

CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

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Case 12-2012: A 10-Month-Old Girl with Vomiting and Episodes of Unresponsiveness

Kenneth C. Sassower, M.D., Lauren M. Allister, M.D., and Sjirk J. Westra, M.D.

PRESENTATION OF CASE

Dr. Helen H. Yeung (Pediatrics): A 10-month-old girl was seen in the emergency department at this hospital because of vomiting and episodes of unresponsiveness.

The patient had been well until 2 a.m. on the day of admission, when she awoke with vomiting that was associated with irritability and frequent crying. During the next 7 hours, her oral intake decreased and four episodes of vomiting occurred, with nonbilious, nonbloody emesis (including formula after feeding). Episodes of unresponsiveness, which were unrelated to the episodes of vomiting, also occurred. During these episodes, her eyes were either open or closed, her head dropped, and she became limp, slumping over if she had been sitting. These episodes did not cluster, and she initially returned to baseline neurologic functioning after each episode. After the fourth or fifth episode, she became increasingly difficult to arouse, and the duration of the episodes increased from 2 to 3 seconds initially up to 30 to 60 seconds. Acetaminophen was administered at 5 a.m., without improvement.

The patient's mother called the pediatrician's office and was advised to go the hospital. Emergency medical services (EMS) personnel arrived at 9:01 a.m. The mother reported that the child had no recent illnesses, fever, or change in bowel habits or stool consistency or color. On examination by EMS personnel, the patient was awake but lethargic, and she was otherwise behaving appropriately for her age. She became agitated when her mother was not present, but was otherwise calm, without evidence of trauma. The pulse was 104 beats per minute, the respiration rate was 26 breaths per minute, and the lungs were clear. She was brought to the emergency department at this hospital at 9:20 a.m.

She had no fever, chills, cough, diarrhea, rash, generalized tonic-clonic movements, twitching, eye rolling or deviation, pallor or cyanosis, known head or other trauma, or toxin exposure or ingestions. Her most recent wet diaper was at 4 a.m. She was born by spontaneous vaginal delivery at 38 weeks' gestation; the pregnancy had been complicated by preeclampsia. The patient's birth weight was 2.85 kg (13th percentile) and length 45.7 cm (6th percentile). She had no jaundice in the newborn period, passed a bowel movement on the first day of life, and was fed formula from birth. She was initially followed by early intervention specialists because of decreased responses to auditory stimuli, but this follow-up had been discontinued a few months earlier; she had met other developmental milestones. At age 3.4 months, a diagnosis of gastroesophageal reflux disease was made, and lansoprazole was ad-

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ministered for 3 months, with resolution of symptoms. Immunizations were reportedly up to date. She had no allergies. Her parents were from South America and had recently separated. She lived with her mother and stayed overnight with her father intermittently, where she had been at the onset of the current symptoms, and she attended day care. Her mother had a history of migraines associated with intermittent “blackouts”; there was no family history of gastrointestinal illnesses.

The patient appeared somnolent and mildly difficult to arouse, and fussy, but consolable. The skin was pink, warm, and dry. The blood pressure was 110/70 mm Hg, the pulse 135 beats per minute, the temperature 36.3°C, the respiratory rate 36 breaths per minute, and the oxygen saturation 100% while she was breathing ambient air. Neurologic examination revealed a sleeping infant who was difficult to arouse but, once awake, had no neurologic abnormalities; there was no evidence of trauma, and the remainder of the physical examination was normal. The hematocrit and levels of hemoglobin, glucose, magnesium, phosphorus, total protein, albumin, globulin, and total and direct bilirubin were normal, as were tests of liver function; toxicology screening of the blood and urine was negative. Other results are shown in Table 1. Urinalysis revealed a specific gravity of 1.020 (reference range, 1.001 to 1.035), 1+ white

cells and ketones, trace albumin, and rare transitional cells; mucin was present, and the examination was otherwise normal. Electrocardiographic findings were normal. Fluids were administered intravenously; thereafter, she alternated between being alert, interactive, cooperative, and interested in toys and being torpid, fretful, and resistant.

Approximately 1.5 hours after her arrival, radiographic assessment of the patient’s chest was normal. Electroencephalography showed no evidence of seizure activity, and magnetic resonance imaging (MRI) of the head was normal. Her mother reported an additional episode of altered mental status at the hospital, which was not witnessed by hospital staff.

A diagnostic procedure was performed.

EMERGENCY MANAGEMENT AND DIFFERENTIAL DIAGNOSIS

Dr. Lauren M. Allister: I am aware of the diagnosis in this case, but Dr. Sassower is not. This 10-month-old girl presented to the emergency department with 7.5 hours of episodic vomiting and unresponsiveness.

The immediate approach to a patient such as this one who presents to the emergency department with altered mental status involves two steps. The first principle of emergency management is

Table 1. Laboratory Data.*

Variable	Reference Range, Age-Adjusted†	On Admission
White-cell count (per mm ³)	6000–17,500	19,400
Differential count (%)		
Neutrophils	17–49	78
Lymphocytes	67–77	19
Monocytes	4–11	2
Eosinophils	0–8	1
Platelet count (per mm ³)	150,000–450,000	483,000
Sodium (mmol/liter)	135–145	138
Potassium (mmol/liter)	3.4–4.8	5.2 (slightly hemolyzed)
Chloride (mmol/liter)	98–106	102
Carbon dioxide (mmol/liter)	22.0–27.0	21.4
Urea nitrogen (mg/dl)	5–20	11
Creatinine (mg/dl)	0.30–1.00	0.28
Calcium (mg/dl)	8.5–10.5	10.9

* To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the values for creatinine to micromoles per liter, multiply by 88.4. To convert the values for calcium to millimoles per liter, multiply by 0.250.

† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are age-adjusted and are for patients who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

evaluation of the ABCs (airway, breathing, and circulation). Added to this evaluation should be the letter D for dextrose (an initial glucose measurement), disability (assessment of the degree of neurologic compromise), and drugs (assessment of overt toxidromes due to ingestion or exposure). After the ABCDs are secured, the second step is to investigate the causes of impaired mental status. Mechanisms of such alterations in mental status include increased intracranial pressure, vascular changes or disease, metabolic derangement, exposure to toxins, abnormal neuronal discharges (i.e., seizures), and temperature irregularities.¹⁻³

Our initial assessment of this patient's ABCs revealed an intact airway, unlabored breathing with normal oxygen saturation, and good central and peripheral pulses and perfusion. A peripheral intravenous line was placed, she was given a bolus of normal saline, and continuous cardiac monitoring and oxygen administration were begun. A bedside glucose measurement was normal. Her mental status was fluctuating. There was no evident seizure activity or obvious toxidrome.

In searching for the cause of her altered mental status, our initial differential diagnosis included both primary neurologic diseases and non-neurologic diseases. Primary neurologic diagnoses included seizures, head trauma, infection (meningitis or encephalitis), or another primary process of the central nervous system (intracranial mass or hemorrhage). We were particularly concerned about either accidental or inflicted head trauma, because the patient had been staying with her recently separated father, who was not her primary caregiver, but there was no history or evidence of this. Non-neurologic diagnoses included accidental ingestion (of a prescription, over-the-counter, or herbal medication in the home), a metabolic derangement or disease, a cardiac arrhythmia or other underlying cardiac disease with resultant cerebral hypoperfusion, or evolving sepsis.¹⁻³

We cast a broad diagnostic net with testing that included a complete blood count, blood tests, liver-function tests, urinalysis, urine and blood cultures and toxicology testing, electrocardiography, preparation for obtaining cerebrospinal fluid by means of lumbar puncture, and MRI of the head. Intravenous lorazepam was kept at the bedside in case of seizure activity. Initial test results showed an elevated white-cell count, but levels of electrolytes, liver-function tests, toxicology screening, imaging of the head, electroencephalography, and

electrocardiography were all normal. Results of blood and urine cultures were pending.

In this patient, altered mental status was the primary persistent clinical feature, with vomiting a seemingly secondary symptom. She had no emesis in the emergency department, and serial abdominal examinations were benign. However, she continued to appear unwell. Her mother reported an additional episode of unresponsiveness that was not witnessed by the medical staff; this episode was self-limited and not associated with emesis. Neurologic consultation was obtained early in this patient's evaluation to expedite what we expected to be a neurologic diagnosis.

NEUROLOGIC ASSESSMENT AND DIFFERENTIAL DIAGNOSIS

Dr. Kenneth C. Sassower: The salient neurologic features of this case are the sudden bouts of nonbilious, nonbloody vomiting after feeding and episodes of unresponsiveness with loss of motor tone. The episodes of unresponsiveness occurred in a nonclustered manner, with the eyes either opened or closed, and with a rapid return to baseline neurologic functioning. The presence of a normal neurologic examination between episodes rules out an encephalopathy.

CLUES FROM THE HISTORY

Several key elements in the medical history and initial evaluation help to streamline the differential diagnosis. The success of early intervention in treating the infant's decreased responsiveness to auditory stimulation suggests that primary deafness and early autism are unlikely causes of her apparent altered mental status. In addition, the possibility of nonaccidental head trauma during the infant's stay with a recently separated parent was ruled out by means of normal neuroimaging.

The single most critical question regarding the clinical history is whether the recurrent bouts of vomiting and episodes of unresponsiveness were related. If the answer to this question is yes, we need to consider both gastrointestinal causes of unresponsiveness and neurologic causes of vomiting.

GASTROINTESTINAL CAUSES OF UNRESPONSIVENESS

The major gastrointestinal causes of unresponsiveness are those associated with an acute abdomen.⁴ In view of this infant's age and the presence of nonbilious vomiting, the most likely cause

of an acute abdomen would be intussusception.⁵ Pyloric stenosis is also in the differential diagnosis; however, this typically presents in the first several months of life. Intestinal malrotation or volvulus is possible, but these conditions are associated with bilious vomiting. An incarcerated hernia is ruled out by the presence of a normal abdominal examination. Porphyria can present with altered mental status and abdominal pain, but it is a rare consideration in infancy. Sandifer's syndrome — the occurrence of dystonic posturing of the head and upper limbs after eating — is associated with reflux or hiatal hernia, but it is not accompanied by altered mentation.

NEUROLOGIC CAUSES OF VOMITING

One always must consider increased intracranial pressure as a cause of lethargy and irritability in infants. Neurologic structural causes of vomiting include brain tumors (particularly choroid plexus papillomas) and other intracranial mass lesions (especially colloid cysts of the third ventricle), which may cause intermittent bouts of vomiting and headache due to an inherent ball-valve mechanism. These entities were ruled out with normal MRI results. In addition, the absence of a bulging fontanelle, increasing head circumference, and split sutures makes increased intracranial pressure unlikely. Vomiting is rarely a sign of increased intracranial pressure in infants, partly because of the presence of open fontanelles.

Other neurologic causes of vomiting include diffuse central nervous system infections (such as meningitis and encephalitis), hydrocephalus, cerebral edema, metabolic disturbances (such as hypoglycemia and inborn errors of metabolism), and migraine. Although vomiting can occur with seizures, it is rarely a primary ictal phenomenon on its own.⁶

One needs to consider paroxysmal nonepileptiform disorders as a cause of altered mental status and decreased motor tone (Table 2).⁷ An altered mental state with intermittent hypotonia may be due to syncope, and in this patient, syncope is a likely cause of the intermittent bouts of unresponsiveness. Vasovagal syncope is the most common subtype of syncope, and it is the cause of altered responsiveness in infants with cyanotic breath-holding spells and pallid infantile syncope. Other forms of syncope include convulsive, situational (after micturition and post-tussive), cardiac, and orthostatic syncope (which is rare in infants).

SLEEP DISORDERS

Intermittently altered mental states with diffuse hypotonia may reflect underlying sleep disorders. Primary pathologic forms of hypersomnia include narcolepsy with cataplexy, idiopathic central nervous system hypersomnolence, and the Kleine-Levin syndrome (recurrent bouts of intermittent hypersomnia), all of which are rare in infancy. In addition, sleep apnea syndromes, periodic limb movement disorder, and non-rapid-eye-movement arousal disorders are part of the differential diagnosis. However, in contrast to sleep-deprived adults, the most likely consequence of sleep deprivation in infants is irritability and inattention rather than somnolence.⁸ Paroxysmal unresponsiveness may also be caused by migraine and migraine equivalents, which would be extremely rare in infancy.⁹

EVIDENCE AGAINST A NEUROLOGIC CAUSE

Although some features of this case suggest a neurologic cause (head drop, a history of decreased response to auditory stimuli, and a maternal history of migraines with “blackouts”), a greater number of findings suggest a non-neurologic cause.¹⁰ These findings include unresponsiveness with eyes open; lack of stereotypy (the changes in mentation and duration of events are widely variable); lack of clustering of the events; the presence of atonic episodes without other major seizure subtypes (e.g., a mixture of tonic, myoclonic, and absence seizures, which occur in epileptic encephalopathies such as the Lennox–Gastaut syndrome); emesis precipitated by feeding; an unusually prolonged duration of episodic hypotonia; head drops with no description of rapid descent, slower ascent, or both; normal neurologic examination between episodes; normal neurodevelopment to date; and normal neuroimaging and electroencephalography.

NEUROLOGIC INTUSSUSCEPTION

The preponderance of evidence suggests a non-neurologic primary cause of unresponsiveness. If the vomiting is an associated feature rather than a coincidence, I suspect the cause is an acute abdomen with syncope, such as in so-called neurologic intussusceptions.¹¹ Numerous case reports, retrospective studies, and clinical reviews describe prominent neurologic features in the absence of common gastrointestinal symptoms of intussusception.¹² Although lethargy is most common, other neurologic symptoms include apathy, listlessness, hypersomnolence, impaired reaction to

painful stimuli, and fluctuating levels of consciousness. Neurologic intussusception is particularly common in infants.

The pathophysiology of neurologic intussusception is not well understood. Proposed mechanisms include release of neuropeptides from strangulated bowel, absorption of toxic metabolites, dehydration and electrolyte imbalance as a result of vomiting, and progressive bowel obstruction. In one study, endogenous opioid release was postulated after the patient had a positive response to naloxone administration.¹³ It is also possible that neurologic impairment is simply a normal behavioral reaction of an infant to severe abdominal pain.

In summary, I suspect that intussusception associated with syncope was the cause of this infant's intermittent unresponsiveness.

Dr. Nancy Lee Harris (Pathology): Dr. Allister, can you tell us what your thinking was at the time?

Dr. Allister: Our colleagues in neurology believed that there was probably not a primary neurologic disorder, in view of the negative findings on imaging of the head, normal electroencephalography, and the patient's overall appearance. At that point, our primary working diagnosis was an infectious process: either a central nervous system infection (meningitis or encephalitis) or evolving sepsis with associated altered mental status.

We then pursued infectious and other previously considered diagnoses, while continuing to support the persistently ill patient. Because of our concern for infection, we administered antibiotics intravenously. We prepared to perform a lumbar puncture and obtained portable chest and abdominal radiographs to evaluate for possible occult pneumonia or intraabdominal disease. We also planned to collect serum and urinary organic acids to assess for undiagnosed metabolic disease. We discussed the possibility of an exposure or ingestion of such substances as lead, heavy metals, or herbal medications not detected on routine drug screening, with plans for consultation with a toxicologist if the diagnosis remained unclear. We continued serial examinations, monitoring, and intravenous fluid administration.

As the investigations continued, one test result was brought to our attention as abnormal.

CLINICAL DIAGNOSIS

Altered mental status, probably due to infection (meningitis, encephalitis, or sepsis).

Table 2. Differential Diagnoses of Paroxysmal Nonepileptiform Disorders.

Causes of altered mental state

Syncope

Vasovagal (cyanotic breath holding, pallid infantile syncope)

Convulsive

Situational (after micturition, post-tussive)

Cardiogenic

Orthostatic

Sleep disorders

Narcolepsy

Idiopathic central nervous system hypersomnolence

Kleine–Levin syndrome

Sleep apnea syndrome

Periodic limb movement disorder

Non–rapid-eye-movement arousal disorder (sleepwalking, night terrors, confusional arousals)

Migraine

Classic migraine

Common migraine

Cluster headache

Migraine equivalents (confusional state; hemiplegic, ophthalmoplegic, or basilar migraine; benign positional vertigo)

Neuropsychiatric or psychogenic

Intermittent explosive disorder (episodic dyscontrol syndrome)

Hyperventilation attacks

Temper tantrums

Porphyria

Causes of altered motor tone

Decreased motor tone

Syncope

Sleep disorders (cataplexy)

Nonepileptic head drop

Increased motor tone

Sandifer's syndrome and gastroesophageal reflux disease (intact mentation)

Paroxysmal torticollis

Familial paroxysmal dyskinesias

DR. KENNETH C. SASSOWER'S DIAGNOSIS

Intussusception associated with syncope (neurogenic intussusception).

RADIOLOGIC DISCUSSION

Dr. Sjirk J. Westra: The diagnosis in this child was first suggested by our emergency radiologist's astute interpretation of a time-tested low-technology

examination: plain abdominal radiography (Fig. 1A and 1B). This test showed decreased air in the right colon, with a masslike lesion surrounded by radiolucent fat (a “target sign”) — features suggestive of intussusception. The reliability of abdominal radiography for the diagnosis or exclusion of intussusception has been debated.^{14,15} In this case, the target sign is very specific. Another highly specific sign for intussusception is the “meniscus” sign, which is caused by distal gas in the colon outlining the head of the intussusception, but these signs are not present in every case.¹⁶ Other typical findings include a paucity of gas in the right upper quadrant and a lack of delineation of the liver outline by gas in the colon, as seen in this patient. The lack of gas in the cecum is the most important sign, but it lacks sensitivity, particularly on radiographs obtained with the patient in the upright position; for this reason, this radiographic technique is no longer recommended. The sensitivity of the frontal radiograph (which is usually obtained with the patient in the supine position) can be increased by positioning the patient left side down (i.e., left decubitus position) or prone. With both maneuvers, the bowel gas can move into the right side of the colon in the absence of an intussusception.¹⁷ Together, these three radiographic positions increased the sensitivity to up to 100% in a study in which the images were interpreted by experienced pediatric radiologists.¹⁸ This sensitivity is in contrast to the reported 48% sensitivity of supine-only radiographic studies when interpreted by emergency department physicians.¹⁹ In this case, these additional views were not taken, since there was no stated clinical suspicion of intussusception.

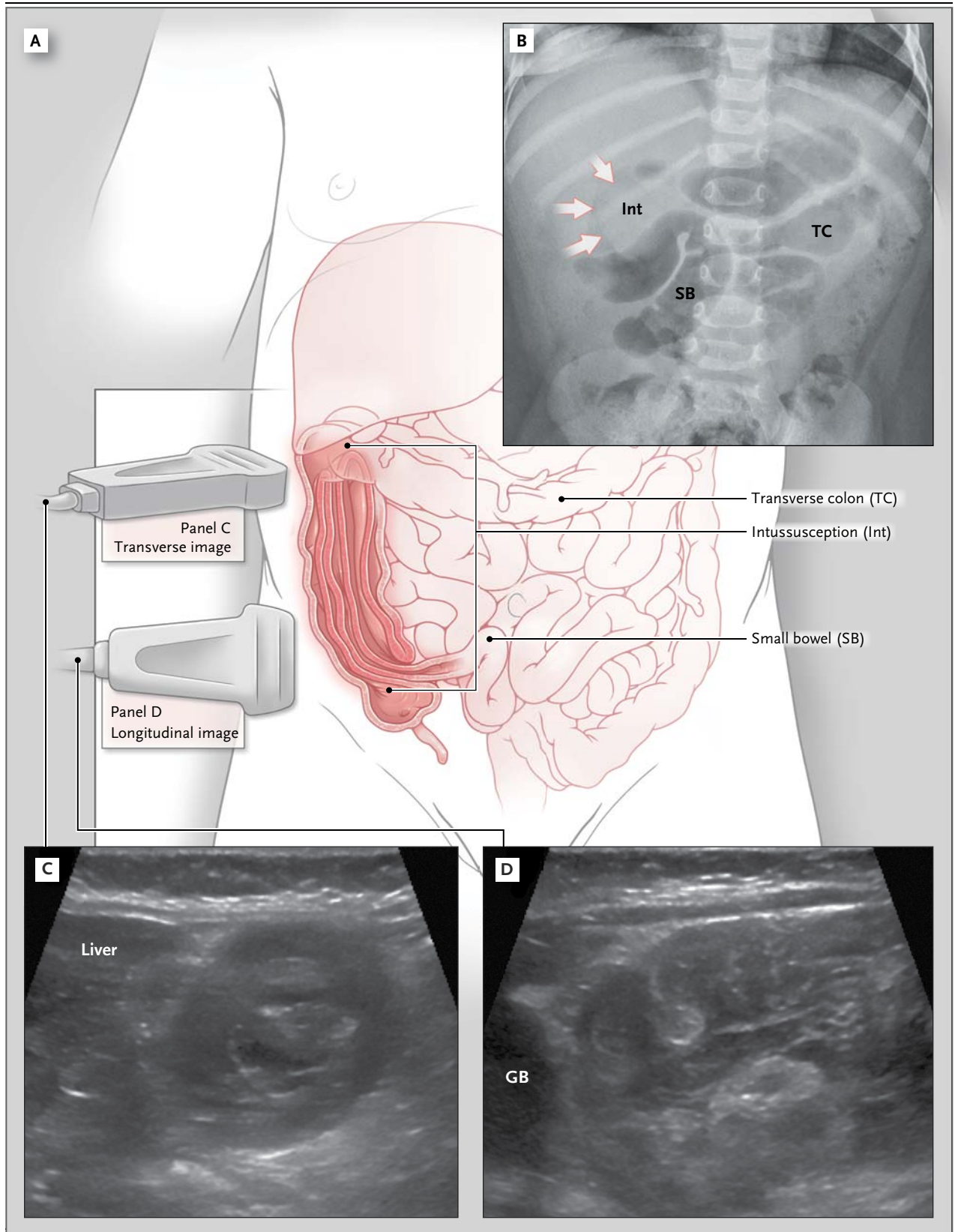
In summary, the above-described three-view abdominal radiographic series, when interpreted by an experienced radiologist, in conjunction with a careful clinical evaluation of the child, may safely be used to rule out an intussusception in facilities where high-level expertise in pediatric ultrasonography is not available around the clock.¹⁸ This fact is of practical importance, because in this era when medical images can be viewed online at a remote location, emergency department staff may obtain a timely expert interpretation even after hours, when patients with intussusception often present.

How sure can we be that the findings on plain radiography indicate the presence of intussusception in this patient? “Highly suggestive”

Figure 1 (facing page). Abdominal Imaging of the Intussusception.

The anatomy of the intussusception (Int), with the leading edge of a loop of terminal ileum terminating in the ascending colon, is shown (Panel A). Abdominal radiography that was performed with the patient in the supine position (Panel B) shows an abnormal pattern of gas in the bowel, raising suspicion of the presence of an intussusception. There is a lack of gas in the colon in the right upper abdomen, where there is a masslike appearance (Int). This mass can be detected because it is outlined by radiolucent peritoneal fat (the “target sign”) (arrows). Gas in the transverse colon (TC) does not extend all the way to the right abdomen to outline the liver shadow, as it normally does. The aerated bowel loop in the right abdomen consequently has to be small bowel (SB), which is mildly distended. Abdominal ultrasonographic images show the target sign of bowel within bowel in the right upper abdomen (Panel C) and a mass mimicking an extra kidney on the right (the “pseudo-kidney” sign of intussusception) inferior to the gallbladder (GB) (Panel D).

abdominal radiographic findings such as these are an important independent predictor of intussusception, in conjunction with rectal bleeding, a history of emesis, and male sex.²⁰ This patient did not have rectal bleeding and was female. In this case, pediatric radiology was called because of the suspicious radiographic findings, and we performed abdominal ultrasonography. Abdominal ultrasonography is more accurate than radiography for this diagnosis, and it has eliminated the need for diagnostic enemas.²¹ However, its correct performance requires the presence of a well-trained operator, ideally a pediatric radiologist. For this reason, I find that ultrasonography is best performed after plain radiography, both in cases such as this in which clinical suspicion is low but the radiographic findings are suggestive, and also in cases in which clinical suspicion of intussusception is intermediate but the radiographic findings are reportedly normal. If there is a low clinical suspicion of intussusception, initial radiographic findings may suggest other causes of the patient’s abdominal symptoms, such as benign infantile colic, gastroenteritis,²² constipation, or pneumonia in the lung bases, obviating the need for ultrasonographic examination. If there is a high clinical suspicion, it may be most efficient to proceed directly to ultrasonography followed by an enema to reduce the intussusception, but I would still obtain plain films, just before the enema, to look for signs of obstruction or free



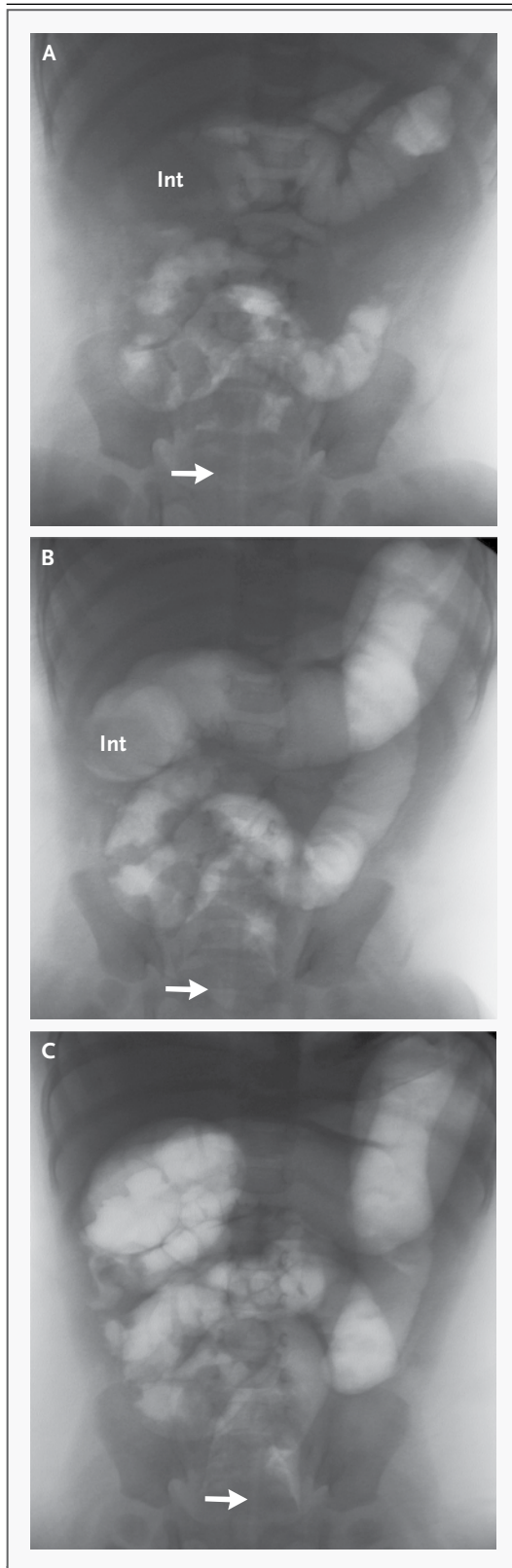


Figure 2. Pneumatic Reduction of the Intussusception.

During the pneumatic reduction, the intussusception (Int) was initially encountered in the proximal transverse colon (Panel A), and it was gradually reduced from the ascending colon with increasing pressures (Panel B). Panel C shows a complete reduction, with air flowing freely into the small bowel. A large, fluid-filled balloon (arrow in all three panels) in the rectum greatly facilitated the reduction.

intraperitoneal air. The latter finding is rarely encountered, because perforations are usually occluded by the intussusceptum, and they manifest only when one tries to reduce it. In this case, abdominal ultrasonography (Fig. 1C and 1D) confirmed the diagnosis of ileocolonic intussusception.

I then recommended a therapeutic enema to reduce the intussusception. Therapeutic enemas can be administered with either air (pneumatic) or fluid (hydrostatic), and there is controversy as to which method is best.^{23,24} Imaging guidance can be provided either with fluoroscopy or ultrasonography. Currently, air enemas under fluoroscopic guidance are typically performed in North America,²⁴ whereas in many parts of Europe and Asia, saline enemas with ultrasonographic guidance are favored.²⁵

I performed a pneumatic reduction with fluoroscopic guidance (Fig. 2). To create a closed conduit for a more effective pneumatic reduction, I placed a Foley catheter with a large fluid-filled balloon in the patient's rectum under fluoroscopic guidance. This technique enabled a safe inflation of the occluding balloon in the lowest part of the rectal ampulla, where it was supported by the extraperitoneal tissues, while avoiding inadvertent inflation of the balloon in the intraperitoneal sigmoid colon, where it might have caused a perforation. Contraindications for a therapeutic enema are peritonitis, shock, and perforation of the bowel, none of which were present in this patient. Nonetheless, a surgical consultation is always required and was obtained; the surgeon needs to know that the patient is in the hospital in case the reduction is unsuccessful, or if there is a complication. Small-bowel obstruction makes it more difficult to reduce the intussusception, but this is not a contraindication. Like most pediatric radiologists, I prefer not to administer sedation, because it becomes more difficult to assess

the patient's condition during the procedure, and a child who is old enough to cooperate will not be able to perform the Valsalva maneuver during the procedure, which may aid the reduction. The main risk of reduction is perforation (this risk in <1% in experienced centers). The procedure was performed without complications.

INTUSSUSCEPTION IN THE EMERGENCY DEPARTMENT

Dr. Allister: There were multiple challenges in arriving at the correct diagnosis in this patient, and it was an important learning experience. Altered mental status was the primary clinical feature; vomiting was initially perceived as secondary and did not continue in the emergency department. This patient did not have any of the many commonly described clinical symptoms of intussusception, including a palpable right-sided abdominal mass, vomiting, bloody or "currant jelly" stools, colicky or intermittent abdominal pain, drawing up of the legs toward the abdomen, and intermittent or inconsolable crying.²⁶ Nonetheless, fewer than a quarter of patients present with the classic triad of clinical symptoms of vomiting, abdominal pain, and bloody stools.²⁶ Intussusception is a common pediatric abdominal emergency, second only to appendicitis as the cause of an acute abdomen,²⁶ and it is the most common cause in children (such as this patient) who are younger than 2 years of age.²⁷ As emergency medicine physicians, we need to recognize that it can present with extraabdominal manifestations, including a spectrum of neurologic findings. Intussusception should be considered in young children and infants who present with altered mental status without a definitive neurologic cause.

Dr. Harris: Dr. Allister, can you tell us how the patient did?

Dr. Allister: The patient was admitted to the hospital overnight after the successful reduction. She was discharged the next day with a normal examination, tolerating feedings, and she appeared well. She had no further episodes of intussusception and no other intraabdominal problems.

A Physician: Was a rectal examination performed?

Dr. Allister: Initially, we believed the patient's presentation was secondary to a neurologic, not a gastrointestinal, cause. The result of a rectal examination would probably not have altered our primary investigations. As we have discussed, many infants and young children will not have a positive test for occult or gross blood on presentation with intussusception. However, had a rectal examination been done initially and been positive for occult or gross blood, it certainly could have focused our attention on the gastrointestinal system earlier in this patient's course.

FINAL DIAGNOSIS

Ileocolonic intussusception associated with syncope (neurologic intussusception).

Presented at the postgraduate course Primary Care Pediatrics (course directors: Peter T. Greenspan, M.D., Ronald E. Kleinman, M.D., John Patrick T. Co, M.D., M.P.H., Ronni L. Goldsmith, M.D., and Janice A. Lowe, M.D.), sponsored by Massachusetts General Hospital for Children and Harvard Medical School Department of Continuing Education.

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