Lower Gastrointestinal Bleeding & Intussusception

Benjamin E. Padilla, MD[a,*], Willieford Moses, MD[b]

KEYWORDS

• Pediatric • Gastrointestinal bleeding • Intussusception • Hydrostatic reduction
• Pneumatic reduction

KEY POINTS

- Lower gastrointestinal bleeding in children is uncommon.
- The differential diagnosis is largely guided by the age of the patient.
- Surgical emergencies in the neonatal period, such as necrotizing enterocolitis, midgut volvulus, and Hirschsprung disease, can present with gastrointestinal bleeding.
- Ileocolic intussusception is a common cause of gastrointestinal bleeding.
- Radiologic reduction of an ileocolic intussusception is first-line therapy for a patient without peritonitis or hemodynamic instability.

LOWER GASTROINTESTINAL BLEEDING

Epidemiology

Although data remain limited regarding the incidence of lower gastrointestinal (GI) bleeding (LGIB) in children, a Healthcare Cost and Utilization Project Nationwide Emergency Department Sample analysis from 2006 to 2011 estimated that there were a total of 437,000 emergency department (ED) visits associated with GI bleeding in the pediatric population (children up to 19 years old).[1] Of these visits, 20% were identified as upper GI bleeding, 30% were LGIB, and the remaining 40% were not specified. By age, 38% of the patients were younger than 5 years, 23% were between 5 and 15 years, and 39% were between 15 and 19 years. Interestingly, only 11.6% of the ED visits required hospitalization with most being treated either in the ED or as an outpatient.

Clinical Presentation and Initial Evaluation

Patients with LGIB can present with nausea, vomiting, diarrhea, and abdominal pain. A complete history should be obtained, including the duration and amount of bleeding

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[a] Division of Pediatric Surgery, University of California, San Francisco, 550 16th Street, 5th Floor, Box 0570, San Francisco, CA 94158-2549, USA; [b] General Surgery, Department of Surgery, University of California, San Francisco, 513 Parnassus Avenue, S-321, San Francisco, CA 94143, USA
* Corresponding author.
E-mail address: benjamin.padilla@ucsf.edu

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and the color and consistency of the blood. Although a well-appearing infant or child is relatively reassuring, the clinical condition can deteriorate precipitously in the face of ongoing bleeding. Initial physical examination and evaluation should focus on ascertaining the child's hemodynamic condition and promptly initiating resuscitation if indicated. A thorough physical examination should be performed, including inspection for anal fissures; a rectal examination for prolapse, polyps, or masses; and stool guaiac testing. The abdomen should be assessed for peritonitis and the presence of an abdominal mass. The findings of petechiae or bruising on the skin may suggest an underlying coagulopathy.

The initial diagnostic test of choice for children presenting with LGIB largely depends on their age, clinical status, and the likelihood for various underlying pathologic conditions. If the source of the bleeding (lower vs upper tract) is unclear, a nasogastric tube can be placed for lavage. Abdominal plain radiographs, particularly in the neonatal and infant population, can aid in the diagnosis of underlying conditions such as necrotizing enterocolitis (NEC) or Hirschsprung disease. Ultrasonography is also helpful in identifying underlying conditions such as intussusception. Although endoscopy (upper and lower) requires general anesthesia, it can help in both identifying the lesion and providing an opportunity for intervention.[2] Red blood cell scintigraphy and angiography can also aid in identifying the source of bleeding, although slow bleeding is often difficult to identify with these modalities.

Differential Diagnosis

The differential diagnosis for LGIB in the pediatric population is broad. However, it can be quickly narrowed based on the age group of the patient and the clinical presentation. It is helpful to categorize the patients into 3 groups to assist in the diagnosis: neonatal, infant and toddler, and school age (**Box 1**).

NEONATAL

LGIB in the neonatal population presents a diagnostic challenge because of the broad range of etiologies, from benign conditions, such as swallowed maternal blood to more concerning conditions, such as midgut volvulus or NEC. As mentioned, the clinical presentation (well child vs distressed) and the hemodynamic status of the patient are essential to effectively diagnose and treat the underlying condition. Although not exhaustive, the following is a summary of the common conditions that can present with LGIB in the neonates.

MIDGUT VOLVULUS

One of the true surgical emergencies in pediatrics, midgut volvulus, is an obstructive condition of the small intestines caused by twisting of the bowel around the axis of the mesentery. In addition to intestinal obstruction, the mesenteric blood vessels are kinked, resulting in ischemia to the midgut that can manifest as LGIB. Bilious emesis in an infant is alarming and should prompt an expeditious workup for midgut volvulus. Midgut volvulus typically occurs in the setting of intestinal malrotation caused by failed rotation and fixation of the gut during in utero development. Melena or hematochezia is present in up to 20% of cases and is an ominous sign suggestive of gut ischemia. Similarly, abdominal wall erythema caused by bowel ischemia is a late and concerning finding. The diagnosis is established with an upper GI contrast study that shows proximal duodenal obstruction (midgut volvulus) or abnormal rotation and positioning of the duodenal-jejunal junction (malrotation).[3,4] Emergency surgical intervention with a

Box 1
Lower gastrointestinal bleeding differential diagnosis

Neonatal

- Swallowed blood
- Fissures
- Necrotizing enterocolitis
- Midgut volvulus
- Hirschsprung disease
- Vascular
- Coagulopathy
- GI duplication

Infant and Toddler

- Fissures
- Allergic colitis
- Intussusception
- Meckel diverticulum
- GI duplication

School Age

- Infections
- Polyps
- IBD
- Malignancies
- Typhlitis
- Henoch-Schonlein purpura
- Hemolytic uremic syndrome

Ladd procedure to relieve the obstruction and restore perfusion to the bowel can be lifesaving.[5,6]

Necrotizing Enterocolitis

Also considered a surgical emergency, NEC likely results from relative gut ischemia and infection of the intestines caused by bacterial translocation across the immature intestinal epithelium.[7,8] Risk factors for NEC include severe prematurity, low Apgar score, low birth weight, and hyperosmolar enteral feedings. Although most commonly occurring in the setting of prematurity, between 5% and 25% of NEC occurs in full-term infants.[9,10] Babies with gastroschisis and congenital heart defects are also at risk for NEC. Patients can present with occult or gross bleeding, often in the setting of clinical decompensation, and abdominal distension, vomiting, or diarrhea. The ileum, cecum, and right colon are most commonly involved. Findings on plain radiographs that support the diagnosis of NEC include dilated or fixed intestinal loops, pneumatosis intestinalis, portal venous gas, or pneumoperitoneum.[11] Although most patients are effectively treated with bowel rest and intravenous antibiotics, evidence of intestinal perforation or necrosis and clinical deterioration despite antibiotics are indications to operate.

Hirschsprung Disease

Hirschsprung disease (HD) is an obstructive disorder of the colon caused by incomplete cranio-caudal migration of enteric ganglion cells in the colon. The incidence is about 1 in 5000 live births with a 4:1 male/female predominance.[12,13] Although most commonly seen in newborns with delayed passage of meconium (>24 hours), some neonates and infants can present with progressively worsening constipation. Patients with HD commonly have enterocolitis that can manifest as LGIB. In fact, enterocolitis is the presenting clinical finding in up to 24% of patients with HD.[14] Hirschsprung enterocolitis can be life threatening and should be promptly treated with intravenous antibiotics, fluid resuscitation and colonic irrigation. Multiple surgical options exist for the treatment of HD, which is beyond the scope of this review.

Anal Fissures

Often associated with constipation, anal fissures are one of the most common causes of LGIB in the neonatal population.[1,15] Similar to older children and adults, the history is often notable for pain with defection. In infants, this can present as grunting, straining, or arching of the back during defection. Blood is typically seen around the stool and on the surface but not mixed within it. On physical examination, spreading the perianal skin will evert the anus and reveal the fissure. Treatment is usually conservative and includes dietary changes or medications depending on the patient's age.

Swallowed Maternal Blood

In a well-appearing infant without any clinical evidence of bleeding, swallowed maternal blood should be considered on the differential diagnosis for rectal bleeding. Either ingested at the time of delivery or during breast feeding, maternal blood often presents as melanotic stools or hematemesis. A history of cracked or fissured maternal nipples is common. To confirm the diagnosis, an Apt-Downey test should be performed, which distinguishes fetal hemoglobin from maternal adult hemoglobin by the degree of denaturation in the setting of alkaline solution. The more stable fetal hemoglobin will remain pinkish in color as opposed to adult hemoglobin, which turns yellowish-brown because of the higher susceptibility to denaturation.

Other Conditions

Vascular anomalies and several types of coagulopathies can all precipitate bleeding in neonates. The hemophilia syndromes along with von Willebrand disease can present in the neonatal period; however, these syndromes are rare. In addition to GI bleeding, clinical history and physical examination findings, such as unexplained bruising, petechiae, or prolonged bleeding episodes after minor trauma such as circumcision, can indicate a possible coagulation disorder.

INFANTS AND TODDLERS

The differential diagnosis for LGIB in the infant/toddler period is narrower than in neonates. Furthermore, the clinical history and presentation tend to lead toward a conclusive diagnosis. Intussusception and Meckel diverticulum often warrant the most immediate intervention, as bleeding can be indicative of a late-stage condition. Below is a summary of the most common conditions and presentations.

Meckel Diverticulum

The etiology of Meckel diverticulum is related to failed closure of the omphalomesenteric duct, also known as the vitelline duct. Embryologically, the duct serves to join the yolk sac to the lumen of the primitive midgut during early fetal development. As the placenta matures the duct no longer serves as a source of nourishment and typically obliterates by the seventh to ninth week of life. If this process fails, one of multiple congenital anomalies can occur, including but not limited to a Meckel diverticulum (most common), an umbilical sinus, an omphalomesenteric fistula, or residual fibrous cords. Despite being common, occurring in approximately 2% of the population, most patients are asymptomatic. However, painless rectal bleeding can be a common presentation because of gastric heterotopia within the diverticulum, leading to mucosal ulceration of the adjacent small intestine.[16] Although the preoperative diagnosis of a Meckel's diverticulum is often difficult, in the appropriate clinical setting, a Meckel scan can be performed in which intravenous technetium 99m concentrates in the ectopic gastric mucosa allowing for identification on scintigraphy.[17] Because the bleeding arises from ulcerations in the small bowel directly adjacent to the diverticulum, definitive therapy requires a segmental bowel resection that encompasses the Meckel diverticulum and ulcerated small bowel. Other complications that can arise from a Meckel diverticulum include intestinal obstruction, intussusception, diverticulitis, and perforation. The presentation of Meckel diverticulitis can be difficult to distinguish from acute appendicitis. If a normal appendix is encountered at the time of surgery for what was thought to be acute appendicitis, it is important to inspect the ileum for the presence of Meckel diverticulitis.

Allergic Colitis

Allergic colitis, or food protein-induced colitis, is most often caused by an inflammatory reaction to cow's milk or soy proteins. Although most commonly occurring in formula-fed infants, it can also occur in those who are breast fed owing to the lactating mother's ingestion of cow's milk. Relatively benign in nature, most infants present with loose diarrhea and occult or frank hematochezia.[18] Once other causes for lower GI bleeding have been excluded, attention should be given to identifying the precipitating agent and removing it from the diet. If formula fed, the infant should be started on a formula with an alternative protein source or amino acid composition. If breast fed, the maternal diet should be interrogated and possible causative agents removed.

Intussusception

Although classic for intussusception, the clinical triad of abdominal pain, a palpable abdominal mass, and currant jelly stools (caused by intestinal ischemia and mucosal sloughing) occurs in less than 25% of cases.[19,20] More typically, infants and toddlers present with abdominal colic: intermittent, crampy abdominal pain every 15 to 20 minutes. Intussusception is generally treated with a therapeutic enema, reserving an operation for the most difficult cases. For a more detailed understanding of intussusception, please see the following section.

SCHOOL-AGE CHILDREN

The differential diagnosis for bleeding in a school-age child (preschool and beyond) is relatively broad because of the variety of possible exposures (infections and toxins) and conditions that can present in this age group. Similar to many of the conditions discussed previously, making the diagnosis relies principally on the clinical history and presentation.

Infections

School-age children are at particular risk for infectious colitis because of their exposure to environmental pathogens (eg, daycare, school). Most food-borne infections, are self-limiting and do not often lead to lower GI bleeding. However, certain pathogens can cause significant hematochezia (eg, *Salmonella, Shigella, Campylobacter, Clostridium difficile*). The mainstay of treatment of bacterial colitis is antibiotic therapy. However, antibiotics should not be initiated if enterohemorrhagic *Escherichia coli* (strains O157:H7 or O104:H4) infection is suspected, as antibiotic treatment is associated with the development of hemolytic uremic syndrome.[21] *E coli* colitis should be suspected in patients with acute bloody diarrhea, or hemolytic uremic syndrome, particularly if associated with abdominal tenderness and the absence of fever. Antibiotic therapy for enterohemorrhagic *E coli* infection does not alter the duration of acute illnesses.[22] Thus, antibiotic therapy is not recommended for enterohemorrhagic *E coli* infections. With the exception of the immunocompromised patient, an operation for infectious colitis is rarely needed.

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD), which includes Crohn disease and ulcerative colitis, is rare in early childhood. However, IBD has a peak in incidence during late adolescence. Distinguishing between the 2 entities is often difficult, as both commonly present with abdominal pain, fevers, and diarrhea. Often the diagnosis is delayed, and patients can have growth delays associated with untreated illness.[23] Gastrointestinal bleeding in the setting of IBD is typically caused by mucosal ulceration and inflammation and is, thus, more suggestive of ulcerative colitis. Malignancy is also on the differential diagnosis as patients with IBD are at higher risk for both small and large bowel malignancy. Diagnosis of IBD typically requires endoscopic evaluation (upper and lower) with biopsies for histologic diagnosis.

Malignancies

Although rare, GI malignancies do occur in the pediatric population. Primary tumors can include GI stromal tumors, lymphomas, and adenocarcinomas. Metastatic lesions to the intestines can also present with GI bleeding. Patients receiving chemotherapy for a non-GI malignancy (often hematologic malignancies) can have typhlitis caused by the neutropenia, which can lead to GI bleeding.

Polyps

Colorectal polyps are a fairly common source for bleeding in this age group. A large analysis of the Pediatric Endoscopy Database System Clinical Outcomes Research Initiative found that polyps were found in 12% of the patients with LGIB undergoing colonoscopy.[24] Most polyps found in the pediatric population are in fact juvenile hamartomatous polyps with low likelihood for malignancy. However, they can be associated with syndromes such as juvenile polyposis syndrome or familial adenomatous polyposis, which necessitate further evaluation and treatment.[25]

INTUSSUSCEPTION

Intussusception is the invagination (telescoping) of a segment of intestine into the adjacent bowel. Intussusception is the most common cause of intestinal obstruction in infants and toddlers. In addition to obstructive symptoms, intussusception often presents with hematochezia in the form of "currant jelly stool." John Hutchinson reported the successful surgical correction of an intussusception in 1873. In 1876, Hirschsprung

described hydrostatic reduction of intussusception by enema, resulting in decreased mortality.[26] Contrast enema eventually became the accepted initial mode of therapy for intussusception in stable pediatric patients.

PATHOPHYSIOLOGY

The intussusceptum is the proximal inner segment of the intestine, whereas the intussuscipiens is the distal outer receiving portion (Fig. 1). Ileocolic intussusception is the most common location and accounts for 80% to 95% of all pediatric intussusceptions. Ileoileal, cecocolic, colocolic, and jejunojejunal intussusceptions occur with decreasing frequency. The intussusceptum drags the associated mesentery into the intussuscipiens, resulting in venous and lymphatic congestion, bowel edema, and ultimately ischemia and perforation. Although some intussusceptions spontaneously reduce, the natural history of an intussusception is to progress to perforation and sepsis unless the condition is recognized and treated appropriately.

Intussusception can occur at any age. In children, 60% occur before the age of 1 year, and 80% to 90% occur before the age of 2 years.[27] A Swiss population-based study reports yearly mean incidences of intussusception were 38, 31, and 26 cases per 100,000 live births in the first, second, and third year of life, respectively.[28] Intussusception is uncommon before 3 months and after 3 years of age. Intussusception in younger and older patients is more commonly associated with a pathologic lead point.

Idiopathic (Primary) Intussusception

Approximately 75% of intussusceptions in children do not have a pathologic lead point and are classified as idiopathic or primary intussusceptions. Idiopathic

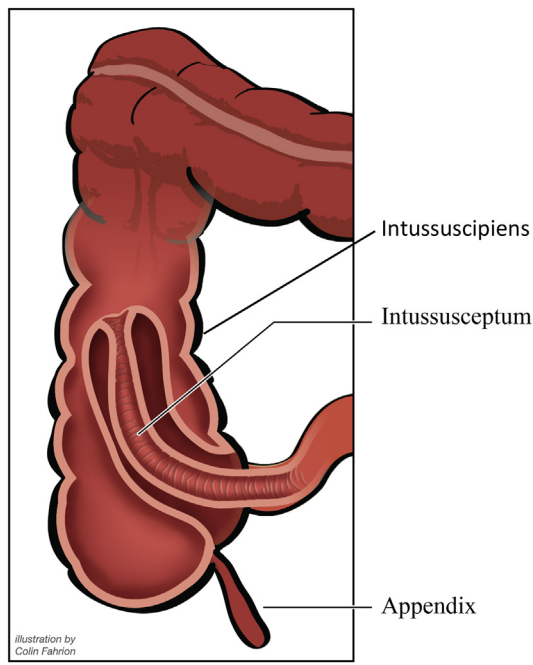

Fig. 1. Ileocolic intussusception.

intussusception is most common in children between 3 months and 3 years of age. For unclear reasons, intussusception is slightly more common in boys.[28] There is an increasing body of evidence implicating the role of viral infections as the trigger for idiopathic intussusception. The putative lead point in idiopathic intussusception is attributed to hypertrophied Peyer's patches in the lymphoid-rich terminal ileum (**Box 2**).

Secondary Intussusception

An intussusception may have an identifiable abnormality in the intestine that acts as a lead point, drawing the proximal bowel into the distal bowel by peristaltic activity. These so-called secondary intussusceptions account for 10% to 25% of intussusceptions. A pathologic lead point is seen more commonly in children younger than 3 months and older than 3 years.[28,32] A Meckel diverticulum is the most common pathologic lead point followed by polyps and enteric duplication cysts. A summary of pathologic lead points is listed in **Box 3**.

CLINICAL MANIFESTATION

Presentation

Intussusception occurs most commonly in healthy, well-nourished infants and toddlers. The typical presentation is the sudden onset of severe, intermittent, crampy abdominal pain. The child is often writhing in pain and crying inconsolably, drawing the knees toward the abdomen seeking a position of comfort. The episodes can resolve as quickly as they begin and usually recur at 15- to 30-minute intervals with increasing intensity. Vomiting is a common symptom and may become bilious as the obstruction progresses. With time, the child becomes dehydrated and lethargic. Initially, bowel movements may result from evacuation of stool distal to the intussusception. Later in the course, the stool may become mucoid and blood tinged as the ischemic mucosa sloughs. It is a diagnostic pitfall to wait for these so-called currant jelly stools, as this is a late sign and is absent in nearly one-third of patients. The classically described triad of pain, a palpable sausage-shaped abdominal mass, and currant jelly stools is seen in less than 15% of patients.[33] Particularly in infants, lethargy can be the only presenting symptom, thus, intussusception should be included in the evaluation of unexplained altered consciousness.[34]

Physical Examination

Early in the course of intussusception, the child's vital signs are usually normal. When in pain, the child is often writhing and difficult to examine. During painless intervals, the

Box 2
Evidence implicating viral trigger in idiopathic intussusception

- Thirty percent of patients experience viral illness (upper respiratory infection, GI, otitis media) before intussusception.
- Incidence of intussusception varies directly with seasonal respiratory viral illness.[29]
- There is an 8-fold increase in intussusception among infants vaccinated with early form of rotavirus vaccine.[30]
- Recent adenovirus documented in up to 50% of intussusceptions.[31]

[a] *Rotashield*, the new generation of rotavirus vaccine, has not been implicated in intussusception.[30]

Data from Refs.[29–31]

Box 3
Pathologic lead points in children

Benign focal bowel abnormalities
- Meckel diverticulum
- Polyps
- Enteric duplication cysts
- Vascular malformation
- Foreign body

Malignant lead points
- Lymphoma
- Melanoma
- Lymphosarcoma
- Bowel tumor

Systemic diseases
- Henoch-Schonlein purpura resulting in small bowel wall hematomas
- Cystic fibrosis
- Crohn disease
- Celiac disease
- Hemolytic uremic syndrome

belly is typically not tender. An abdominal mass may be palpable or even visible in a thin child. Rectal examination may yield blood or bloody mucus. As the obstruction worsens and the intussusceptum becomes more ischemic, the child may become febrile, hypovolemic, and tachycardic. Hypotension and peritonitis are ominous findings owing to bowel perforation and sepsis, thus, prompt diagnosis, resuscitation, and operation are imperative for survival.

DIAGNOSIS

Laboratory Studies

No specific laboratory studies point to the diagnosis of intussusception. Late in the disease process there may be electrolyte abnormalities caused by hypovolemia and anemia. There may be a leukocytosis caused by gut ischemia.

Ultrasonography

Ultrasonography for the evaluation of intussusception has been in use since the 1970s and has become the method of choice for detecting intussusception at most institutions.[35] Ultrasonography is an ideal screening modality because it avoids radiation exposure, and in experienced hands has a sensitivity and specificity approaching 100%. The intussusception is usually detected in the right side of the abdomen. The classic finding is referred to as the *target sign*, in which concentric alternating rings of low and high echogenicity are seen, representing the layers of intestine and mesenteric fat within the intestine (**Fig. 2**). Ultrasonography can also detect the rare case of ileoileal intussusception and is effective at identifying a pathologic lead point when present. More recently, ultrasonography is used to monitor the success of intussusception reduction procedures.[36]

Abdominal Radiographs

Plain radiographs are often obtained as part of the routine evaluation of patients with abdominal symptoms (**Fig. 3**). Plain radiographs are much less sensitive than ultrasonography at detecting intussusception. Thus, if there is a high clinical suspicion

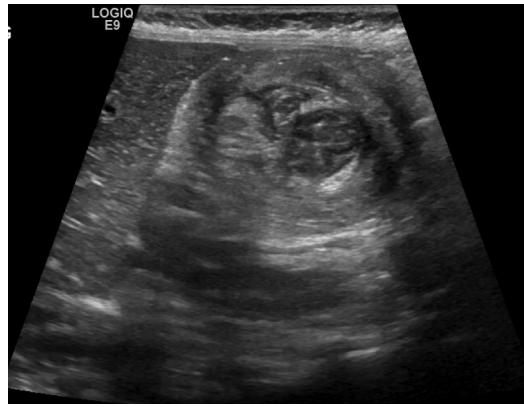

Fig. 2. Ultrasound scan of ileocolic intussusception shows the target sign.

for intussusception, ultrasonography, not plain radiographs, should be the first diagnostic maneuver. Radiographic findings suggestive of intussusception are listed in **Box 4**.[37]

Computed Tomography and MRI

An intussusception can be identified on both computed tomography (CT) and MRI. However, neither modality is routinely used to evaluate for intussusception because they cannot reduce the intussusception, often require sedation in young children, and can be time consuming. CT also exposes the child to a considerable dose of radiation. Small bowel intussusceptions that are detected on CT or MRI are usually transient and clinically insignificant.

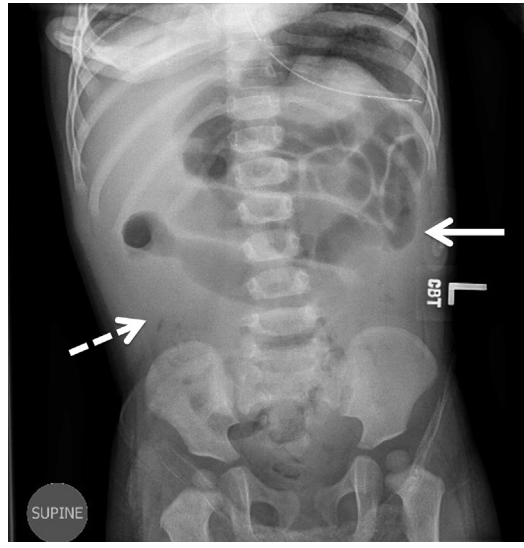

Fig. 3. Abdominal plain film shows concern for small bowel obstruction with multiple air-filled loops of dilated small bowel (*solid arrow*) with the absence of gas in the right lower quadrant (*dashed arrow*).

Box 4
Radiographic findings concerning for intussusception

- Signs of intestinal obstruction (dilated small bowel and air-fluid levels)
- Abdominal mass
- Target sign
- Absence of air in cecum
- Obscured liver margin
- Lateralization of the ileum

Contrast Enema

The availability of ultrasonographic expertise is limited at many institutions. Therefore, some institutions use contrast enema under fluoroscopy to establish the diagnosis of intussusception. In the presence of an intussusception, the study is converted to a therapeutic hydrostatic reduction of the intussusception.

TREATMENT

Stable patients with a high clinical suspicion or radiographic evidence of intussusception should undergo nonoperative reduction as the first-line therapy. Evidence of intestinal perforation, peritonitis, or hemodynamic instability is an absolute contraindication to nonoperative reduction and warrants an emergency operation. Before attempting reduction, the patient should be adequately resuscitated with intravenous fluids.

NONOPERATIVE REDUCTION

Nonoperative reduction of an ileocolic intussusception using hydrostatic or pneumatic pressure enema is effective and safe in children. Both reduction techniques can be performed under sonographic or fluoroscopic guidance. Although ultrasound scan avoids radiation exposure, sonographic and fluoroscopic guidance techniques have comparable success rates.[38] In practice, the choice between hydrostatic or pneumatic reduction and between sonographic or fluoroscopic guidance is largely dictated by the expertise of the individual performing the procedure.

Although the risk of intestinal perforation is only about 1%,[39] the surgical team should be notified that reduction is being attempted in the event of perforation or failed reduction. Bowel perforation during attempted pneumatic reduction can result in massive pneumoperitoneum and respiratory compromise from abdominal compartment syndrome. Emergency decompression of the pneumoperitoneum with an angiocatheter can relieve the compartment syndrome until the perforation and intussusception can be definitively repaired. The utility of antibiotics before reduction to prevent bacterial translocation or as preemptive treatment in the event of perforation has not been established.[40] Fevers are common after reduction of an intussusception because of bacterial translocation or the release of endotoxin and cytokines. The intussusceptum is edematous after reduction, which may itself act as a lead point and lead to recurrent intussusception in the short term. After successful reduction of an intussusception, the patient should be observed for 12 to 24 hours or until tolerating a normal diet.

Hydrostatic Reduction

Hydrostatic reduction was historically performed with barium under fluoroscopic guidance. Today, water-soluble contrast is commonly used because of the potential hazards of barium peritonitis in the event of intestinal perforation. The standard technique is to place a large, lubricated catheter into the rectum and tape the buttocks together to prevent leakage. Balloon catheters are generally avoided. As described in **Box 5**, the contrast reservoir is placed 3 feet above the patient to generate a hydrostatic column of pressure. Under fluoroscopy or ultrasound guidance, the contrast agent is instilled until a concave filling defect is seen (**Fig. 4**). Hydrostatic pressure is administered as long as the intussusceptum is reducing. Reduction is complete when contrast flows freely into the terminal ileum. Successful reduction rates range from 80% to 95%.[41]

Pneumatic Reduction

Pneumatic reduction of intussusceptions gained popularity in the 1980s. Air enemas reduce the intussusception more easily and are more effective than hydrostatic enemas. A recent meta-analysis showed the success rate of reducing an intussusception with an air enema was 83% compared with 70% for hydrostatic enema.[42] A large catheter is placed into the rectum and the buttocks are taped closed. A sphygmomanometer is used to monitor the intracolonic pressure, typically not to exceed 120 mm Hg. The colon is pressurized under fluoroscopy or ultrasonography, monitoring for the reflux of air into the terminal ileum and disappearance of the mass at the ileocecal valve that indicates complete reduction of the intussusception (**Fig. 5**). Carbon dioxide can be used instead of air because it is rapidly absorbed and may be less uncomfortable.

Several studies found improved reduction rates with delayed, repeated attempts at nonoperative reduction in clinically stable patients. In one large series, delayed repeated attempts at reductions were made in 15% of cases and were successful in 50% of cases.[43] Protocols call for a 30-minute to 24-hour interval between attempts at reducing an intussusception.[44,45]

RECURRENCE

Intussusception recurs in approximately 10% of patients after successful reduction. The recurrence rate is similar for the different modes of reduction. About one-third of recurrent intussusceptions occur within the first 24 hours, often after initiation of feedings.[46,47] The parents are most closely attuned to the signs of intussusception in their child and should be on the lookout for evidence of recurrence. Recurrence does not mandate operative reduction. Each recurrence should be treated as if it were an initial episode of intussusception.[44,48] However, multiple recurrences should heighten the concern for the presence of a pathologic lead point (**Box 6**).

Box 5
Rule of 3s for hydrostatic reduction

1. Hydrostatic column 3 feet above the patient
2. No more than 3 attempts at reduction
3. Each attempt less than 3 minutes long

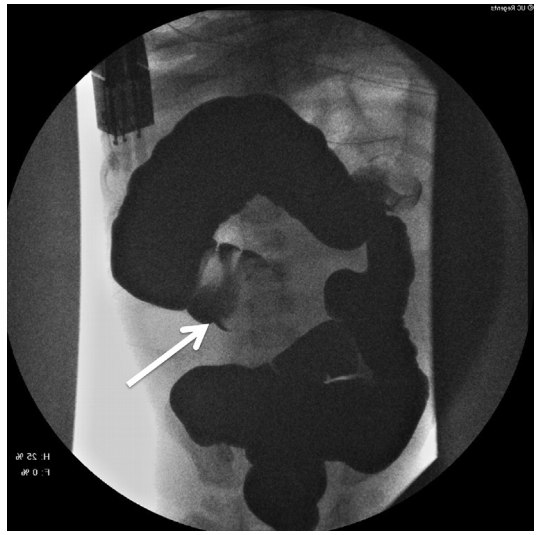

Fig. 4. Hydrostatic contrast enema. Arrow shows the leading edge of the intussusceptum.

OPERATIVE TREATMENT

Open Reduction

Operative reduction of the intussusception is indicated when enema reduction has failed, there is peritonitis, or there is evidence for a pathologic lead point. An open exploration is typically performed via a transverse right lower quadrant incision, as intussusception most commonly involves the terminal ileum and cecum. The classical teaching is to avoid pulling the intussusceptum and intussuscipiens apart but rather push the leading edge of the intussusceptum from distal to proximal. Once the

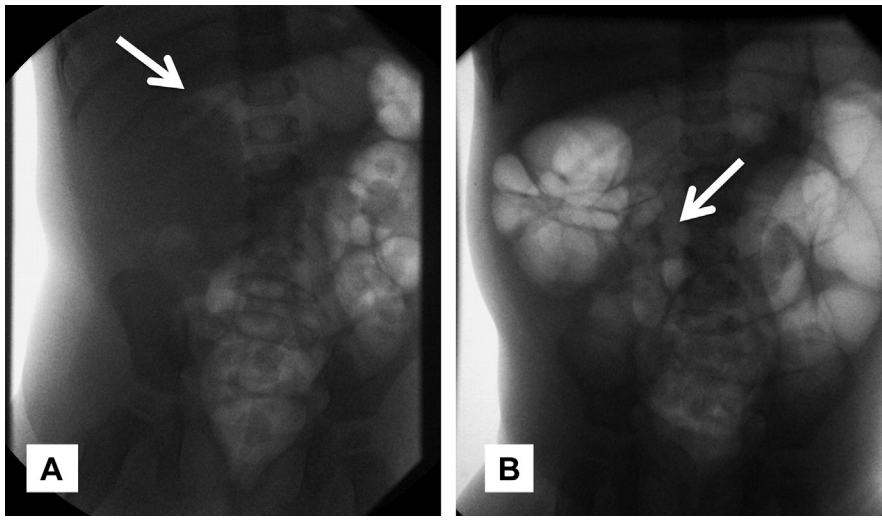

Fig. 5. Pneumatic reduction of intussusception. (*A*) Arrow shows the leading edge of the intussusceptum. (*B*). Successful reduction of intussusception. Arrow marks air in the terminal ileum.

Box 6
Clinical summary of nonoperative reduction of intussusception

- Pneumatic reduction is superior to hydrostatic reduction.
- Ultrasonography and fluoroscopy are comparable for guiding reduction.
- If initial attempts at reduction fail, delayed repeat enema may be successful.
- Intussusception recurs in about 10%.
- Each recurrence should be treated as if it were an initial episode of intussusception.

intussusception is reduced, the bowel should be evaluated for viability, perforation, and pathologic lead point. The bowel is generally ischemic and edematous. In the absence of a pathologic lead point, bowel resection is rarely required.[49]

Laparoscopic Reduction

Laparoscopy was initially used as a diagnostic adjunct in intussusception to either confirm the diagnosis or search for a pathologic lead point. Several recent studies describe the laparoscopic reduction of intussusception through 3 small abdominal ports.[50] Unlike the open operation, it is necessary to place gentle traction on the intussusceptum and intussuscipiens. It is important to evaluate for traumatic injury to the bowel after reduction. Little is lost by first attempting laparoscopic reduction. If this cannot be accomplished safely, the operation should be converted to a laparotomy. About 30% of attempted laparoscopic reductions are converted to laparotomy.[50,51]

SUMMARY

The differential diagnosis for LGIB in children is broad; however, the age of the patient helps narrow the diagnosis. Although most episodes of LGIB are benign and do not warrant hospitalization or surgical intervention, physical examination findings and the clinical condition dictate the need for further diagnostic tests and possible intervention. Intussusception is most common in children younger than 3 years. The diagnosis of intussusception is typically made with ultrasonography. Nonoperative reduction, either hydrostatic or pneumatic, should be attempted in a clinically stable patient without evidence of perforation, peritonitis, or hemodynamic instability.

REFERENCES

1. Pant C, Olyaee M, Sferra TJ, et al. Emergency department visits for gastrointestinal bleeding in children: results from the Nationwide Emergency Department Sample 2006-2011. Curr Med Res Opin 2015;31(2):347–51.
2. Balkan E, Kiriştioğlu I, Gürpinar A, Ozel I, Sinmaz K, Dogruyol H. Sigmoidoscopy in minor lower gastrointestinal bleeding. Arch Dis Child 1998;78(3):267–8.
3. Applegate KE. Evidence-based diagnosis of malrotation and volvulus. Pediatr Radiol 2009;39(Suppl 2):S161–3.
4. Long FR, Kramer SS, Markowitz RI, et al. Radiographic patterns of intestinal malrotation in children. Radiographics 1996;16(3):547–56 [discussion: 556–60].
5. Mehall JR, Chandler JC, Mehall RL, et al. Management of typical and atypical intestinal malrotation. J Pediatr Surg 2002;37(8):1169–72.
6. Ladd WE. Surgical diseases of the alimentary tract in infants. N Engl J Med 1936;215(16):705–8.

7. Emami CN, Petrosyan M, Giuliani S, et al. Role of the host defense system and intestinal microbial flora in the pathogenesis of necrotizing enterocolitis. Surg Infect (Larchmt) 2009;10(5):407–17.
8. Petrosyan M, Guner YS, Williams M, et al. Current concepts regarding the pathogenesis of necrotizing enterocolitis. Pediatr Surg Int 2009;25(4):309–18.
9. Lin PW, Stoll BJ. Necrotising enterocolitis. Lancet 2006;368(9543):1271–83.
10. Ng S. Necrotizing enterocolitis in the full-term neonate. J Paediatr Child Health 2001;37(1):1–4.
11. Muller A, Schurink M, Bos AF, et al. Clinical importance of a fixed bowel loop in the treatment of necrotizing enterocolitis. Neonatology 2014;105(1):33–8.
12. Amiel J, Sproat-Emison E, Garcia-Barcelo M, et al. Hirschsprung disease, associated syndromes and genetics: a review. J Med Genet 2008;45(1):1–14.
13. Badner JA, Sieber WK, Garver KL, et al. A genetic study of Hirschsprung disease. Am J Hum Genet 1990;46(3):568–80.
14. Austin KM. The pathogenesis of Hirschsprung's disease-associated enterocolitis. Semin Pediatr Surg 2012;21(4):319–27.
15. O'Connor JJ. Pediatric proctology. Dis Colon Rectum 1975;18(2):126–7.
16. Cserni G. Gastric pathology in Meckel's diverticulum. Review of cases resected between 1965 and 1995. Am J Clin Pathol 1996;106(6):782–5.
17. Sagar J, Kumar V, Shah DK. Meckel's diverticulum: a systematic review. J R Soc Med 2006;99(10):501–5.
18. Willetts IE, Dalzell M, Puntis JW, et al. Cow's milk enteropathy: surgical pitfalls. J Pediatr Surg 1999;34(10):1486–8.
19. Applegate KE. Intussusception in children: evidence-based diagnosis and treatment. Pediatr Radiol 2009;39(Suppl 2):S140–3.
20. Harrington L, Connolly B, Hu X, et al. Ultrasonographic and clinical predictors of intussusception. J Pediatr 1998;132(5):836–9.
21. Freedman SB, Xie J, Neufeld MS, et al. Shiga toxin-producing escherichia coli infection, antibiotics, and risk of developing hemolytic uremic syndrome: a meta-analysis. Clin Infect Dis 2016;62(10):1251–8.
22. Proulx F, Turgeon JP, Delage G, et al. Randomized, controlled trial of antibiotic therapy for Escherichia coli O157:H7 enteritis. J Pediatr 1992;121(2):299–303.
23. Sawczenko A, Sandhu BK. Presenting features of inflammatory bowel disease in Great Britain and Ireland. Arch Dis Child 2003;88(11):995–1000.
24. Thakkar K, Alsarraj A, Fong E, et al. Prevalence of colorectal polyps in pediatric colonoscopy. Dig Dis Sci 2012;57(4):1050–5.
25. Thakkar K, Fishman DS, Gilger MA. Colorectal polyps in childhood. Curr Opin Pediatr 2012;24(5):632–7.
26. Hirschsprung H. 107 falle van darmin agination bei kindern, behandelt inkoningin louisin-kinderhospital in Kopenhagen wahrend der jahre 1871-1904. Mitt Grenzgeb Medezin Chir 1905;14:555–74.
27. Mandeville K, Chien M, Willyerd FA, et al. Intussusception: clinical presentations and imaging characteristics. Pediatr Emerg Care 2012;28(9):842–4.
28. Buettcher M, Baer G, Bonhoeffer J, et al. Three-year surveillance of intussusception in children in Switzerland. Pediatrics 2007;120(3):473–80.
29. West K, Grossfeld JL. Intussusception. In: Wyllie R, Hyams JS, editors. Pediatric gastrointestinal disease: pathophysiology, diagnosis and management. Philadelphia: Saunders; 1999. p. 427.
30. Weintraub ES, Baggs J, Duffy J, et al. Risk of intussusception after monovalent rotavirus vaccination. N Engl J Med 2014;370(6):513–9.

31. Bines JE, Liem NT, Justice FA, et al. Risk factors for intussusception in infants in Vietnam and Australia: adenovirus implicated, but not rotavirus. J Pediatr 2006; 149(4):452–60.
32. Rubinstein JC, Liu L, Caty MG, et al. Pathologic leadpoint is uncommon in ileocolic intussusception regardless of age. J Pediatr Surg 2015;50(10):1665–7.
33. Yamamoto LG, Morita SY, Boychuk RB, et al. Stool appearance in intussusception: assessing the value of the term "currant jelly". Am J Emerg Med 1997; 15(3):293–8.
34. Kleizen KJ, Hunck A, Wijnen MH, et al. Neurological symptoms in children with intussusception. Acta Paediatr 2009;98(11):1822–4.
35. Ko HS, Schenk JP, Troger J, et al. Current radiological management of intussusception in children. Eur Radiol 2007;17(9):2411–21.
36. Di Renzo D, Colangelo M, Lauriti G, et al. Ultrasound-guided Hartmann's solution enema: first-choice procedure for reducing idiopathic intussusception. Radiol Med 2012;117(4):679–89.
37. Saverino BP, Lava C, Lowe LH, et al. Radiographic findings in the diagnosis of pediatric ileocolic intussusception: comparison to a control population. Pediatr Emerg Care 2010;26(4):281–4.
38. Hadidi AT, El Shal N. Childhood intussusception: a comparative study of nonsurgical management. J Pediatr Surg 1999;34(2):304–7.
39. Maoate K, Beasley SW. Perforation during gas reduction of intussusception. Pediatr Surg Int 1998;14(3):168–70.
40. Al-Tokhais T, Hsieh H, Pemberton J, et al. Antibiotics administration before enema reduction of intussusception: is it necessary? J Pediatr Surg 2012;47(5):928–30.
41. Flaum V, Schneider A, Gomes Ferreira C, et al. Twenty years' experience for reduction of ileocolic intussusceptions by saline enema under sonography control. J Pediatr Surg 2016;51(1):179–82.
42. Sadigh G, Zou KH, Razavi SA, et al. Meta-analysis of air versus liquid enema for intussusception reduction in children. AJR Am J Roentgenol 2015;205(5):W542–9.
43. Navarro OM, Daneman A, Chae A. Intussusception: the use of delayed, repeated reduction attempts and the management of intussusceptions due to pathologic lead points in pediatric patients. AJR Am J Roentgenol 2004;182(5):1169–76.
44. Lautz TB, Thurm CW, Rothstein DH. Delayed repeat enemas are safe and cost-effective in the management of pediatric intussusception. J Pediatr Surg 2015; 50(3):423–7.
45. Pazo A, Hill J, Losek JD. Delayed repeat enema in the management of intussusception. Pediatr Emerg Care 2010;26(9):640–5.
46. Daneman A, Alton DJ, Lobo E, et al. Patterns of recurrence of intussusception in children: a 17-year review. Pediatr Radiol 1998;28(12):913–9.
47. Gray MP, Li SH, Hoffmann RG, et al. Recurrence rates after intussusception enema reduction: a meta-analysis. Pediatrics 2014;134(1):110–9.
48. Niramis R, Watanatittan S, Kruatrachue A, et al. Management of recurrent intussusception: nonoperative or operative reduction? J Pediatr Surg 2010;45(11):2175–80.
49. Banapour P, Sydorak RM, Shaul D. Surgical approach to intussusception in older children: influence of lead points. J Pediatr Surg 2015;50(4):647–50.
50. Bonnard A, Demarche M, Dimitriu C, et al. Indications for laparoscopy in the management of intussusception: a multicenter retrospective study conducted by the French Study Group for Pediatric Laparoscopy (GECI). J Pediatr Surg 2008; 43(7):1249–53.
51. Apelt N, Featherstone N, Giuliani S. Laparoscopic treatment of intussusception in children: a systematic review. J Pediatr Surg 2013;48(8):1789–93.