s” [70]: it is present in approximately 2% of the population with only 2% of affected patients becoming symptomatic. Forty-five percent of symptomatic patients are less than 2 years of age [74]. The most common location is 2 feet (40–100 cm) from the ileocecal valve, and the diverticulum typically is 2 inches long.

Clinical presentation

The classic presentation of Meckel’s diverticulum is painless or minimally painful rectal bleeding. Isolated, red rectal bleeding is common, particularly in boys less than 5 years of age [75]. Such painless bleeding is a result of heterotopic gastric tissue in the diverticulum or in the adjacent ileum. Abdominal pain, distension, and vomiting may occur if obstruction has occurred, and the presentation may mimic appendicitis or diverticulitis. Meckel’s diverticulum may also ulcerate and perforate, presenting as a bowel perforation, or act as a lead point, resulting in intussusception.

Differential diagnosis

The differential diagnosis includes both painful and nonpainful conditions. Rectal bleeding associated with abdominal pain may be caused by peptic ulcer disease, intussusception, and volvulus. Nonpainful rectal bleeding may be caused by polyps, arteriovenous malformations, and tumors.

Laboratory tests

Although no laboratory test is diagnostic of Meckel’s diverticulum, children with gastrointestinal bleeding should undergo screening laboratory tests such as a complete blood count, coagulation profile, and a type and screen.

Diagnostic radiology

Abdominal films may show signs of obstruction such as dilated loops of bowel or a paucity of bowel gas. Scanning Meckel’s diverticulum involves an intravenous injection of technetium-pertechnetate. This test relies on the presence
of gastric mucosa in or near the diverticulum that has an affinity for the radionucleotide. A scan of Meckel’s diverticulum can detect the presence of gastric mucosa within the diverticulum with up to 85% accuracy [76]. Mesenteric arteriography can detect the site of active bleeding if bleeding is profuse.

Management

As carried out in any patient with active bleeding, fluid resuscitation is warranted, starting with boluses of normal saline, 20 cc/kg. A blood transfusion may be necessary, with a packed red blood cell increment of 10 cc/kg. The patient should have nothing by mouth, and a nasogastric tube should be placed. Antibiotic therapy must be initiated if there are peritoneal signs. Surgical consultation should be obtained emergently. Surgical intervention may involve a diverticulectomy or a more extensive small bowel segmental resection if there is irreversible bowel ischemia.

Very young infants

Very young infants, those less than a few months old, also have unique gastrointestinal conditions. Colic should be considered a diagnosis of exclusion. Hypertrophic pyloric stenosis is a common presentation, and surgical correction does not need to be immediate. Volvulus caused by congenital malrotation is a true surgical emergency, and consultation with a pediatric surgeon should be immediate once the diagnosis is considered. Fortunately, necrotizing enterocolitis, another gastrointestinal condition of newborns with serious sequelae, is usually seen by pediatric colleagues in the newborn nursery or neonatal intensive care unit.

Colic

Colic affects 1 in 6 families and is more likely to be reported by older mothers with longer full-time education and nonmanual occupations. To this day, the cause of colic remains unclear but is believed to be related to increased gas production in the infant’s intestines and, possibly, to neurologic or psychologic reasons. Other experts consider colic to be part of the normal distribution of crying.

Presentation

Colic appears usually during the second week of life and is characterized by screaming episodes and a distended or tight abdomen; some infants will draw up
their legs, pass gas, cry, and act miserable for hours. Episodes may last minutes to hours, occurring usually in the evening. One common definition used is 3 hours per day, 3 days per week, and at least 3 weeks in duration. Severity can increase around 4 to 8 weeks of age and will usually resolve around 12 weeks of age.

Growth and development remain unchanged, and the physical examination is unremarkable. No vomiting, diarrhea, fever, or weight loss occurs with colic. For any inconsolable crying infant, other correctable causes must be considered (Box 2). Parents may become overwhelmed and frustrated with a constantly crying young infant; look for signs that a parent is not coping before it becomes a child abuse case. This diagnosis occurs early in life; a suddenly irritable or poorly feeding 8-week-old who was previously healthy is less likely to have colic.

Treatment

There are no medications or treatments that have proven to be very effective and yet safe. Anticholinergic medications work but have too many side effects, such as seizures, respiratory trouble, syncope, and coma; therefore, they are not recommended. Simethicone has not been found to reduce colic. Switching to soy- or whey-based formulas has not been proven definitely to work [77]. Techniques such as swaddling the infant, using a pacifier or the rocking motion of car ride, or placing the infant in a car seat on top of a moving clothes dryer (watch car seat does not fall off dryer) also may work to calm the infant. Reassuring parents that episodes of colic will pass is the best antidote. Encourage parents to allow themselves “time outs” from the child, allowing someone else they trust to care for the child during a crying episode.

Box 2. The inconsolable, crying young infant

- Anal fissures
- Corneal abrasions
- Diaper pins
- Formula intolerance
- Fractures
- Hair tourniquets
- Hematoma
- Hernias
- Infections (eg, UTI or meningitis)
- Intussusception
- Otitis media
- Reactions to medications such as decongestants
**Hypertrophic pyloric stenosis**

*Pathophysiology*

Hypertrophic pyloric stenosis (HPS) is a narrowing of the pyloric canal caused by hypertrophy of the musculature. The cause of this condition remains unclear, but some experts theorize that HPS is caused by *Helicobacter pylori*, the same bacteria associated with peptic ulcer disease. This theory is based on non-specific evidence, such as the temporal distribution, seasonality, and familial clustering of HPS, along with the pathologic finding of leukocytic infiltrates, and the increased incidence seen in association with bottle-feeding [78].

*Causes*

HPS occurs in 1 of every 250 births and appears predominantly in males (male to female ratio of 4:1). The condition also has racial variation. It is observed to be more common in whites than in African Americans and is rare in Asians. Originally, first-born males were believed to be affected more often, but it is now known that birth order is not a factor. A child of an affected parent has an increased chance of HPS, with the risk being higher if the mother was affected [79].

*Presentation*

HPS usually presents during the third to fifth week of life. Symptoms begin rather benignly, with occasional vomiting at the end of feeding or soon thereafter. This is when HPS is often confused with a viral syndrome, gastroesophageal (GE) reflux, or milk intolerance. Emesis is nonbilious because the stenosis is proximal to the duodenum. As the disease progresses, the incidence of vomiting increases, now following every feed, and can become projectile. Comparing birth weight to current weight is a key element in the evaluation of a neonate with vomiting. After the first week, healthy neonates should gain approximately 20 to 30 g (1 ounce) per day. Healthy normal infants who “spit up” (regurgitate) will continue to gain weight and grow well. Infants with HPS will continue to be hungry but, because of repeated vomiting, may reach a plateau or even lose weight. An infant with HPS may also become constipated as the result of dehydration and decreased intake.

On examination, the neonate with HPS may appear normal but hungry, or the may have signs of dehydration. Dehydration may lead to the appearance of jaundice. Peristaltic waves moving from left to right may be seen in the left upper quadrant after feeding. A palpable “olive” or small mass in the right upper or middle quadrant, at the lateral margin of the right rectus muscle just below the liver edge, may also be detected during physical examination. Decompressing the stomach with a nasogastric tube first and using a lubricant on the fingertips may improve the ability to palpate this “olive.” Clinicians’ ability to palpate the
pyloric “olive” has decreased over the years, probably because of the addition of ultrasonography in confirming the diagnosis. In 1999, Abbas and colleagues [80] reported that many infants with HPS who have palpable masses on examination still undergo one or more unnecessary and redundant tests. This situation is associated with a delay in diagnosis, increased costs, and possibly adverse clinical health problems.

Differential diagnosis

The differential diagnosis for a vomiting neonate includes the life-threatening disorder of volvulus with or without associated malrotation of the intestine. Infants with volvulus deteriorate rapidly, and the vomiting will be bilious, eventually with signs of sepsis and bowel necrosis. Incarcerated hernias also can present similarly, as well as intussusception (although less commonly in the neonatal period). Viral gastroenteritis can occur in the neonate, but caution is advised when making this diagnosis in infants less than 6 weeks old. At a minimum, significant diarrhea and the presence of ill contacts should both be present before considering viral gastroenteritis.

GE reflux is much more common than pyloric stenosis, and vomiting in the neonatal period is often attributed to GE reflux when other diagnoses should be considered. Vomiting caused by GE reflux usually occurs during feeds or immediately afterwards. The amount of vomitus is smaller, and the neonate will continue to gain weight. Infections, especially in the urinary tract, also can present with vomiting as a chief complaint and an examination of the genitalia and urine is imperative in any infant who presents with vomiting.

Laboratory tests

Prolonged vomiting in HPS causes the infant to lose large quantities of gastric secretions rich in H⁺ and Cl⁻ ions. As a result of dehydration, the kidney attempts to conserve Na⁺ ions by exchanging them for K⁺ ions. The net result is a loss of both H⁺ and K⁺ ions. Therefore, the infant with HPS will initially demonstrate a hypokalemic, hypochloremic, metabolic alkalosis [81]. If the infant remains dehydrated for a long period, this alkalosis may eventually turn to acidosis.

Imaging studies

If no small mass or “olive” is palpable in the right upper or middle quadrant of a young infant with a clinical picture suggestive of HPS, further studies are warranted. Ultrasonography measures the thickness of the pyloric wall (normally ≤ 2.0 mm but in HPS is ≥ 4.0 mm) and the length of the pyloric canal (normally ≤ 10.0 mm but in HPS is ≥ 14–16 mm), leading to a diagnosis of HPS. Ultrasonography has been shown to have a sensitivity and specificity as high as 100% [82,83]. A false-negative result may occur if the ultrasonographer
measures through the distal stomach or antrum and not through the pylorus itself. A false-positive results if pyloric spasm is present and not pyloric stenosis.

If ultrasonography is nondiagnostic and HPS remains a concern, the next radiologic test of choice is an upper-GI series. The upper GI will show the classic “string sign” as contrast flows through the narrowed pyloric lumen. There will also be delayed gastric emptying. As with ultrasonography, false-positive results may occur because of pyloric spasm, which also gives the appearance of a string sign. Endoscopy also can be used to diagnose HPS but is not used commonly [84].

Management

Once HPS has been diagnosed, admission to the hospital is indicated. Often these infants are dehydrated and therefore hydration and correction of any electrolyte abnormalities should be started in the ED. The surgical procedure required to correct the stenosis is the Ramstedt procedure, which involves incising and separating the hypertrophic muscle fibers of the pylorus.

In Japan, intravenous atropine has been used to decrease the spasm of the pylorus as an alternative to surgery. It is then administered orally for several weeks until the child “outgrows” the stenosis. Surgery has been avoided in many cases [85]; however, surgery remains the standard treatment in the United States.

Malrotation with midgut volvulus

Pathophysiology

Congenital malrotation of the midgut portion of the intestine is often the cause of volvulus in the neonatal period. Malrotation occurs during the fifth to eighth week in embryonic life when the intestine projects out of the abdominal cavity, rotates 270°, and then returns into the abdomen. If the rotation is not correct, the intestine will not be “fixed down” correctly at the mesentery, and the vascular mesentery will appear more stalk-like in its structure and is at risk later for twisting, called volvulus. Volvulus is the twisting of a loop of bowel about its mesenteric base stalk attachment; ischemia subsequently develops, and this constitutes a true surgical emergency because bowel necrosis can occur within hours. The entire small bowel is at risk for ischemia and necrosis.

Causes

The incidence of volvulus peaks during the first month of life but can present anytime in childhood. The male to female ratio is 2:1, and this is rarely a familial disorder. The exact frequency of midgut volvulus is not known because it is frequently asymptomatic. Congenital adhesions, called Ladd’s bands, extending
from the cecum to the liver, are associated with congenital malrotation. These adhesions may cause external compression of the duodenum and obstruction. This condition is not generally considered a surgical emergency, but it eventually requires surgical intervention to lyse these bands.

Presentation

Volvulus may present in one of three ways: (1) as a sudden onset of bilious vomiting and abdominal pain in a neonate; (2) as a history of “feeding problems” with bilious vomiting that now appears like a bowel obstruction; and (3) although less commonly, as a failure to thrive with severe feeding intolerance [86]. Bilious vomiting in a neonate is always worrisome and is a surgical emergency until proven otherwise. If the bowel is already ischemic or necrotic, the neonate may present with a pale complexion and grunting. The abdomen may or may not be distended depending on the location of the volvulus. If the obstruction is proximal, there may be no distension. The abdominal wall may appear “blue” if the bowel is already ischemic or necrotic. The pain is constant, not intermittent, and the neonate will appear irritable. Jaundice also may be present. Hematochezia is a late sign and indicates intestinal necrosis. Neonates who have volvulus will gradually deteriorate if bowel remains ischemic.

Differential diagnosis

As stated earlier, bilious vomiting in a neonate is considered a surgical emergency until proven otherwise. However, in the early presentation of volvulus, vomitus may be nonbilious, and a misdiagnosis of acute gastroenteritis may result. As in the discussion of pyloric stenosis, the acute gastroenteritis should be diagnosed cautiously in young infants. In pyloric stenosis, vomitus is always nonbilious. The duration of symptoms with pyloric stenosis is usually longer, and the child usually appears well, although possibly dehydrated and hungry. Incarcerated hernias may also present with bilious vomiting. It is therefore imperative to thoroughly examine a vomiting neonate for signs of a hernia. Rarer causes of bilious vomiting include duodenal or ileal atresia, although this is discovered typically in the newborn nursery or soon after. With intestinal atresia, the neonate will not be as ill appearing as with volvulus. Necrotizing enterocolitis also can rarely appear in term neonates. Intestinal hematomas may occur in cases of child abuse.

Congenital adrenal hyperplasia (CAH) can cause bilious vomiting without anatomical obstruction. It may present in the first few weeks of life. CAH results in adrenal insufficiency with decreased cortisol levels and salt wasting. Infants will present with hypotension and electrolyte imbalance (low Na+ and high K+). It is more likely that CAH will be seen in male infants who present in the ED. Female newborns who have this condition are less commonly missed in the newborn nursery because the accumulation of androgenic compounds affects the external genitalia to a greater extent. Hirschsprung’s disease or congenital
intestinal aganglionosis also may also present with bilious vomiting. In this condition, there should also be a history of decreased stool output since birth.

Laboratory tests

Laboratory tests are nonspecific for volvulus. Typically, blood tests will show signs of dehydration and acidosis.

Diagnostic imaging

The classic finding on abdominal plain films is the “double bubble sign,” which shows a paucity of gas (airless abdomen) with two air bubbles, one in the stomach, and one in the duodenum (Fig. 1). Other findings may include air–fluid levels, a paucity of gas distally, or dilated loops over the liver shadow. The plain film also can be entirely normal.

An upper-GI contrast study is considered the gold standard for diagnosing volvulus. The classic finding is that of the small intestine rotated to the right side of the abdomen (an indication of the malrotation), with contrast narrowing at the site of obstruction, causing a “cork-screwing” appearance. Air as a contrast agent has recently gained more acceptance for the diagnosis of high-GI obstructions such as volvulus and low-GI obstructions such as intussusception [87]. Ultrasonography also has been studied for diagnosing volvulus. The ultrasonography may show a distended, fluid-filled duodenum, increased peritoneal fluid, and dilated small bowel loops to the right of the spine [88,89]. Sometimes, spiraling of the small bowel around the superior mesenteric artery also can be observed [90].

Fig. 1. The classic finding of malrotation with midgut volvulus on abdominal plain films is the “double bubble sign,” which shows a paucity of gas (airless abdomen) with two air bubbles, one in the stomach and one in the duodenum.
Management

Because of the risk of bowel necrosis and resulting sepsis, diagnosing this life-threatening condition as early as possible is imperative. Once malrotation with midgut volvulus has been diagnosed, aggressive resuscitation using boluses of normal saline, 20 cc/kg, and the placement of a nasogastric tube should occur. Antibiotics should be administered to cover gram-positive, gram-negative, and anaerobic flora (eg, ampicillin, gentamicin, and clindamycin). Consultation with a pediatric surgeon should not be delayed for diagnostic studies. The sooner the child is admitted to the operating room, the lower the morbidity and mortality of this condition. Some pediatric surgeons will take an ill-appearing neonate with bilious vomiting directly to the operating room without any additional diagnostic tests.

Necrotizing enterocolitis

Causes

Necrotizing enterocolitis (NEC) is seen typically in the neonatal intensive care unit, occurring in premature infants in their first few weeks of life. Occasionally, it is encountered in the term infant, usually within the first 10 days after birth. The cause of NEC is unknown, but a history of an anoxic episode at birth and other neonatal stressors are associated with the diagnosis [91,92].

Pathophysiology

The pathologic finding of NEC is that of a necrotic segment of bowel with gas accumulation in the submucosa. Necrosis can lead to perforation, sepsis, and death. The distal ileum and proximal colon are most commonly involved. Clostridium spp, E coli, Staphylococcus epidermidis, and rotavirus are the pathogens recovered most commonly [72,73].

Presentation

Infants who have NEC will present typically as appearing quite ill, with lethargy, irritability, decreased oral intake, distended abdomen, and bloody stools. Symptoms may present in a range from fairly mild, with only occult-blood positive stools, to a much more critically ill presentation. Because this condition is diagnosed typically in the neonatal intensive care unit, it still must be considered in a term infant who has experienced significant stress, such as anoxia, at birth.

Radiologic studies

The plain abdominal film finding of pneumatosis intestinalis, caused by gas in the intestinal wall, is diagnostic of NEC.
Management

Management includes fluid resuscitation, bowel rest, and broad-spectrum antibiotic coverage. Early surgical consultation is imperative.

Summary

Abdominal pain or gastrointestinal symptoms are common complaints in young children. It is the emergency physician’s duty to understand current recommendations regarding the evaluation and management of more benign conditions such as gastroenteritis and also be able to differentiate a true surgical condition such as appendicitis.

References


