An Occurrence of Sepsis During Inpatient Fecal Disimpaction

abstract

Functional constipation is a common pediatric problem that is often treated through well-established algorithms. Fecal disimpaction is the initial therapeutic step, and severe cases require hospitalization for intensive therapies. We describe a significant unexpected complication of this common clinical situation. An 8-year-old boy with suspected chronic functional constipation was hospitalized for disimpaction by continuous nasogastric administration of polyethylene glycol electrolyte (PEG-E) solution. On the sixth day of disimpaction, the patient abruptly developed fever, tachycardia, and tachypnea. Evaluation included blood culture, which grew Escherichia coli, and treatment with a course of appropriate antibiotics was provided. The safety of PEG-E solutions has been shown in studies of children with constipation, which made this patient’s illness surprising. Several potential etiologies of his infection were considered, including bacterial translocation (BT). BT is defined as the passage of live microbes and microbial products from the gastrointestinal tract to extraintestinal sites, such as the bloodstream. It has been shown to occur in a variety of clinical conditions but is of unclear clinical significance. In this case, physical damage to the intestinal mucosa was thought to contribute to the potential occurrence of BT, and prolonged disimpaction was considered as a risk factor. E coli sepsis in a child undergoing inpatient nasogastric fecal disimpaction with PEG-E represents a clinical problem never before reported in the literature and should increase clinicians’ indices of suspicion for uncommon complications of common procedures. Pediatrics 2014;133:e235–e239
Constipation is a common clinical problem encountered in pediatric practices. It may be simply defined as a delay or difficulty in defecation, present for ≥2 weeks. Rome III criteria for functional constipation have also been established for cases without an organic etiology. Once a diagnosis of functional constipation has been determined, treatment can be accomplished through well-established algorithms. Disimpaction of the stool burden must be performed before initiating maintenance therapy. This can be done orally, rectally, or through a combination of these methods. Although typically prescribed as outpatient therapy, specialty consultation with pediatric gastroenterology is indicated with treatment failures. In certain cases, inpatient hospitalization is necessary where therapies such as enemas, suppositories, and/or nasogastric (NG) administration of polyethylene glycol electrolyte (PEG-E) solutions may be used alone or in combination to accomplish adequate disimpaction. We describe a case that demonstrates a significant, unexpected complication of an inpatient bowel cleanout.

PATIENT PRESENTATION

Our patient is an 8-year-old boy admitted to the pediatric ward for fecal disimpaction after failing outpatient management of his chronic constipation and encopresis. His symptoms began after suffering verbal abuse during toilet training as a toddler before which he had a normal stooling pattern. Functional constipation was suspected given this history, and the patient’s symptoms met the Rome III criteria. A digital rectal examination revealed no strictures or masses other than palpable firm stool in the rectal vault, and stool was present throughout the colon on outpatient abdominal radiographs. This patient also carries diagnoses of attention-deficit/hyperactivity disorder and autism spectrum disorder including oral textural sensitivity that led him to reject all standard outpatient medications (polyethylene glycol, lactulose, magnesium citrate, mineral oil) with the occasional exception of senna. On admission a contrast enema was performed by pediatric radiology with Gastrografin diluted with 3 times its volume of water. This procedure assisted in evacuation of the large distal stool burden, and an abnormal dilation of the distal colon and rectum was noted as well (Fig 1). No stricture or mass was identified, although visualization of the colon was limited to the extent of the splenic flexure. An NG tube was then easily inserted, and an abdominal radiograph confirmed proper placement along with continued pancolonic stool burden without evidence of obstruction or perforation 4 hours after enema administration (Fig 2). A continuous infusion of PEG-E solution was then begun and gradually increased to the maximum pump rate of 300 mL/hr, which provided 80 mL/kg every 6 hours. This infusion rate was well tolerated and was continued until the patient’s stool cleared, which took 7 days. As our patient’s only tolerated home medication, senna was also provided during this admission to avoid undermining his established regimen.

During the first 6 days of hospitalization, intravenous fluids and a clear liquid per os diet were provided. Then out of concern for inadequate nutrition due to his prolonged lack of enteral intake, parenteral nutrition (PN) and intralipids (ILs) were begun through the patient’s previously established peripheral IV in the afternoon of hospital day 6. Later that evening the patient’s course was complicated by development of a fever of 40.4°C, tachycardia (heart rate 138), and tachypnea (respiratory rate 38). PN and IL were stopped because of the possibility of contamination, and cultures of these fluids and the patient’s blood were obtained. The vital sign abnormalities resolved within 3 hours, and the patient remained clinically stable on his disimpaction protocol. No abdominal discomfort was noted. The next day (hospital day 7), as the patient’s stools cleared, he was discharged with outpatient follow-up. The following morning (31 hours from blood draw), the patient’s blood culture grew Gram-negative rods, whereas cultures of the PN and IL remained negative. He was readmitted for additional evaluation and management.

FIGURE 1
Image taken during contrast enema demonstrating distal stool burden and abnormal rectal dilation.

FIGURE 2
Abdominal radiograph taken just before start of PEG-E infusion showing pancolonic stool burden with NG tube in place.
At the time of readmission, the patient reported fatigue, malaise, and 38.8°C fever since discharge. A repeat blood culture was obtained, an abdominal radiograph showed no free air or signs of megacolon, and he was empirically covered for Gram-negative rods with ceftriaxone. The patient quickly defervesced, and his initial blood culture grew Escherichia coli sensitive to ceftriaxone. After 48 hours, he was discharged on a 14-day course of oral cefdinir, and his repeat blood culture showed no growth at 5 days.

**DISCUSSION**

Effective treatment of constipation requires disimpaction before maintenance therapy, and use of high dose PEG-E solutions is an effective, widely recommended way to accomplish this goal.1–4,7 The safety of this method of disimpaction has been shown with only clinically minor adverse effects reported, including abdominal pain, nausea, vomiting, abdominal distension, fecal incontinence, and perineal irritation.4–7 Alterations of routine laboratory studies such as serum electrolytes and complete blood counts were clinically benign as well.4–6 It is notable that these solutions are also safe and effective in studies of chronic use for constipation.8–10 Thus, the occurrence of sepsis in the patient presented here is surprising, and his case represents a clinical situation not previously reported.

The source of the patient’s E coli sepsis was not decisively known. Given the temporal relationship between the initiation of the PN and IL infusions and the patient’s fever, there was initial concern that these infusates may have been contaminated. However, both were cultured and yielded no bacterial growth. Contamination of the blood sample sent for culture was considered as a potential confounder as well, but a false-positive result seemed unlikely given our patient’s clinical change when this blood was drawn. Enterocolitis or, in the setting of our patient’s severe constipation, Hirschsprung disease–associated enterocolitis were also considered but thought unlikely in the absence of vomiting, abdominal distension, or pain. The patient’s history and contrast enema results were also inconsistent with Hirschsprung disease, although he had not yet had a rectal suction biopsy or anal manometry. He also lacked localizing signs or symptoms suggestive of other bacterial process such as urinary tract infection, pneumonia, meningitis, or skin or soft tissue infection. Lateralization of bacteria into the bloodstream was possible but was not thought probable because our patient’s peripheral IV line was properly maintained per nursing protocol and was in good condition. No central line was present and no new venipunctures were performed in the hours before the onset of his illness. Given these considerations, our finding of viable enteric bacteria in the peripheral circulation and its correlation to the patient’s sepsis prompted us to reflect on bacterial translocation (BT) as a potential underlying etiology.11

BT is defined as the passage of live microbes and microbial products from the gastrointestinal tract to extraintestinal sites, such as the bloodstream.12 Multiple reviews of the literature have concluded that BT occurs in humans, although its clinical significance has been questioned.11,15–18 BT has been studied or described in humans in many varied clinical scenarios such as laparotomy,17,18 emergency and elective surgery,17–20 hematologic and colorectal malignancies,21,22 intestinal obstruction,23,24 burns,25 inflammatory bowel disease,26,27 aortic aneurysm repair,28 liver resection,29,30 obstructive jaundice,31,32 trauma,33–35 pediatric small bowel transplant,36 and surgical neo-

nates and infants receiving PN,37 among others. Several of these studies also related BT to increased morbidity,17–19,21,23–26,28,29,31,33,34,37 but others did not.20,22,27,30,32,35,38

A commonly proposed promotional mechanism of BT is physical damage to the barrier function of the intestinal mucosa leading to increased permeability.13,15,38 A stercoral injury resulting from our patient’s chronic fecal impaction could cause such damage directly, although such injuries are rare and typically seen in elderly, nursing home–dependent, bedridden, or narcotic-dependent patients.38 A mucosal injury sustained during rectal catheterization or enema instillation is also possible, although none was noted during our patient’s procedure. No manual disimpaction was performed. Our patient’s prolonged PEG-E disimpaction regimen may have conceivably led to such damage as well, especially given the important roles that luminal factors including mucus, gastric acid, pancreatic enzymes, and bile play in maintaining the host defense of the gut.39

**CONCLUSIONS**

To the best of our knowledge this case of E coli sepsis in a child undergoing inpatient NG fecal disimpaction with PEG-E represents a clinical problem never before reported in the literature. The etiology of this complication remains uncertain largely because of its unanticipated nature and subsequent lack of comprehensive evidence collection. Urine collection for analysis and culture would have been helpful to rule out urosepsis, and completing the evaluation for conditions such as Hirschsprung’s disease and anal atresia was previously noted would have better clarified our patient’s risk. Our patient’s disimpaction was also technically limited by the infusion pump used, which may have lengthened the
duration of his infusion and hospitalization. This pump’s maximum infusion rate could not reach the goal rate of 100 mL/kg every 6 hours set by our institutional disimpaction protocol or the 25 mL/kg/hr (up to 1000 mL/hr) recommended in the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition constipation guidelines. The use of senna during NG fecal disimpaction in this case is also nonstandard but was considered safe with a nonobstructive radiographic bowel gas pattern and reassuring abdominal examination. Future directions could include additional investigation into the effects of prolonged fecal disimpactions on intestinal barrier function and their safety in general as it has thus far been extrapolated from studies of shorter, less invasive procedures. It is conceivable that longer durations of inpatient disimpaction may increase the risk of complications such as was seen in this case. Overall, we hope to remind clinicians to maintain high indices of suspicion for potentially life-threatening complications during even common procedures such as inpatient fecal disimpaction.

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