Pediatric Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Volume 16, Number 10 / October 2011 www.ahcmedia.com

Authors:

Casey Grover, MD, Stanford/Kaiser Emergency Medicine Residency, Stanford, CA.

Ewen Wang, MD, Associate Professor of Surgery/Emergency Medicine, Associate Director, Pediatric Emergency Medicine, Stanford School of Medicine, Stanford, CA.

Peer Reviewer:

Nick Erbrich, MD, FAAP, Department of Emergency Medicine, St. Christopher's Hospital for Children, Philadelphia, PA.

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Dietrich (editor), Dr. Skrainka (CME question reviewer), Dr. Wang (author), Dr. Grover (author), Dr. Erbrich (peer reviewer), Ms. Mark (executive editor), and Ms. Hamlin (managing editor) report no relationships with companies related to the field of study covered by this CME activity.

AHC Media

Abdominal Pain and Vomiting in the Infant

Abdominal pain with vomiting is a common presenting complaint to the emergency department (ED) in infants. In the majority of cases, the patient's symptoms come from a benign cause such as gastroenteritis. However, it remains essential for the emergency physician (EP) to thoroughly understand the differential diagnosis of abdominal pain and vomiting in infants, particularly identifying those causes requiring surgical intervention.[1-3] This article will discuss seven key causes of abdominal pain and vomiting in infants: malrotation with volvulus, necrotizing enterocolitis, Hirschsprung's disease, pyloric stenosis, intussusception, appendicitis, and acute gastroenteritis. A summary of the presenting age and frequency of each condition is displayed in Table 1 for quick reference.

—The Editor

Introduction

A review of gastrointestinal (GI) tract development is essential to understand the pathophysiology of acute abdominal processes occurring in the infant. At approximately the fourth to fifth week of development, the midgut begins to develop, extending from the anterior intestinal portal to the yolk sac. As the gut develops, it initially extrudes into the umbilical cord between weeks five and 10. The bowel then rotates counter-clockwise through a path of 270 degrees around the superior mesenteric artery, ultimately attaching to the posterior abdominal wall at about the fourth to fifth month of development. The ascending and descending colon normally attach and anchor to the right and left abdominal gutters, respectively. It is during this period that failure of the normal process of rotation and fixation of the intestine can lead to malrotation and predispose to volvulus in infancy. In such cases, the colon develops abnormal attachments to the right side of the retroperitoneum, commonly referred to as Ladd's bands, which may cause duodenal obstruction and also allow for the bowel to twist upon its mesentery.[5-7] Furthermore, during the first trimester of development, neural crest cells migrate distally from the central neurologic structures toward the anal end of the intestinal tract. Enteric nervous system precursor cells ultimately develop from these neural crest cells, allowing for the development of the enteric nervous system. Failure of this migration of neural crest cells can cause Hirschsprung's disease in infancy.[8]

Malrotation with Volvulus

Etiology. Malrotation represents any failure during development of the normal rotation pattern, leading to a congenitally abnormal position of the bowel within the abdomen. Subsequently, the poorly attached intestine is at an increased risk of twisting, producing a volvulus, which can lead to obstruction. It is important to recognize that malrotation itself is not a condition requiring emergent surgical management; it is only when patients develop acute intestinal obstruction from volvulus that emergency surgery is needed.[3-7,9]

Executive Summary

- Bilious vomiting is a hallmark feature of malrotation with volvulus.
- NEC is a disease in neonates associated with prematurity and hypoxia, occurring in up to 12% of infants weighing less than 1500 grams.
- The cardinal presenting symptom in Hirschsprung's disease is delayed passage of meconium.
- Lethargy is a common presentation of intussusception.

Malrotation can occur as an isolated phenomenon; however, it is also associated with a number of congenital syndromes, such as Cornelia de Lange syndrome, cat-eye syndrome, or Marfan syndrome. This condition also frequently occurs with other gastrointestinal abnormalities, such as duodenal stenosis or atresia, Hirschsprung's disease, omphalocele, and gastroschesis.[9]

Epidemiology. Malrotation occurs in approximately one in every 500 births, with one in every 5000 births developing volvulus and becoming symptomatic. While infants may present any time, approximately 80% of cases of malrotation with volvulus occur within the first month of life. Of infants who develop this condition within the first month of life, the majority will present within the first week of life.[7]

Presentation. The classic presentation is one of a sudden onset of vomiting, apparent abdominal pain, and difficulty feeding. As a result of the intestinal obstruction, infants develop bilious vomiting, which is a hallmark feature of malrotation with volvulus. Other symptoms that may be seen include diarrhea, constipation, and hematochezia. In infants with advanced disease, signs and symptoms of peritonitis may be present, as well as hemodynamic compromise.[3,7]

Laboratory Studies and Diagnostic Imaging. Routine laboratory studies are often sent, such as a complete blood count, basic metabolic panel, and type and screen. However, these tests are not helpful in making the diagnosis of malrotation with volvulus. Routine abdominal radiographs also may not be helpful. In confirmed cases of malrotation with volvulus, plain abdominal X-rays may demonstrate a variety of findings, from obstruction to a normal bowel gas pattern.[3] Findings on radiograph that increase concern for malrotation are those that suggest an abnormal anatomical location of the bowel, such as the "double bubble" sign when duodenal dilation is present.[7]

The upper GI series is the imaging reference standard for the diagnosis of malrotation with or without volvulus, with sensitivities as high as 93-100%. Malrotation with volvulus is suspected when there are signs of proximal intestinal obstruction — such as an abrupt end to the contrast material within the bowel — and an abnormal location of the duodenal junction. At the point of obstruction, one may see a "beaked" or "corkscrew" appearance. Other signs indicating proximal intestinal obstruction may include a Z-shaped configuration of the duodenum secondary to obstructing peritoneal bands.[3,7,9] (*See Figure 1.*)

In the event that the upper GI series is inconclusive, other imaging studies may be used. If an ultrasound is performed, special attention should be paid to the relationship between the superior mesenteric vein and mesenteric artery. Normally, the vein is to the right of the artery. In malrotation, the vein is to the left of the artery or rotates around the artery as a result of the bowel twisting on its axis. Additionally, a volvulus may appear as a "whirled" appearance of vasculature entering and within the volvulus, referred to as the "whirlpool" sign. Computed tomography may be

Table 1. Presenting Age and Frequency of Conditions Causing Abdominal Pain and Vomiting in Infants

Condition	Presenting Age	Frequency
Pyloric stenosis	3rd - 8th week of life	2-4 per 1000 births
Intussusception	3rd month - 5th year of life (most common in 1st year of life)	18-56 per 100,000 births
Necrotizing enterocolitis	1st - 10th day of life	12% of babies under 1500 g 10% of all cases in term
Malrotation with volvulus	Any age 80% within first month of life	Malrotation: 1 per 500 births; malrotation with volvulus: 1 per 5000 births
Gastroenteritis	Any age	3.7 million physician visits in the United States annually
Hirschsprung disease	First days of life	1 per 5000 births
Acute appendicitis	Any age; < 5% younger than 5 years, most frequent in 2nd decade of life	86 per 100,000 patients per year

used in challenging cases and can be helpful in evaluating malposition of the bowel as well as the relationship between the superior mesenteric vein and arteries.[3,7]

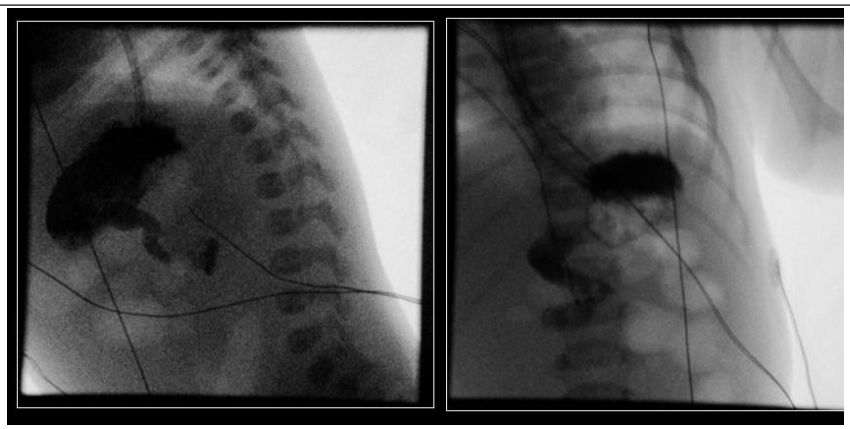
Treatment. Malrotation with volvulus is a surgical emergency. When the diagnosis is either suspected based on a history of bilious vomiting or confirmed via imaging studies, infants should be aggressively resuscitated. Pediatric surgical consultation should be obtained emergently, even prior to definitive imaging, since volvulus can result in necrotic bowel within hours. Intra-operatively, the volvulus is untwisted, and any necrotic sections of bowel are resected. Furthermore, any obstructing bands within the abdomen are lysed, and the bowel is fixed in an appropriate location. As the appendix is commonly in an abnormal position in patients with malrotation, appendectomy is frequently performed during this procedure as well.[5,10]

Necrotizing Enterocolitis

Etiology. While the exact predisposing cause of necrotizing enterocolitis (NEC) is not known, this condition is characterized by an inflammatory process of the neonatal bowel wall that often involves necrosis. In term infants, NEC is nearly always associated with a precipitating event, such as asphyxia, congenital heart disease, abdominal wall defects, neural tube defects, polycythemia, and anoxia, to name a few. This condition is characterized by a necrotic section of intestine, leading to accumulation of gas in the submucosa of the bowel. NEC may present at any point within the neonatal gastrointestinal system, but is most commonly seen involving the jejunum, ileum, and cecum, leading to a presentation of disease in the right lower quadrant.[1,11-13]

The marked intestinal inflammation leads to disruption of the function of intracellular tight junctions in the intestinal epithelium. As a result, infants with this condition are at high risk for third-spacing fluid in the abdomen and intra-abdominal sepsis.[13]

Figure 1. Malrotation with Volvulus



Two images from an upper GI study demonstrating lack of transit of contrast from the stomach and duodenal bulb into the proximal small bowel with corkscrew configuration of the proximal duodenum consistent with malrotation with volvulus. Images courtesy of Casey Grover, MD.

Epidemiology. NEC is a disease in neonates associated with prematurity and hypoxia, occurring in up to 12% of infants weighing less than 1500 grams. This condition is much less common in term infants, with only 10% of cases being in babies born at term. NEC is the most common gastrointestinal emergency in premature and newborn infants, and while it is largely a disease seen and managed in the neonatal intensive care unit (NICU). The fact that it can occur in term infants makes this an important condition for emergency physicians to understand. When seen in term infants, it most commonly presents within the first 10 days of life, but in rare cases can present in infants up to several months of age.[1,11-13]

Presentation. The first signs of NEC are nonspecific and, thus, subtle, and include poor feeding tolerance and abdominal distension. As the disease progresses, the abdomen may become tender to palpation, suggestive of peritonitis. As a result of the inflammation of the gastrointestinal tract, motility is decreased, leading to vomiting, which may be bilious in severe cases. Stools, while normal in the majority of cases, may demonstrate occult blood in early cases and frank blood in more advanced cases.[1,11,12]

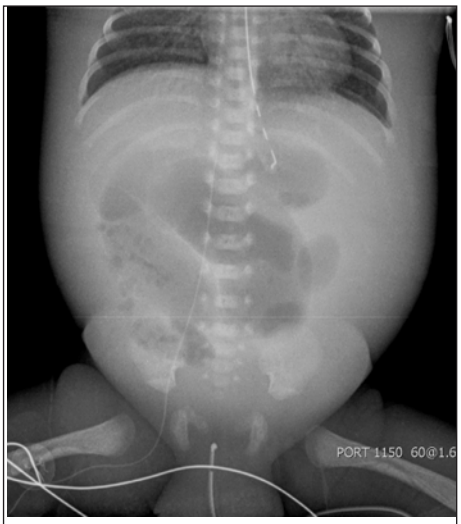
Laboratory Studies and Diagnostic Imaging. Laboratory findings that are more common in NEC include elevated C-reactive protein, thrombocytopenia, elevated or decreased white blood cell count (WBC), and acidosis.[14] However, none of these tests are of high enough sensitivity or specificity to confirm the diagnosis.

Radiologic studies are the best modality for making the diagnosis. Plain X-ray films of the abdomen are the first and best imaging study, with dilated bowel loops suggestive of this condition, and pneumatosis intestinalis (air in the bowel wall) pathognomonic for NEC. (*See Figure 2.*) In severe cases in which there has been perforation, pneumoperitoneum may be present.

Ultrasonography has limited utility in necrotizing enterocolitis. Ascites and hyperemia of the bowel are most suggestive of this condition, but may be non-specific findings. In advanced cases, ultrasound may demonstrate intra-abdominal fluid with debris consistent with perforation.[15]

Treatment. Necrotizing enterocolitis requires aggressive treatment, including fluid resuscitation and bowel rest. Enteral feeds should be stopped, and gastric decompression with a nasogastric or orogastric tube

Figure 2. Necrotizing Enterocolitis



Plain film of the abdomen demonstrating intramural air (pneumatosis intestinalis) in the right lower abdomen, consistent with necrotizing enterocolitis. Image courtesy of Casey Grover, MD.

should be performed. As injury to the bowel lumen predisposes the patient to bacterial translocation across the injured bowel wall with subsequent infection, broad-spectrum antibiotics should be initiated. In infants who deteriorate with medical management, surgical intervention is needed. Specific signs that indicate the need for surgical management include intestinal gangrene, clinical deterioration, abdominal wall edema or erythema, worsening pneumatosis intestinalis or portal venous gas, or worsening acidosis.[1,11,12]

Hirschsprung's Disease/ Intestinal Aganglionosis

Etiology. Hirschsprung's disease (HD) is characterized by a lack of normal migration of ganglion cells in the myenteric and submucosal plexus of the intestine. As a result of this deficit of intestinal neurons, colonic motility is markedly impaired, leading to obstruction. In approximately 75% of cases, the extent of neural crest cells is near normal, and only the rectum is affected. While HD is generally confined to the colon, in rare cases the small bowel and even the entire gut may be affected.[8,16]

Down syndrome increases the risk of Hirschsprung's disease by 100-fold as compared to the general population.[17] Variability exists in the length of gut affected in this condition, with total colonic aganglionosis (TCA) being the most severe form. TCA is estimated to occur in 5% to 15% of all cases of Hirschsprung's disease.[18]

Epidemiology. HD is the most common congenital disorder of intestinal motility, and is estimated to occur in one out of every 5,000 births. Approximately 80-90% present in the neonatal period. This discussion will only focus on presentation in neonates. Hirschsprung's disease is more common in male infants, and appears to have differential incidence based on ethnic heritage, occurring less frequently in Hispanic and white patients and more frequently in African-American and Asian patients.[17]

Presentation. Initial symptoms may include constipation, abdominal distention, and vomiting. The cardinal presenting symptom in HD is delayed passage of meconium. Nearly all normal infants will pass stool within the first 24 hours of life. By comparison, more than 90% of patients with HD will fail to pass meconium within the first 24 hours of life. A rectal examination may reveal a tight anus and may also allow for the passage of meconium in an infant with HD.[8,16,19]

Between 5% and 44% of patients may develop Hirschsprung-associated enterocolitis, which is characterized by foul-smelling diarrhea, abdominal distension, and fever. This condition should be recognized quickly, as in severe cases, infants may develop toxic megacolon. Signs of toxic megacolon include marked abdominal distension, bile-stained vomit, fever, and shock.[8,19]

Laboratory Studies and Diagnostic Imaging. As with many of the conditions producing abdominal pain in infants, basic laboratory studies are not specific for making the diagnosis. Plain abdominal radiographs can be obtained initially and may demonstrate gaseous colonic dilation with an undilated rectum. Contrast enema also may be used in the initial work-up, predominantly to differentiate HD from other causes of lower intestinal obstruction. Findings on contrast enema that are suggestive of this disease include an abrupt change in caliber of bowel size from the normal-sized aganglionic segment to the dilated colon proximal to the aganglionic segment. Contrast enema is estimated to have a 76% sensitivity and 97% specificity for the diagnosis of HD.[8,19]

A definitive diagnosis of Hirschsprung's disease requires more specific testing. Anorectal manometry allows for the measurement of pressures of the anorectal region and, thus, of the functioning of the sphincters of the intestine. Furthermore, this test can be used to look for the presence of the rectoanal inhibitory reflex (RAIR), a finding that is absent in infants with HD.[8,19]

Finally, histopathologic analysis of rectal tissue to evaluate for presence or absence of intestinal ganglionic cells remains the gold standard in the diagnosis of Hirschsprung's disease.[8,19]

Treatment. In patients with Hirschsprung-associated enterocolitis, infants should receive immediate fluid resuscitation and antibiotics should be started. A pediatric surgeon should be emergently consulted, and the intestine should be decompressed.

Treatment of Hirschsprung's disease is surgical. Initially, a diverting ostomy is done to bypass the aganglionic segment causing obstruction. A second operation is performed later, in which the affected bowel is removed with anastomosis of functional bowel to the anus.[8,19]

Pyloric Stenosis

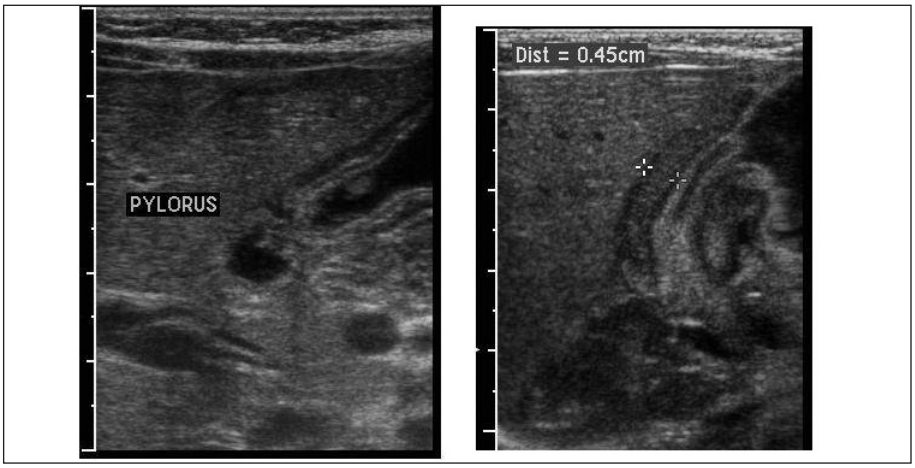
Etiology. This condition results from hypertrophy of the pyloric muscle at the distal end of the stomach. The muscular enlargement causes narrowing of the pyloric channel, which leads to gastric outlet

obstruction. While the exact etiology of this disease has not been elucidated, there are several factors that have been implicated in the development of pyloric stenosis. First, there appears to be a genetic predisposition for this condition. While pyloric stenosis has been associated with specific genetic syndromes such as Cornelia de Lange syndrome, no specific gene has been isolated. One extrinsic factor that has been implicated in the development of pyloric stenosis is exposure to erythromycin. Infants exposed to erythromycin in the first two weeks of life carry up to a 10-fold higher risk of pyloric stenosis, and there is some evidence that maternal exposure to erythromycin during pregnancy and breastfeeding may also increase the risk of this condition.[20-23]

Epidemiology. Pyloric stenosis is a relatively rare condition, with approximately 2-4 cases per 1000 births. It is more common in males, with a four- to five-fold greater risk of developing pyloric stenosis in males relative to females. Furthermore, this condition is more common in first-born children as compared to later-born children. There is also an increased risk in infants with a family history of this disease.[20,21]

Presentation. Pyloric stenosis typically presents between the third and eighth week of life with projectile vomiting. Vomiting tends to occur within 30 minutes of feeding and is non-bilious. The physical examination typically demonstrates a child who appears hungry and eager to feed, even after an episode of vomiting. The abdominal examination is classically described as having a palpable "olive" in the mid-epigastric region of the abdomen, representing the hypertrophied muscle. However, this may be a difficult finding to appreciate in an active and awake infant. In the early stages of disease, infants may appear well; however, as the disease progresses and patients are unable to maintain hydration secondary to vomiting, infants will develop moderate to severe dehydration.[3,20]

Figure 3. Pyloric Stenosis



Laboratory Studies and Diagnostic Imaging. Routine laboratory studies, such as a complete blood count and basic metabolic panel, are not commonly helpful. However, they may demonstrate hypokalemic, hypochloremic, or metabolic alkalosis from vomiting.[3,24]

The diagnosis of pyloric stenosis may be made by one of two different diagnostic imaging studies. First, an upper GI contrast study may be used to make the diagnosis. A positive study will demonstrate a narrowed pyloric channel, often referred to as the "string sign." Advantages to this modality are that it is relatively operator-independent and it has a sensitivity of 96% and a specificity of 100% in the hands of an experienced radiologist. The disadvantages of this modality are that it is relatively invasive and exposes the child to ionizing radiation.[1,25] Second, an abdominal ultrasound may be used to make the diagnosis of pyloric stenosis, with a positive study demonstrating pyloric muscle thickness of greater than or equal to 4 mm or length of the pyloric canal being greater than or equal to 14 mm. (*See Figure 3.*) This modality boasts a sensitivity of 98% and a specificity of 100%, with no radiation exposure to the child. The disadvantage of this study is that, as with all ultrasound, it is somewhat operator-dependent. Generally speaking, due to the less invasive nature of ultrasound and the lack of radiation exposure, ultrasound is the first-line imaging study in the evaluation of the infant with possible pyloric stenosis.[1,3,25]

Treatment. Infants with pyloric stenosis should receive fluid resuscitation intravenously to correct dehydration and provide maintenance fluid needs. Affected patients should be made NPO, and pediatric surgical consultation should be obtained. Definitive treatment consists of surgical pyloromyotomy. This procedure is well tolerated and is associated with a low incidence of morbidity and mortality.[21]

Intussusception

Etiology. Intussusception is the result of prolapse of one part of the intestine into the lumen of an adjacent portion. While the vast majority of cases in children are idiopathic, certain conditions may act as a "lead point" for intussusception. Such conditions include lymphoma, Meckel's diverticulum, and Henoch-Schonlein purpura. When one portion of the intestine prolapses into another, this results in compression of the lumen with subsequent obstruction of venous return. This, in turn, leads to edema, increased pressure on the bowel, and, ultimately, obstruction to arterial flow — with gangrene and perforation of the bowel as manifestations of end-stage disease.[1,26]

Epidemiology. Approximately 18-56 cases of intussusception

Figure 4. Intussusception

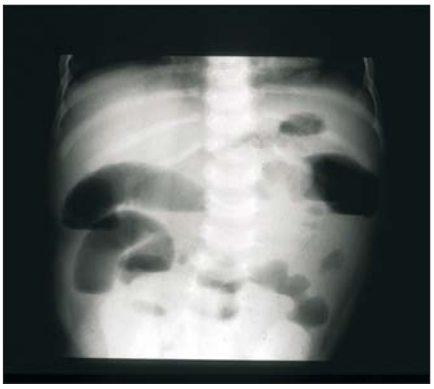
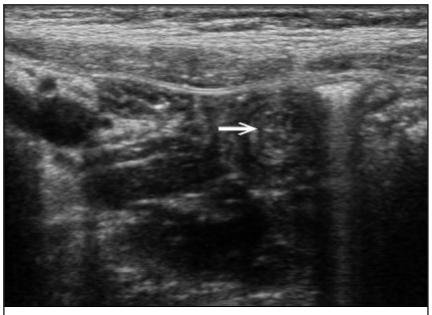


Figure 5. Small Bowel Intussusception

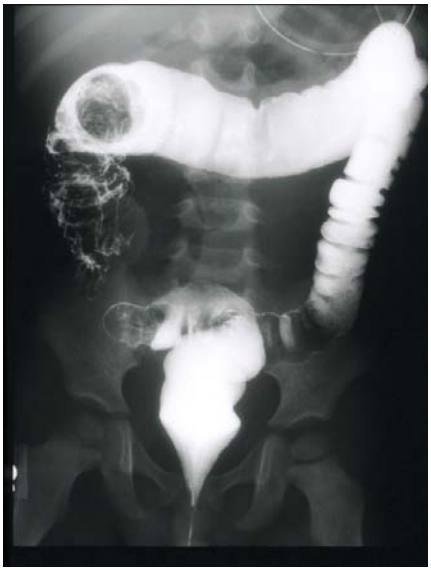


Ultrasound of the abdomen demonstrating a target sign (white arrow) consistent with a small bowel intussusception. Image courtesy of Casey Grover, MD.

occur per 100,000 births. While it may be seen any time between the ages of 3 months and 5 years, it is most common in the first year of life. Peak incidence is between 6 and 11 months of age, with a male predominance.[1,24]

Presentation. The clinical presentation of intussusception may be variable. Common symptoms include irritability and intermittent crying. Features seen in more advanced cases include bloody stool, often referred to as "currant jelly stool." Parents may describe the pain as intermittent, in which the child draws his or her knees up to the chest for up to five minutes at a time, only to be followed by an asymptomatic period of up to 20 minutes. Lethargy remains another common presentation of intussusception.

Figure 6. Intussusception



On physical examination, the child may appear uncomfortable or lethargic. The abdomen may appear distended and tender, and one may appreciate a mass on the right side of the abdomen, most commonly in the right upper quadrant. A rectal examination classically demonstrates grossly bloody stool, which usually is a late finding.[1,26] Early on, there may be a normal-appearing stool that may be positive for occult blood.

Laboratory Studies and Diagnostic Imaging. There are no laboratory studies that are specific for the diagnosis of intussusception.[1]

Imaging studies are essential in the diagnosis of intussusception. Plain abdominal radiography is often the initial imaging study and may demonstrate a mass in the right upper quadrant, reduced air in the small intestine, or small bowel obstruction. (*See Figure 4.*) Studies of the accuracy of plain abdominal radiography in the diagnosis of intussusception have demonstrated high sensitivity but only moderate specificity.[27,28]

Ultrasonography has proven to be an outstanding imaging modality for the evaluation of this condition. A technique of graded compression is used, yielding a sensitivity of 98-100% and a specificity of more than 90%. A positive examination demonstrates an intussusception mass, usually a complex structure greater than 5 cm with a hypoechoic region surrounding an echogenic center — often described as resembling a target or doughnut. (*See Figure 5.*) When viewed on ultrasound in long axis, the hypoechoic layers on each side of the echogenic center may resemble a kidney, creating an appearance often referred to as the "pseudokidney" sign. In addition to the increased sensitivity and specificity of ultrasound over plain abdominal radiography, ultrasound may also be able to identify the lead point of the intussusception.[28]

Treatment. To prevent ischemia, necrosis, and subsequent perforation of intestine, the intussusception must be reduced. Barium, water-soluble contrast, and air have all been used as enemas for the reduction of intussusception. While the barium enema has previously been the gold standard for the diagnosis and treatment of this condition (*see Figure 6*), air enemas have become the more commonly used technique, with a higher rate of successful reduction. Air enemas are preferred because they eliminate the possibility of barium peritonitis in the case of colonic perforation during reduction. In addition, they impart a lower radiation dose to the child. Recurrence of intussusception after air reduction may occur in 5-20% of cases. As such, all children with intussusception must be admitted to the hospital so that additional efforts at enema reduction or surgical intervention may be made if the condition recurs.[1,26,29]

The most feared complication of enema reduction of intussusception is intestinal perforation, which may occur in up to 1% of all cases of attempted reduction with enema. For this reason, all cases of intussusception ideally should be evaluated by a pediatric surgeon prior to enema reduction, so as to facilitate swift operative intervention should a perforation occur.[1,26]

Acute Appendicitis

Etiology. Appendicitis may be described as a disease process that occurs in five stages, usually progressing over a 24-36 hour period. The process begins with obstruction of the appendiceal lumen, usually from a fecalith or lymphoid hyperplasia. More unusual inciting causes include foreign bodies, malignancy, and parasites. As a result of this obstruction, the appendiceal lumen becomes unable to drain and dilates. This distension leads to the second stage of disease, in which the eighth through 10th visceral afferent thoracic nerves are stimulated by the increasing appendiceal distension. During this second phase, patients experience peri-umbilical pain. With increasing distension of the appendix, perfusion to the luminal wall of the appendix decreases, producing the third stage of acute appendicitis. Decreased perfusion allows for bacterial invasion of the appendiceal lumen, creating the fourth stage, consisting of inflammation of the wall of the appendix. Finally, inflammation spreads outside of the appendix and into the peritoneum, leading to the fifth and final stage of appendicitis. During this period, as a result of the irritation of parietal peritoneum, patients experience pain in the right lower quadrant.[17,30]

Epidemiology. Appendicitis is the most common surgical cause of abdominal pain in pediatric patients presenting to the ED, with a worldwide incidence of appendicitis estimated to be 86 cases per 100,000 patients per year. It is estimated that there are 70,000 hospital discharges each year for acute pediatric appendicitis, with approximately one-third of all children developing rupture of the appendix prior to surgical intervention. Rates of rupture increase in an inverse relationship to patient age, with perforation rates as high as 82% in children younger than 5 years old and 100% in children younger than 1 year old.[17,30,31]

Presentation. Abdominal pain remains one of the most common presentations of acute appendicitis in children. However, this symptom may be challenging to elicit. Fever is also commonly present in infants with acute appendicitis, but is not universally present. As a result of the intra-abdominal inflammation and discomfort, infants with acute appendicitis may also develop poor feeding (a manifestation of anorexia or nausea) and vomiting. However, it remains important to remember that infants with appendicitis often present with atypical symptoms, making acute appendicitis a very difficult diagnosis to determine based on history alone. A review of a large number of studies on acute appendicitis in the pediatric population identified fever as the most predictive symptom of appendicitis, with a positive likelihood ratio of 3.4. However, being a relatively common symptom, fever is of low specificity for the diagnosis of acute appendicitis, making it of limited utility in detecting this condition.[31,32]

The physical examination for appendicitis may be challenging in infants due to their inability to communicate and lack of cooperation. The examination should focus on trying to determine the location of pain and assessing whether or not signs of peritoneal inflammation, such as rebound tenderness or guarding, are present. When performing the abdominal examination, it is important to remember that voluntary and involuntary guarding are very different. While voluntary guarding is a state of contraction of the abdominal musculature due to fear or pain, which can be overcome by distraction, involuntary guarding is spasm of the abdominal musculature secondary to peritoneal inflammation, which remains constant despite distraction or relaxation. Furthermore, rebound tenderness is a physical exam finding that is suggestive of peritoneal irritation, but may be difficult to elicit in infants. Rebound tenderness is defined as increasing pain with release of pressure on the abdomen rather than with compression of the abdomen.

When assessing an infant with suspected appendicitis, the focus of the examiner's gaze should be on the patient's face to look for grimacing or increased crying with palpation in a particular area. While classically tenderness to palpation in the right lower quadrant of the abdomen is strongly suggestive of acute appendicitis, a review of studies of the physical examination in appendicitis has shown rebound tenderness to be most predictive of appendicitis, with a positive likelihood ratio of 3.0. This same review found signs of diffuse peritoneal irritation on abdominal examination, younger age, and higher temperature elevation as predictive of ruptured appendicitis rather than unperforated appendicitis. In children who are able to ambulate, asking them to walk or jump can be an excellent screening test to look for peritonitis. As a result of the intra-abdominal inflammation in peritonitis, any movement causes pain, and so affected patients will be unwilling to walk or jump. A similar technique may be used in non-ambulatory infants. Asking the caretaker to bounce the child up and down on their lap will cause pain if peritonitis is present, and will induce crying or fussiness.[1,31,32]

In the majority of cases, the appendix lies in the right lower quadrant of the abdomen. When the appendix lies in the retrocecal position, it may produce pain in the right upper quadrant or persistent pain in the epigastrium. The psoas sign can be used in cooperative children to detect retrocecal appendicitis. A positive psoas sign occurs when pain is elicited in the right lower quadrant with passive extension of the hip.[33,34]

One key point to remember about acute appendicitis is that it is commonly mistaken for gastroenteritis. As such, the diagnosis of acute appendicitis should be strongly considered in patients who have previously been diagnosed with gastroenteritis who return to the ED with vomiting and abdominal pain. Additionally, any patient with vomiting and abdominal pain who has abdominal tenderness should be evaluated for appendicitis, as gastroenteritis generally does not produce abdominal tenderness on examination.[1,3]

Table 2. Likelihood Ratios for Laboratory Tests in Appendicitis[37]

Test	Cutoff Value	Likelihood Ratio
White blood cell count (WBC)	10,000/µL	Over 10,000: + LR 2.0 Under 10,000: - LR 0.22
	15,000/µL	Over 15,000: + LR 3.4
C reactive protein (CRP)	25 mg/L	Over 25: + LR 5.2
	10 mg/L	Under 10: - LR 0.45
Erythrocyte sedimentation rate (ESR)	20 mm/h	Over 20: + LR 3.8 Under 20: - LR 0.68

Laboratory Studies and Diagnostic Imaging. As acute appendicitis is a condition of infection and inflammation, many clinicians obtain a C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and complete blood count, specifically looking for an elevated white blood cell count. The predictive value of each is dependent on the threshold value used to decide whether or not a patient is likely to have acute appendicitis. A summary of the predictive values of laboratory tests used in the diagnosis of acute appendicitis is presented in Table 2. It is important to recognize that any individual laboratory study lacks adequate sensitivity, specificity, or predictive value to be used to determine whether or not the patient being evaluated has acute appendicitis.[31]

While previously appendicitis was a clinical diagnosis, imaging has become the mainstay of confirming or excluding the diagnosis. As there is no ionizing radiation associated with it, ultrasound is becoming the first-line radiologic study. This study consists of graded compression in the right lower quadrant to displace loops of bowel and visualize the appendix. A positive study consists of a non-compressible, fluid-filled tubular appendix, with a diameter of greater than 6 mm. One may also see hyperemia of the appendix or an appendicolith. Ultrasound has been extensively studied for its use in the diagnosis of acute appendicitis, with estimated sensitivity of 78-94% and specificity 89-98% in the pediatric population. (*See Figure 7.*) The main limitation of ultrasound is the fact that it is operator-dependent, and visualization of the appendix can be impaired by a number of patient factors, most notably obesity and bowel gas. As adults tend to have more abdominal adipose tissue than children, ultrasound for the detection of appendicitis is generally reserved for pediatric patients.[17]

Computed tomography (CT) of the abdomen has become the gold standard for the diagnosis of acute appendicitis. (*See Figure 8.*) A positive study demonstrates an appendix with a diameter greater than 6 mm, and demonstrates inflammatory changes of the appendix, including thickening of the appendiceal wall, contrast enhancement of the appendiceal wall, and fat stranding around the appendix. Studies evaluating the performance of CT in diagnosing appendicitis in the pediatric population purport sensitivities of 88% to 97% and specificities of 94% to 97%. The majority of studies on the topic have found sensitivities greater than 95%. The main limitations of CT include the fact that children may require sedation or restraint to lie still for the study, IV contrast is required at most centers, and that CT exposes children to ionizing radiation, which may increase the risk of malignancy in the future.[17]

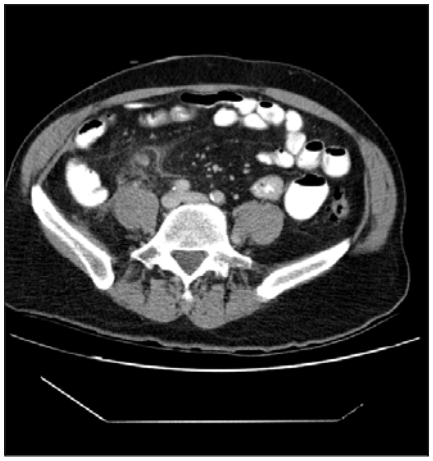
Although ultrasound is an excellent modality for the evaluation of suspected appendicitis, the sensitivity of ultrasound in detecting acute appendicitis is less than that of CT. To minimize the amount of radiation and also to address the imperfect sensitivity of ultrasound, a staged ultrasound and CT pathway may be used. In such a protocol, ultrasound is used as the initial imaging study, with CT only performed if the ultrasound is equivocal. Research on such protocols has demonstrated that half of patients may be managed with ultrasound alone, with a rate of missed appendicitis less than 0.5%. Furthermore, this approach has a sensitivity of 99% with a specificity of 91% and reduces radiation exposure from CT scans.[35,36]

While currently used only infrequently in pediatric patients, magnetic resonance imaging (MRI) is a technology that may be used more in the future, as it provides outstanding image detail and carries no risk of radiation.[37]

Treatment. In infants with acute appendicitis, treatment should primarily focus on stabilizing and correcting metabolic derangements. IV access should be obtained, patients should be rehydrated, if necessary, with IV fluids, and pediatric surgical consultation should be obtained. In non-perforated appendicitis, patients may be started on a single agent, with cefoxitin, cefotetan, piperacillin/tazobactam, or ampicillin/sulbactam being frequently used agents. For perforated appendicitis, many surgeons elect to use "triple" therapy, such as with ampicillin, gentamycin, and metronidazole, or other combination regimens such as ceftriaxone and metronidazole. However, a review of the literature on this topic has demonstrated that single-agent, broad-spectrum antibiotics may be used for both perforated and non-perforated appendicitis, with piperacillin/tazobactam being a commonly used antimicrobial for both situations. In patients with non-perforated appendicitis, appendectomy should be performed, most commonly via the laparoscopic approach. Recent research on the topic has shown that the risk of perforation is not increased if appendectomy is delayed for 12-24 hours, suggesting that appendectomy may be performed on a semi-urgent basis. In tune with this evidence, nearly 70%

Figure 7. Ultrasound of Appendicitis

Figure 8. CT of Appendicitis



of pediatric surgeons perform appendectomies on non-perforated appendicitis on a semi-urgent basis.[38,39]

Management of perforated appendicitis is somewhat controversial, with some surgeons preferring to operate immediately, while others prefer a non-operative approach consisting of initially treating the patient with intravenous antibiotics followed by an interval appendectomy in two to three months after initial hospital discharge. In cases in which a discrete abscess has formed, patients may be managed initially with intravenous antibiotics and percutaneous drainage. It is important to recognize that the non-operative approach to appendicitis should be used only in non-toxic infants; any infant with significant toxicity likely deserves surgical intervention.[38,39]

Gastroenteritis

Etiology. While viral gastroenteritis may be caused by a number of different viral pathogens, the two most common causes are rotavirus and norovirus. In the United States, norovirus accounts for more than 90% of all outbreaks of viral gastroenteritis and is the most common cause of gastroenteritis worldwide. Rotavirus, in comparison, is the leading cause of viral gastroenteritis in children younger than 5 years old. Fortunately, vaccines are available against rotavirus, and have been shown to decrease both incidence of disease and severity of symptoms.[40-43]

Both agents are primarily transmitted by the fecal-oral route, although viral particles may be spread by respiratory droplets as well. Interpersonal transmission may be rapid, leading to outbreaks of these infections in groups of people in close proximity, such as on cruise ships, in nursing homes, and in daycare centers.[40,41]

Epidemiology. Viral gastroenteritis is an extremely frequent entity in children, accounting for 150,000 hospitalizations and 3.7 million physician visits annually in the United States. It is also estimated to be the cause of up to 10% of all hospitalizations in the United States in children younger than 5 years of age. With good access to medical care and rehydration, deaths from gastroenteritis in developed nations are relatively uncommon. Globally, gastroenteritis is a major problem as a result of poor access to adequate rehydration and is estimated to cause up to 870,000 deaths per year.[40,42,44]

Presentation. Gastroenteritis can be defined as a syndrome of vomiting, diarrhea, or the combination of both. Viral infections account for approximately 70% of all cases of acute gastroenteritis, and generally present with nausea, vomiting, and watery diarrhea. Affected individuals generally also have low-grade fevers, abdominal pain, cramping, and some degree of malaise. Blood or mucus seen in vomitus or stool is unusual in viral gastroenteritis and is suggestive of an invasive bacterial pathogen. Viral gastroenteritis is a self-limited disease, usually lasting anywhere from 24-60 hours in immunocompetent hosts. In immunocompromised patients and patients at the extremes of age, symptoms may be more severe and prolonged.[40,41]

Infants with abdominal pain, vomiting, and diarrhea may also be suffering from bacterial or even protozoal gastroenteritis. Many of these conditions produce vomiting, abdominal pain, and diarrhea in a manner that is very difficult to distinguish clinically from viral infections.[40]

Laboratory Studies and Diagnostic Imaging. Viral gastroenteritis is a clinical diagnosis. Routine laboratory studies, such as a complete blood count or basic metabolic panel, are largely unhelpful when trying to determine the etiologic pathogen in a patient with vomiting and diarrhea. Urinalysis may also be helpful in assessing a patient's level of dehydration.[40] Plain X-rays of the abdomen should only be obtained if another etiology of the patient's symptoms is suspected.

PCR studies of vomitus or stool are the best tests to isolate the viral pathogen causing a patient's symptoms. However, such tests are not available in all clinical laboratories. As viral gastroenteritis is a self-limiting condition, the utility of PCR testing is limited; a patient's symptoms may have already resolved prior to the results of the test being available. Stool studies, such as a stool culture, are only performed when there is concern for a non-viral cause of vomiting or diarrhea, such when symptoms last longer than two weeks or there is blood or mucus in the stool. A stool white blood cell can also be performed to look for intestinal inflammation, but is of poor sensitivity and specificity and does not indicate the cause of inflammation.[40,41]

Treatment. No specific treatment exists for viral gastroenteritis. However, the fluid losses from vomiting and diarrhea, in combination with poor oral intake from nausea, can lead to dehydration. Treatment

of this condition, thus, is focused on rehydration. The first step in managing patients with viral gastroenteritis is to assess their level of dehydration. Signs and symptoms of moderate dehydration include: decreased urine output, capillary refill greater than two seconds, decreased skin turgor, and tachypnea. Tachycardia also is frequently a sign of mild to moderate dehydration. Laboratory abnormalities that suggest dehydration include low serum bicarbonate, hypokalemia, and hypoglycemia. In infants with mild to moderate dehydration, oral rehydration remains first-line therapy. Oral intake can be increased with the use of the antiemetic ondansetron. Infants unable to adequately hydrate orally should receive intravenous hydration. Indications for admission to the hospital include moderate to severe dehydration with inability to tolerate oral fluids, intractable emesis, marked electrolyte disturbances, and poor social situation, including lack of parental reliability.[41,44-46]

Other Conditions in the Differential Diagnosis of Abdominal Pain and Vomiting

The above listed conditions, while essential to understand when approaching an infant with vomiting and abdominal pain, only represent a very small number of the possible causes of abdominal pain and vomiting. Infants are particularly prone to vomiting, and may vomit for any number of infectious causes, including urinary tract infections, otitis, pneumonia, pharyngitis, hepatitis, sepsis, and upper respiratory infections. Furthermore, infants with metabolic conditions such as diabetic ketoacidosis, adrenal insufficiency, and inborn errors of metabolism may present with abdominal pain and vomiting. One should also consider additional intra-abdominal pathology, such as small bowel obstruction, incarcerated hernia, and gonadal torsion.[2,47]

Conclusion

Abdominal pain and vomiting in the infant patient remains a challenging complaint to evaluate. A solid understanding of the differential diagnosis, combined with a thorough history and physical examination, provide an excellent start to the evaluation of this presentation. Use of appropriate laboratory testing and imaging studies aids in the confirmation of surgical causes of abdominal pain and vomiting, and can guide appropriate and timely surgical consultation when necessary.

References

1. McCullough M, Sharieff GQ. Abdominal surgical emergencies in infants and young children. *Emerg Med Clin North Am* 2003;21:909-935.
2. Marin JR, Alpern ER. Abdominal pain in children. *Emerg Med Clin North Am* 2011;29:401-428.
3. D'Agostino J. Common abdominal emergencies in children. *Emerg Med Clin North Am* 2002;20:139-53.
4. Sondheimer JM. Vomiting. In: Walker WA, Goulet O, Kleinman, RE, et al (Eds). *Pediatric Gastrointestinal Disease: Pathopsychology, Diagnosis, Management*, 4th ed. BC Decker, Ontario; 2004:203.
5. Torres AM, Ziegler MM. Malrotation of the intestine. *World J Surg* 1993;17: 326-331.
6. Lampl B, Levin TL, Berdon WE, et al. Malrotation and midgut volvulus: A historical review and current controversies in diagnosis and management. *Pediatr Radiol* 2009;39:359-66.
7. Strouse PJ. Disorders of intestinal rotation and fixation ("malrotation"). *Pediatr Radiol* 2004;34:837-51.
8. de Lorijn F, Boeckxstaens GE, Benninga MA. Symptomatology, pathophysiology, diagnostic work-up, and treatment of Hirschprung disease in infancy and childhood. *Curr Gastroenterol Rep* 2007;9:245-53.
9. Applegate KE, Anderson JM, Klatte EC. Intestinal malrotation in children: A problem-solving approach to the upper gastrointestinal series. *Radiographics* 2006;26:1485-500.
10. Shew SB. Surgical concerns in malrotation and midgut volvulus. *Pediatr Radiol* 2009;39:S167-71.
11. Neu J, Walker WA. Necrotizing enterocolitis. *N Engl J Med* 2011;364:255-64.
12. Bradshaw WT. Necrotizing enterocolitis: Etiology, presentation, management, and outcomes. *J Perinatal Neonatal Nurs* 2009;23:87-94.
13. Petrosyan M, Guner YS, Williams M, et al. Current concepts regarding the pathogenesis of necrotizing enterocolitis. *Pediatr Surg Int* 2009;25:309-318.
14. Evennett N, Alexander N, Petrov M, et al. A systematic review of serologic tests in the diagnosis of necrotizing enterocolitis. *J Pediatr Surg* 2009;44:2192-201.
15. Oh S, Young C, Gravenstein N, et al. Monitoring technologies in the neonatal intensive care unit: Implications for the detection of necrotizing enterocolitis. *J Perinatol* 2010;30:701-708.
16. Kenny SE, Tam PK, Garcia-Barcelo M. Hirschsprung's disease. *Semin Pediatr Surg* 2010;19:194-200.
17. Brennan GD. Pediatric appendicitis: Pathophysiology and appropriate use of diagnostic imaging. *CJEM* 2006;8: 425-432.
18. Marquez TT, Acton RD, Hess DJ, et al. Comprehensive review of procedures for total colonic aganglionosis. *J Pediatr Surg* 2009;44:257-265.
19. Haricharan RN, Georgeson KE. Hirschsprung disease. *Semin Pediatr Surg* 2008;17:266-275.
20. MacMahon B. The continuing enigma of pyloric stenosis of infancy: A review. *Epidemiology* 2006;17:195-201.
21. Aspelund G, Langer JC. Current management of hypertrophic pyloric stenosis. *Semin Pediatr Surg* 2007;16:27-33.
22. Panteli C. New insights into the pathogenesis of infantile pyloric stenosis. *Pediatr Surg Int* 2009;25:1043-1052.
23. Maheshwai N. Are young infants treated with erythromycin at risk for developing hypertrophic pyloric stenosis? *Arch Dis Child* 2007;92:271-273.
24. Shekherdimian S, Lee SL. Management of pediatric intussusception in general hospitals: Diagnosis, treatment, and differences based on age. *World J Pediatr* 2011;7: 70-73.
25. Hernanz-Schulman M. Pyloric stenosis: Role of imaging. *Pediatr Radiol* 2009;39:S134-S139.
26. Ho KS, Schenk JP, Troger J, et al. Current radiological management of intussusceptions in children. *Eur Radiol* 2007;17:2411-2421.
27. Mendez D, Caviness AC, Ma L, et al. The diagnostic accuracy of an abdominal radiograph with signs and symptoms of intussusception. *Am J Emerg Med* 2011 [Epub ahead of print].
28. Vasavada P. Ultrasound evaluation of acute abdominal emergencies in infants and children. *Radiol Clin North Am* 2004;42:445-456.
29. Kirks DR. Air intussusceptions reduction: "The winds of change." *Pediatr Radiol* 1995;25:89-91.
30. Birnbaum BA, Wilson SR. Appendicitis at the millennium. *Radiology* 2000;215: 337-348.
31. Bundy DG, Byerley JS, Liles EA, et al. Does this child have appendicitis? *JAMA* 2007;298:438-451.
32. Gosain A, Williams RF, Blakely ML. Distinguishing acute from ruptured

appendicitis preoperatively in the pediatric patient. *Adv Surg* 2010;44:73-85.

33. Kim S, Lim HK, Lee JY, et al. Ascending retrocecal appendicitis: Clinical and computed tomographic findings. *J Comput Assist Tomogr* 2006;30:772-776.
34. John H, Neff U, Kelemen M. Appendicitis diagnosis today: Clinical and ultrasonic deductions. *World J Surg* 1993;17:243-249.
35. Ramarajan N, Krishnamoorthi R, Barth R, et al. An interdisciplinary initiative to reduce radiation exposure: Evaluation of appendicitis in a pediatric emergency department with clinical assessment supported by a staged ultrasound and computed tomography pathway. *Acad Emerg Med* 2009;16:1258-1265.
36. Krishnamoorthi R, Ramarajan N, Wang NE, et al. Effectiveness of a staged US and CT protocol for the diagnosis of pediatric appendicitis: Reducing radiation exposure in the age of ALARA. *Radiology* 2011;259:231-239
37. Singh A, Danrad R, Hahn RF, et al. MR imaging of the acute abdomen and pelvis: Acute appendicitis and beyond. *Radiographics* 2007;27:1419-1431.
38. Morrow SE, Newman KD. Current management of appendicitis. *Semin Pediatr Surg* 2007;16:34-40.
39. Lee SL, Islam S, Cassidy LD, et al. Antibiotics and appendicitis in the pediatric population: An American Pediatric Surgical Association Outcomes and Clinical Trials Committee systematic review. *J Pediatr Surg* 2010;45:2181-2185.
40. Getto L, Zeserson E, Breyer M. Vomiting, diarrhea, constipation, and gastroenteritis. *Emerg Med Clin North Am* 2011;29:211-237.
41. Koo HL, Ajami N, Atmar RL, et al. Noroviruses: The leading cause of gastroenteritis worldwide. *Discov Med* 2010;10:61-70.
42. Szajewska H, Dziechciarz P. Gastrointestinal infections in the pediatric population. *Curr Opin Gastroenterol* 2010;26:36-44.
44. Colletti JE, Brown KM, Sharieff GQ, et al. The management of children with gastroenteritis and dehydration in the emergency department. *J Emerg Med* 2010;38:686-698.
43. Dennehy PH. Rotavirus vaccines: An overview. *Clin Microbiol Rev* 2008;21:198-208.
45. DeCamp LR, Byerley JS, Doshi N, et al. Use of antiemetic agents in acute gastroenteritis: A systematic review and meta-analysis. *Arch Pediatr Adolesc Med* 2008;162:858-865.
46. Freedman SB, Adler M, Seshadri R, et al. Oral ondansetron for gastroenteritis in a pediatric emergency department. *N Engl J Med* 2006;354:1698-1705.
47. McIntosh M. Infants and toddlers. In: Adams JG, ed. *Emergency Medicine*. Saunders Elsevierz; Philadelphia, PA; 2008: 199-2010.

Physician CME Questions

1. Which of the following confers an increased risk of developing pyloric stenosis?
   A. female gender
   B. exposure to erythromycin in the neonatal or prenatal period
   C. exposure to penicillin in the neonatal or prenatal period
   D. formula feeding

2. Which of the following constitutes a positive ultrasound for the diagnosis of pyloric stenosis?
   A. pyloric muscle thickness of 2 mm
   B. pyloric muscle thickness of 5 mm
   C. pyloric canal length of 8 mm
   D. pyloric canal length of 12 mm

3. Which of the following treatment modalities is used as first-line therapy for intussusception?
   A. air contrast enema
   B. surgery
   C. laparotomy
   D. intravenous antibiotics and inpatient observation

4. Necrotizing enterocolitis is most common in which areas of the intestine?
   A. duodenum and jejunum
   B. descending colon and rectum
   C. jejunum, ileum, and cecum
   D. transverse colon

5. Which of the following findings on plain abdominal X-ray is pathognomonic for necrotizing enterocolitis?
   A. thumbprinting
   B. the double bubble sign
   C. visualization of the falciform ligament
   D. pneumatosis intestinalis

6. Which of the following genetic syndromes confers an increased risk of Hirschsprung's disease?
   A. fragile X syndrome
   B. neurofibromatosis type 2
   C. Down syndrome
   D. Marfan syndrome

7. Which of the following constitutes a positive contrast enema for Hirschsprung's disease?
   A. inability to inject contrast into the rectum
   B. abrupt change in caliber of bowel size, with dilated bowel proximally
   C. abrupt change in caliber of bowel size, with dilated bowel distally
   D. an abnormally narrowed terminal ileum

8. Which of the following has been shown to be the most predictive symptom for acute appendicitis in infants?
   A. vomiting
   B. lethargy
   C. fever
   D. decreased feeding

9. Which of the following is a limitation of computed tomography in evaluating infants with suspected appendicitis?
   A. In most centers, IV contrast must be administered during the CT scan to make the diagnosis of acute appendicitis.
   B. CT exposes the patient to ionizing radiation.
   C. Infants may require sedation or restraint to minimize movement during the study.
   D. All of the above are limitations.

10. Which of the following is an antibiotic that can be given as monotherapy for both unruptured and ruptured appendicitis?
    A. metronidazole
    B. piperacillin/tazobactam
    C. ceftriaxone
    D. gentamycin

CME Instructions

Here are the steps you need to take to earn credit for this activity:

1. Read and study the activity, using the provided references for further research.

2. Log on to **www.cmecity.com** to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*

3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.

4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.

5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.** You will no longer have to wait to receive your credit letter.

Pediatric Emergency Medicine Reports™ (ISSN 1082-3344) is published monthly by AHC Media, a division of Thompson Media Group LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Donald R. Johnston
Executive Editor: Shelly Morrow Mark
Managing Editor: Leslie Hamlin

GST Registration No.: R128870672
Periodicals Postage Paid at Atlanta, GA 30304 and at additional mailing offices.

POSTMASTER: Send address changes to *Pediatric Emergency Medicine Reports*, P.O. Box 105109, Atlanta, GA 30348.

Back issues: $65. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail Address: customerservice@ahcmedia.com
Editorial E-Mail Address: shelly.mark@ahcmedia.com
World-Wide Web page: http://www.ahcmedia.com

Subscription Prices

1 year with 30 ACEP, AMA, or AAP Category 1 credits: $439;
1 year without credit: $389;
Add $17.95 for shipping & handling

Multiple copies:
Discounts are available for group subscriptions, multiple copies, site-licenses or electronic distribution. For pricing information, call Tria Kreutzer at 404-262-5482.
One to nine additional copies: **$350 each;**
10 or more additional copies: **$311 each.**
Resident's Rate: **$194.50**
All prices U.S. only. U.S. possessions and Canada, add $30 postage plus applicable GST.

Accreditation

AHC Media is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

AHC Media designates this enduring material for a maximum of 30 *AMA PRA Category 1 Credits™*. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Approved by the American College of Emergency Physicians for 30 hours of ACEP Category 1 credit.

This continuing medical education activity has been reviewed by the American Academy of Pediatrics and is acceptable for up to 30 (2.5 per issue) AAP credits. These credits can be applied toward the AAP CME/CPD Award available to Fellows and Candidate Fellows of the American Academy of Pediatrics.
This CME activity is intended for emergency and pediatric physicians.

It is in effect for 36 months from the date of the publication.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

AHC Media

Exclusive to our subscribers

Rapid Access Management Guidelines

Pediatric Emergency Medicine Reports

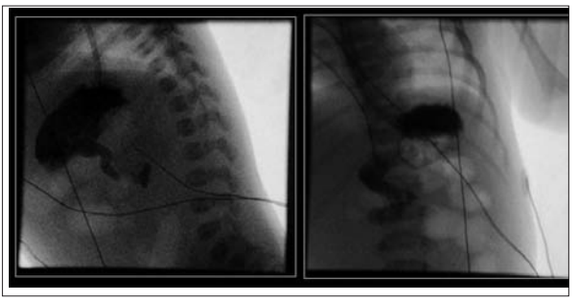
The Practical Journal of Pediatric Emergency Medicine

Abdominal Pain and Vomiting in the Infant

Presenting Age and Frequency of Conditions Causing Abdominal Pain and Vomiting in Infants

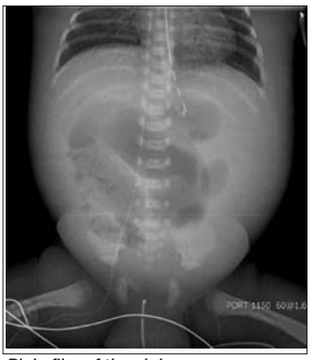
Condition	Presenting Age	Frequency
Pyloric stenosis	3rd - 8th week of life	2-4 per 1000 births
Intussusception	3rd month - 5th year of life (most common in 1st year of life)	18-56 per 100,000 births
Necrotizing enterocolitis	1st - 10th day of life	12% of babies under 1500 g 10% of all cases in term
Malrotation with volvulus	Any age 80% within first month of life	Malrotation: 1 per 500 births; malrotation with volvulus: 1 per 5000 births
Gastroenteritis	Any age	3.7 million physician visits in the United States annually
Hirschsprung disease	First days of life	1 per 5000 births
Acute appendicitis	Any age; < 5% younger than 5 years, most frequent in 2nd decade of life	86 per 100,000 patients per year

Malrotation with Volvulus



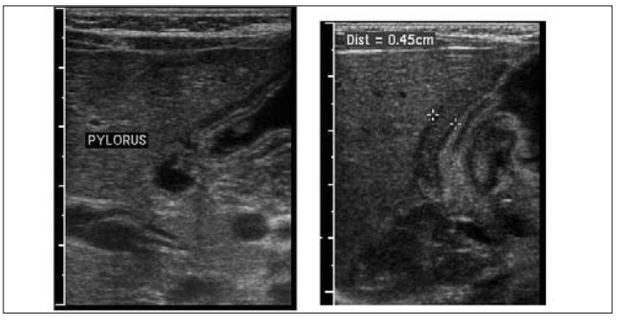
Two images from an upper GI study demonstrating lack of transit of contrast from the stomach and duodenal bulb into the proximal small bowel with corkscrew configuration of the proximal duodenum consistent with malrotation with volvulus. Images courtesy of Casey Grover, MD.

Necrotizing Enterocolitis



Plain film of the abdomen demonstrating intramural air (pneumatosis intestinalis) in the right lower abdomen, consistent with necrotizing enterocolitis. Image courtesy of Casey Grover, MD.

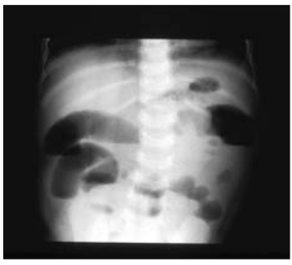
Pyloric Stenosis



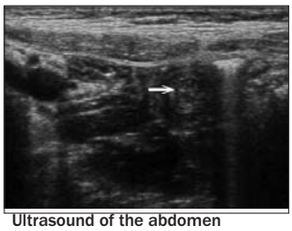
Likelihood Ratios for Laboratory Tests in Appendicitis

Test	Cutoff Value	Likelihood Ratio
White blood cell count (WBC)	10,000/µL	Over 10,000: + LR 2.0 Under 10,000: - LR 0.22
	15,000/µL	Over 15,000: + LR 3.4
C reactive protein (CRP)	25 mg/L	Over 25: + LR 5.2
	10 mg/L	Under 10: - LR 0.45
Erythrocyte sedimentation rate (ESR)	20 mm/h	Over 20: + LR 3.8 Under 20: - LR 0.68

Intussusception



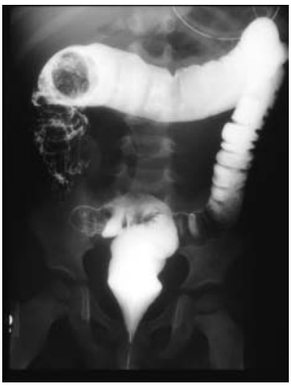
Small Bowel Intussusception



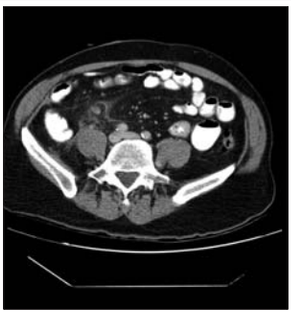
Ultrasound of the abdomen demonstrating a target sign (white arrow) consistent with a small bowel intussusception. Image courtesy of Casey Grover, MD.

Ultrasound of Appendicitis

Intussusception



CT of Appendicitis



Supplement to *Pediatric Emergency Medicine Reports*, October 2011: "Abdominal Pain and Vomiting in the Infant." *Authors:* **Casey Grover, MD,** Stanford/Kaiser Emergency Medicine Residency, Stanford, CA; and **Ewen Wang, MD**, Associate Professor of Surgery/Emergency Medicine, Associate Director, Pediatric Emergency Medicine, Stanford School of Medicine, Stanford, CA.