Abdominal surgical emergencies in infants and young children

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Infants and children commonly present to the emergency department (ED) with abdominal and gastrointestinal (GI) symptoms. In most cases these symptoms are caused by a self-limited process such as viral gastroenteritis; however, they might also be the harbingers of life-threatening surgical emergencies. Because symptoms such as vomiting, diarrhea, abdominal pain, and fever are so common and so nonspecific in children, the recognition of surgical emergencies is frequently delayed or missed altogether. When one also considers the difficulties inherent to the pediatric examination, it is not surprising that the diagnoses of intussusception, pyloric stenosis, malrotation with volvulus, and bowel obstruction continue to be among the most elusive diagnoses for the emergency physician (EP). Appendicitis in the infant or young child is especially difficult to detect in its early stages and carries significant morbidity and mortality. Testicular torsion, another surgical emergency, might also present with vague abdominal complaints. This article reviews abdominal surgical emergencies in infants and young children that are often mistaken for more benign, self-limited illnesses.
General approach to the child who has abdominal pain

Important information can often 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 might 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 into tears. Observing the child’s behavior before any interaction might 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 his surroundings. An older child who walks slowly down a corridor in the ED holding his right lower quadrant has similarly given the examiner a great deal of information. When the child is approached, using a nonthreatening manner might pay dividends during the assessment. For example, sitting down or kneeling to bring the examiner closer to the child’s eye level makes the examiner appear 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 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 vice versa?” might be difficult. Parents of young infants might only describe their child as irritable and not realize that the abdomen is the source of pain. Adolescents might be embarrassed to talk about bodily functions or sexual issues, especially with physicians of the opposite sex. It is also important to question adolescents about their medical history separate from their parents because they might 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 might be distracted by a set of car keys. Hand and finger puppets can also 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 might show the child that the examination is nothing to be feared. An older child can also be allowed to place his or her hand on top of the examiner’s and simultaneously apply pressure, and they can also be questioned about school or play activities.
Before touching the 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, which might 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 might be possible to have the mother gently push on different areas of the abdomen, with the examiner merely observing the child’s response. Another technique is to have the mother hold the child over her shoulder with the child facing away. The examiner can then stand behind the child and slip a hand between mother and child to palpate the abdomen. Peritoneal signs can 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 simply be asked to jump up and down.

Rectal examinations are not imperative in a child presenting with abdominal pain. In particular, they have been shown to not be helpful in the diagnosis of appendicitis [6]. Rectal examinations can, however, aid in the diagnosis of a GI bleed, intussusception, rectal abscess, or impaction. If a rectal examination is necessary, it can be done by partially introducing a small finger. Inspection of the genitalia might 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 who have abdominal pain. For example, failure to examine the throat might lead to a missed diagnosis of pharyngitis, which can be associated with abdominal pain. Lower lobe pneumonias can also mimic an abdominal emergency. The general examination 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 turgor, prolonged capillary refill, and decreased urine output. Most of these signs have not been well studied, and some might 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 who have abdominal pain does not appear to increase the risk of misdiagnosis [1]. A better physical examination can often be accomplished when the patient’s pain has been addressed.

Appendicitis

Epidemiology

Appendicitis is the most common surgical cause of abdominal pain in childhood, affecting four of every 1000 children. Appendicitis is the cause of pain in 2.3% of all children seen in ambulatory clinics or EDs with abdominal
pain. Of the children admitted to the hospital with abdominal pain, 82% are diagnosed with appendicitis [2]. Because of the difficulty in evaluating young children who have 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 of appendicitis, consisting of generalized abdominal pain migrating to the right lower quadrant associated with nausea, vomiting, and fever, is seen less often in pediatric patients [3]. In addition, children often present earlier in their clinical course than adults, 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 [4]. If available, a history that abdominal pain preceded vomiting can be helpful in distinguishing appendicitis from acute gastroenteritis. Young children commonly have diarrhea as the presenting symptom [5]. Bearing in mind the special techniques discussed previously for eliciting peritoneal irritation, the EP should also remember that position of the appendix can vary greatly, and tenderness might be found locations other than the classic McBurney’s point. Although the rectal examination is not usually helpful in making the diagnosis of appendicitis [6], some authors advocate rectal examination in infants, in whom there might be a palpable rectal mass in up to 30% of cases [7]. Changes in skin temperature over the area of the appendix have not been shown to be helpful in the diagnosis of appendicitis [8].

Differential diagnosis

Gastroenteritis is the most common diagnosis in cases of missed appendicitis. Although *Yersinia enterocolitica* and *Y. pseudotuberculosis* enteritis have been termed the “great imitators” of appendicitis, the amount of diarrhea in gastroenteritis is usually more pronounced. Appendicitis is also frequently mistaken for a urinary tract infection (UTI), which might also present with abdominal pain and vomiting. A study reported by Reynolds [9] 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 (WBC) count can be helpful in the diagnosis, although by itself it is neither specific nor sensitive for appendicitis and cannot therefore
be used alone to rule in or rule out the disease [10]. WBC count can be used as an adjunct after the clinical suspicion of appendicitis has been estimated. If clinical suspicion is low before any laboratory or other investigations (eg, in a child who has vomiting and diarrhea but minimal abdominal tenderness) and the WBC count is normal, the likelihood of appendicitis is low. If the WBC count is high, the likelihood of appendicitis is sufficiently high to warrant further tests or observation.

A urinalysis (UA) should be performed, but caution must be exercised in its interpretation, remembering that mild pyuria, hematuria, and bacteriuria can be present if an inflamed appendix is located adjacent to a ureter. C-reactive protein has also been studied as a marker for appendicitis [11–14], but it is not significantly more sensitive or specific than WBC count.

**Diagnostic radiology**

Plain film abdominal series typically have nonspecific findings and are of low yield in cases of appendicitis [15]. Appendicoliths are only present 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, but 10% to 30% of normal appendices are not visualized with barium studies, creating a high number of false-positive results [15].

Ultrasound is considered by many clinicians to be the imaging test of choice in children. Ultrasound is noninvasive, rapid, and can be performed at the bedside. It does not require oral contrast, which is an advantage for patients who might require surgery. It also spares the pediatric patient exposure to radiation. The normal appendix in pediatric patients is readily visualized by ultrasound because they usually have less abdominal wall fat than 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 is larger than 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 might be intact or poorly defined, and a fecolith might or might not be present. Periappendiceal fluid collection might indicate an early perforation but might result simply from inflammation. Experienced ultrasonographers can achieve sensitivities of 85% to 90% and specificities of 95% to 100% in acute appendicitis [16–24], but studies have not shown an improvement in outcome measures such as a decrease in negative laparotomies or time to the operating room [25,26]. Color flow Doppler is now being added to increase the accuracy of sonographic examinations. Doppler measurement demonstrates an increase in blood flow to the area of an inflamed appendix [27].

In recent years computed tomography (CT) has become the test of choice for pediatric surgeons when ultrasound fails to give a definitive diagnosis [28]. Everything from triple-contrast (intravenous, oral, and rectal) CT
scanning to noncontrast, unenhanced CT has been used [29,30]. CT offers the advantage of greater accuracy, the ability to identify alternative diagnoses, and (in some studies) lower negative laparotomy rates [31]. Although CT appears to be better than ultrasound for making the diagnosis of appendicitis in children [32], 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 GI tract can be a challenge, and antiemetics might be required.

Leukocyte imaging studies [33] and technetium scans [34] have been used for equivocal cases of abdominal pain in children. Their overall sensitivity, specificity, and accuracy, however, are lower than CT. Magnetic resonance imaging (MRI) is also superior in its ability to diagnose appendicitis in children [35], but it might not be available or practical. No study can be relied upon 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 before any radiologic study is warranted. Nonetheless, many surgeons request a diagnostic study to decrease the likelihood of a negative laparotomy. When the diagnosis of appendicitis has been made, preparing the child for the operating room is essential. The oral intake of these children has usually 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 who have appendicitis.

If there are clinical or radiologic signs of perforation, antibiotics with gram-negative and anaerobic coverage should be started in the ED [36]. A few studies have also shown a benefit to antibiotic therapy in decreasing infectious complications in children who have uncomplicated, nonperforated appendicitis [37]. 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 who is considered to be 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.

**Hypertrophic pyloric stenosis**

**Pathophysiology**

Hypertrophic pyloric stenosis (HPS) is caused by a narrowing of the pyloric canal caused by hypertrophy of the musculature. The etiology of this condition remains unclear, but some experts theorize that HPS is caused by
*Helicobacter pylori*, the same bacterium associated with peptic ulcer disease. This theory is based on nonspecific evidence such as the temporal distribution, seasonality, and familial clustering of HPS along with the pathological finding of leukocytic infiltrates and the increased incidence seen in association with bottle-feeding [38].

**Epidemiology**

HPS occurs in one of every 250 births and has a male predominance (male to female ratio 4:1). The condition also has racial variation; it is observed to be more common in Caucasians than in African-Americans, and it is rare in Asians. Originally, first-born males were thought 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 risk for HPS, with the risk being still higher if the mother is affected [39].

**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, vomiting increases to follow every feed and can become projectile. Comparing birth weight to current weight is a key element in the evaluation of a vomiting neonate. 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 who have HPS will continue to be hungry but because of repeated vomiting they might plateau or even lose weight. An infant who has HPS might also become constipated because of dehydration and decreased intake.

On examination, the neonate who has HPS might appear normal, only hungry, or might have signs of dehydration, which can lead to the appearance of jaundice. Peristaltic waves moving from left to right can 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, might also be detected during physical examination. Decompressing the stomach with a nasogastric tube first and using a lubricant on the fingertips might improve the ability to palpate this “olive.” Clinicians’ ability to palpate the pylorus “olive” has decreased over the years, probably because of the addition of ultrasound in confirming the diagnosis. In 1999 Abbas [40] reported that many infants who have HPS who have palpable masses on examination still undergo one or more unnecessary and redundant tests, which 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 who have volvulus deteriorate rapidly, and the vomiting is bilious, eventually exhibiting signs of sepsis and bowel necrosis. Incarcerated hernias can present similarly, as can intussusception (although it occurs less commonly in neonates).

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 be present before viral gastroenteritis should be considered.

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 afterward. The amount of vomitus is smaller and the neonate continues to gain weight. Infections, especially in the urinary tract, can also present with vomiting as a chief complaint, and 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 $H^+$ and $K^+$ ions. The infant who has HPS will therefore initially demonstrate a hypokalemic, hypochloremic metabolic alkalosis [41]. If the infant remains dehydrated for a long period of time, the alkalosis can 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 who has a clinical picture suggestive of HPS, further studies are warranted. Ultrasound measures the thickness of the pyloric wall (normal < 2.0 mm; HPS > 4.0 mm) and length of the pyloric canal (normal < 10.0 mm; HPS > 14–16 mm) to diagnosis HPS (Fig. 1). Ultrasound has been shown to have a sensitivity and specificity as high as 100% [42,43]. A false-negative result can occur if the ultrasonographer measures through the distal stomach or antrum and not through the pylorus itself. A false-positive result might occur if pyloric spasm is present but not pyloric stenosis.

If ultrasound is nondiagnostic and HPS remains a concern, the next radiologic test of choice is an upper GI series. An upper GI series will reveal the classic “string sign” as contrast flows through narrowed pyloric lumen (Fig. 2). There will also be delayed gastric emptying. As with ultrasound, false-positive results might occur because of pyloric spasm, which also gives
the appearance of a string sign. Endoscopy can also be used to diagnose HPS, but it is not used commonly [44].

**Management**

When the diagnosis of HPS has been made, admission to the hospital is indicated. These infants are often dehydrated, so 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 to decrease the spasm of the pylorus has been used as an alternative to surgery. Atropine is then administered orally for several weeks until the child “outgrows” the stenosis. Surgery has been avoided in many cases [45]; however, surgical treatment 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°, then returns into the abdomen. If the rotation is not correct, the intestine will not be “fixed down” correctly at the mesentery. The vascular mesentery will appear to be more “stalk-like” in its structure and is at risk for later twisting, called volvulus. Volvulus is the twisting of a loop of bowel about its mesenteric base “stalk” attachment. Ischemia subsequently develops, which constitutes a true surgical emergency because bowel necrosis can occur within hours. The entire small bowel is at risk for ischemia and necrosis.

**Epidemiology**

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 the disorder is rarely familial. The exact frequency of midgut malrotation is not known because it is frequently asymptomatic.

Congenital adhesions (Ladd’s bands), which extend from the cecum to the liver, are associated with congenital malrotation, which can cause external compression of the duodenum and obstruction. This condition is not generally considered to be a surgical emergency, but it eventually requires surgical intervention to lyse the bands.

**Presentation**

Volvulus can present is one of three ways: (1) sudden onset of bilious vomiting and abdominal pain in a neonate, (2) history of feeding problems with bilious vomiting that appears like a bowel obstruction, and, less commonly, (3) failure to thrive with severe feeding intolerance [46]. 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 might present pale and grunting. The abdomen might or might not be
distended depending upon location of the volvulus. If the obstruction is proximal, there might be no distension. The abdominal wall might appear blue if the bowel is already ischemic or necrotic. The pain is constant, not intermittent, and the neonate will appear irritable. Jaundice might also be present. Hematochezia is a late sign that indicates intestinal necrosis. Neonates who have volvulus will gradually deteriorate if the bowel remains ischemic.

**Differential diagnosis**

Bilious vomiting in a neonate is considered to be a surgical emergency until proven otherwise; however, in early presentations of volvulus, vomitus can be nonbilious, and a misdiagnosis of acute gastroenteritis might result. As mentioned previously, the diagnosis of acute gastroenteritis should be made 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 might also present with bilious vomiting. It is therefore imperative to examine a vomiting neonate thoroughly for signs of a hernia. A more rare cause of bilious vomiting is duodenal or ileal atresia, although this is typically discovered in the newborn nursery or soon after. With intestinal atresia, the neonate will not be as ill-appearing as with volvulus. Necrotizing enterocolitis can also rarely appear in term neonates. Intestinal hematomas can occur in cases of child abuse.

Congenital adrenal hyperplasia (CAH) can cause bilious vomiting without anatomical obstruction, and it can 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\(^+\), high K\(^+\)). It is more likely that CAH will be seen in male infants 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, might also present with bilious vomiting. In this condition there should 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. Other findings might include
air–fluid levels, a paucity of gas distally, or dilated loops over the liver shadow. Plain film can also be entirely normal.

An upper GI contrast study is considered to be the gold standard for making the diagnosis of volvulus. The classic finding is that of small intestine rotated to the right side of the abdomen (an indication of the malrotation), with contrast narrowing at the site of obstruction causing "corkscrewing" (Fig. 3). Air as a contrast agent has gained more acceptance recently for the diagnosis of high GI obstructions such as volvulus and low GI obstructions such as intussusception [47]. Ultrasound has also been studied for making the diagnosis of volvulus. Ultrasound might show a distended, fluid-filled duodenum, increased peritoneal fluid, and dilated small bowel loops to the right of the spine [48,49]. Sometimes spiraling of the small bowel around the superior mesenteric artery can also be observed [50].

Management

Because of the risk of bowel necrosis and resulting sepsis, diagnosing this life-threatening condition as early as possible is imperative. When the

Fig. 3. Barium follow-through showing midgut volvulus showing typical “corkscrewing” of the displaced duodenum. (From Kealey WD, McCallion WA, Brown S, et al. Midgut volvulus in children. Br J Surg 1996;83(1):105–6; with permission.)
diagnosis has been made, aggressive resuscitation of the neonate using 10 mL/kg boluses as needed of normal saline and placement of a nasogastric tube should occur. Antibiotics should be administered to cover gram-positive, gram-negative, and anaerobic flora (eg, ampicillin, gentamycin, 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 who has bilious vomiting directly to the operating room without any additional diagnostic tests.

**Intussusception**

*Pathophysiology*

Intussusception, which was first described more than 300 years ago, 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, which leads to edema, bleeding of the mucosa, increased pressure in the area, and eventually obstruction to arterial flow. Gangrene and perforation result.

*Epidemiology*

Intussusception is most frequently seen in children between the ages of 3 months and 5 years, with 60% of cases occurring in the first year of life, and a peak incidence at 6 to 11 months of age. This disorder has a male predominance and was once believed to occur more often in the spring and autumn, but recent reports do not support this assertion [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-Schönlein purpura and require a workup to determine the underlying etiology.

*Presentation*

The classic triad of intermittent colicky abdominal pain, vomiting, and bloody mucous stools is encountered only 20% to 40% of the time. At least two of these findings will be present in about 60% of patients. The vomit 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, lasting for approximately 1 to 5 minutes at a time then abating for 5 to 20 minutes. During episodes of pain, the child cries and might 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 can also develop as the child deteriorates.

If the ED staff does not witness a colicky episode, the EP should ask the parents to describe or demonstrate what the child does during the episodes. Most parents of a child who has gastroenteritis do not indicate that their child is in pain. Parents of a child presenting with intussusception usually believe that the child is in pain before or during episodes of vomiting. Intussusception can also 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 might be distended and tender, but usually the pain appears to be out of proportion to the physical examination. There might be an elongated mass in the right upper or lower quadrants. Any type of blood in the stool might be caused by intussusception. Rectal examination might reveal occult blood or frankly bloody foul-smelling stool, classically described as “currant jelly” [55], but 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 might be something else, such as red fruit punch or gelatin. Guaiac testing might prevent this error when there is some question regarding the source of the redness.

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 typically presents 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 is usually painless unless the diverticulum becomes inflamed.

An incarcerated hernia or testicular or ovarian torsion might 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 is generally not seen in young children.

**Laboratory tests**

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

Plain abdominal films are neither sensitive nor specific for intussusception [56,57]. Plain films might initially be normal. As the disease progresses, a variety of abnormalities can 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 might take the shape of a crescent, although other patterns might be seen.

Ultrasound is used in some institutions for the diagnosis of 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 can be used to identify bowel ischemia. If signs of intussusception are not identified by ultrasound in patients in whom the diagnosis is suspected clinically, proceeding with a barium or air enema should be still be considered.

Management

The main focus in management of a child who has intussusception is emergent reduction of the obstructed bowel. Classically, this is accomplished by a barium enema, which acts as a diagnostic and therapeutic radiologic study (Fig. 4). The barium enema has been the gold standard for diagnosis and treatment of intussusception for decades [59]. Saline enemas have also been employed successfully [60,61], and newer modalities such as air enemas and ultrasound-guided enemas have emerged.

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

Visualization of the entire colon to the terminal ileum is mandatory to exclude ileocolic intussusception. Ileo–ileo intussusception can be much
harder to diagnose and to reduce. Spontaneous reduction of intussuscepted bowel has been reported, although therapeutic intervention should not be delayed in a patient who has significant symptoms [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 h), evidence of obstruction (eg, air–fluid levels on plain abdominal films), and ultrasound findings of intestinal ischemia or trapped fluid.

Even in well-selected patients, enemas can cause 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 can 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, and malrotation with midgut volvulus and tumors should be considered as possible etiologies. In addition to inguinal hernias, umbilical, obturator, and femoral canal hernias can also lead to small bowel obstruction [56].

Fig. 4. Barium enema in ileocolic intussusception. The intussusceptum is visible in the ascending colon (arrows). (From Lazar J. Greenfield surgery: scientific principles and practice. Baltimore (MD): Lippincott Williams & Wilkins; 2001; with permission.)
Presentation

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

Differential diagnosis

Abdominal pain and vomiting can also be seen with other processes such as appendicitis. As time passes, a bowel obstruction will develop more abdominal distension than is typically seen 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 might be as important to recognize as the obstruction itself.

Laboratory tests

No laboratory test is diagnostic of a bowel obstruction. An elevated blood urea nitrogen, creatinine, and hematocrit might 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 who have bowel obstruction. Distended loops of bowel might be seen; however, smooth bowel walls are more common than distended bowel in small children. Multiple air–fluid levels are also commonly seen with small bowel obstruction. In later presentations, the bowel might resemble a tangle of hoses or sausages. An upright or lateral decubitus film will help to determine if free air caused by perforation is present. Further study with ultrasound, CT, upper GI series, or 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.

**Necrotizing enterocolitis**

*Epidemiology*

Necrotizing enterocolitis (NEC) is typically seen in the neonatal intensive care unit (NICU), 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 not known, but history of an anoxic episode at birth and other neonatal stressors are associated with the diagnosis [72,73].

*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, *Escherichia coli*, *Staphylococcus epidermidis*, and *Rotavirus* are the most commonly recovered pathogens [72,73].

*Presentation*

Infants who have NEC typically present appearing quite ill, with lethargy, irritability, decreased oral intake, distended abdomen, and bloody stools. Symptoms might present fairly mildly, with only occult blood-positive stools, to a much more critically ill presentation. Although this diagnosis is typically made in the NICU, it nonetheless must be considered in a term infant who experienced significant stress at birth (eg, anoxia).

*Imaging 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.

**Incarcerated hernia**

*Epidemiology*

Inguinal hernias occur in 1% to 3% of all children, more often in males (6:1), and more often on the right side (2:1). Premature infants are at higher
risk for inguinal hernias, with an overall incidence of 3% to 5%. In addition, more than two thirds of incarcerated inguinal hernias requiring operative reduction occur during the first year of life [74]. Umbilical hernias are also more common in premature infants, but unlike inguinal hernias umbilical hernias rarely become incarcerated and usually close spontaneously by 2 to 3 years of age [74,75].

Other conditions that place children at increased risk for abdominal hernias include connective tissue diseases (eg, Marfan’s syndrome, Ehlers-Danlos syndrome), cystic fibrosis, and metabolic disorders (eg, mucopolysaccharidoses). Patients who have ventriculoperitoneal shunts or patients who are receiving peritoneal dialysis are also at increased risk [75,76].

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 can become bilious and sometimes feculent.

Inguinal hernias can be palpated as smooth, firm, sausage-shaped, mildly tender masses in the groin; the hernia originates proximal to 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 is well-appearing without vomiting, fever, or redness to the inguinal area, the hernia is not likely incarcerated.

Differential diagnosis

Many conditions can mimic an inguinal hernia, but the most common is a hydrocele. A hydrocele is a result of incomplete obliteration of the process vaginalis, which allows an outpocketing of peritoneum to appear in the scrotum. This fluid-filled sac can appear anywhere from the spermatic cord to the testis, and if large it can be transilluminated. Hydroceles can be palpated separate 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 ultrasound can differentiate an inguinal hernia from a hydrocele.

Management

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

**Meckel’s diverticulum**

*Pathophysiology and epidemiology*

Meckel’s diverticulum is the most common congenital abnormality of the small intestine. A Meckel’s diverticulum is a remnant of the omphalomesenteric (vitelline) duct, which normally disappears by the seventh week of gestation. It is a true diverticulum, containing all layers of the bowel wall. Up to 60% of these diverticulae contain heterotopic gastric tissue, and heterotopic pancreatic, endometrial, and duodenal mucosa have also been reported [77,78]. The features of Meckel’s diverticulum are commonly described by “the rule of 2s” [79]: 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 [80]. 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 a Meckel’s diverticulum is that of painless or minimally painful rectal bleeding. Isolated red rectal bleeding is common, particularly in boys less than 5 years of age [81]. Such painless bleeding is a result of heterotopic gastric tissue in the diverticulum or in the adjacent ileum. Abdominal pain, distension, and vomiting can occur if obstruction has occurred, and the presentation can mimic appendicitis or diverticulitis. A Meckel’s diverticulum can also ulcerate and perforate, presenting as a bowel perforation or acting as a lead point, resulting in intussusception.

*Differential diagnosis*

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

*Laboratory tests*

Although no laboratory test is diagnostic for a Meckel’s diverticulum, children who have GI bleeding should undergo screening laboratory tests such as a complete blood count, coagulation profile, and a type and screen.
Diagnostic radiology

Abdominal films might show signs of obstruction such as dilated loops of bowel or a paucity of bowel gas. A Meckel’s scan involves injection of Technetium–pertechnetate intravenously. This test relies on the presence of gastric mucosa in or near the diverticulum, which has an affinity for the radionucleotide. A Meckel’s scan can detect the presence of gastric mucosa within the diverticulae with up to 85% accuracy [82]. Mesenteric arteriography can detect the site of active bleeding if bleeding is profuse.

Management

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

Ovarian and testicular torsion

Epidemiology

While ovarian torsion in the ED is rare, a missed diagnosis can result in significant patient morbidity and medicolegal consequences. Both conditions are more common in preadolescent and adolescent patients, but they can occur in all age groups, even in utero.

Torsion of the testes or spermatic cord is more common than ovarian torsion, occurring in one of 4000 males, with a peak incidence at age 13 [83]. Delay in diagnosis and treatment can result in a loss of spermatogenesis or necrotic, gangrenous testes. Testicular salvage rates are time-dependent, with a 96% success rate if detorsion is performed within 4 hours of symptom onset decreasing to less than 10% at 24 hours.

Pathophysiology

Older girls can have an ovarian torsion secondary to a corpus luteal or follicular cyst, whereas younger children might have no underlying ovarian abnormality or a mature cystic teratoma [84]. Bilateral ovarian torsion has been reported [85].

The testes enter the scrotum through the inguinal canal after descent from the abdomen. The peritoneum invaginates through the canal and partially covers the testis and epididymis, forming the tunica vaginalis. Typically, the tunica vaginalis attaches to the posterior wall of the
hemiscrotum and the superior pole of the testes to achieve testicular fixation. If the tunica completely covers the testis and attaches higher up on the spermatic cord (bell clapper deformity), proper testicular fixation does not occur and there is a predisposition to torsion. In intravaginal torsion, the testis can rotate within the tunica vaginalis and thereby constrict arterial blood flow. Extravaginal torsion, seen more commonly in premature neonates, can also occur antenatally. If torsion is prolonged, testicular infarction and atrophy has been reported after 4 to 6 hours of ischemia.

Clinical presentation

Ovarian torsion typically presents with an acute onset of pain on the side of the torsion; however, pain can radiate to the flank, back, or groin. Associated symptoms might include nausea, vomiting, constipation, urinary tract symptoms, and fever. Pelvic examination in older girls will reveal adnexal tenderness with a mass. Cervical motion tenderness might also be present. In younger children, when a pelvic examination is not possible, ultrasound will be diagnostic.

Testicular torsion can present as an acutely tender and swollen hemiscrotum, but it might present more subtly, especially in younger boys, underscoring the importance of the genital examination in the evaluation of pediatric abdominal pain. Although in younger boys the only presenting symptoms might be abdominal pain, vomiting, and a low-grade fever, examination will reveal scrotal tenderness and swelling with an elevated testis. The loss of the cremasteric reflex is another important sign; it was notably absent in 100% of the patients who had torsion in one series [86]. Torsion should also be considered in boys who have an undescended testis.

Differential diagnosis

Other disease processes that can present with sudden abdominal pain and vomiting include intussusception and renal colic. In girls, ectopic pregnancy and ruptured corpus luteal cyst or tubo-ovarian abscess must also be considered.

Laboratory tests

Leukocytosis is present in up to 50% of patients, and UA is typically normal.

Diagnostic radiology

Diagnostic studies might be appropriate in certain cases, but they should never delay surgical consultation, especially in patients who present with less than 12 hours of symptoms. A suggestive history and physical examination are all that are necessary to prompt emergent surgical exploration.
Color-flow Doppler ultrasound has a sensitivity of 82% to 86% with a specificity of almost 100% for testicular torsion. Sensitivity and specificity are lower for ovarian torsion. Scintigraphy for testicular torsion has a sensitivity ranging from 80% to 100% and a specificity of 89% to 100% [87]. Decreased blood flow to the affected gonad is considered to be diagnostic of torsion.

Because torsion can be intermittent, diagnostic tests might be negative at the time of examination. If suspicion for the diagnosis still exists, consultation with a urologist or gynecologist and admission to the hospital is recommended.

Management

If ovarian torsion is suspected or confirmed on ultrasound with Doppler flow studies [88], laparoscopy should be performed to attempt ovarian salvage. Unfortunately, ovarian salvage is rare, even when the interval between diagnosis and operative intervention is short [89], which might be caused by a delay in the initial diagnosis. Symptom onset has varied from 6 hours to 3 weeks before ultimate diagnosis [90,91] Although most torsed ovaries are necrotic on exploration, there have been reports of ovarian salvage after ovarian detorsion and oophoropexy [92].

If a male patient presents within 12 hours of symptoms, immediate surgical exploration is indicated. While awaiting surgery, an attempt at manual detorsion in the ED is appropriate, which is accomplished by rotating the testis in an “open-book” fashion from medial to lateral after administration of adequate analgesia. Orchidopexy of both testes is usually performed after detorsion to avoid recurrence; approximately 40% of patients have a bellclapper deformity of the contralateral testis.

Summary

Surgical emergencies can be missed easily in children, who are not always able to volunteer relevant information. Awareness of the entities discussed in this review might help the EP uncover subtle clues to early diagnoses that might not be initially apparent.

Ill-appearing children who have abdominal pain and vomiting should be considered to have ischemic or necrotic bowel until proven otherwise. Possible diagnoses include volvulus, intussusception, and necrotizing enterocolitis. Bilious vomiting, especially in a young infant, should be considered to be an indication of a high bowel obstruction such as midgut volvulus, which warrants immediate surgical consultation. Significant rectal bleeding with abdominal pain can result from intussusception, volvulus, or an inflamed Meckel’s diverticulum. Rectal bleeding with unstable vital signs can result from an upper GI bleed (eg, peptic ulcer disease). Painless rectal bleeding can result from a Meckel’s diverticulum, polyps, arteriovenous
malformation, or a tumor. Examination of the genitalia is imperative, especially in boys, to exclude the possibility of an incarcerated hernia or testicular torsion.

References


