A Neonate with Persistent Vomiting and Lethargy

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CASE PRESENTATION

History

A 9-day-old girl was brought to the emergency department (ED) at 6:15 PM by her parents. She presented with an 18-hour history of vomiting and lethargy.

The parents reported 8 episodes of vomiting, 2 of which were described as "forceful" and 2 others that occurred while the infant was asleep. The infant tolerated a bottle of formula at 10 AM (8 hours prior to presentation) but vomited after all other feedings. The emesis was nonbloody and nonbilious. The parents denied any gagging, choking during feedings, diarrhea, or fever. The parents further reported that the infant was sleepy for the last 18 hours and did not awaken for feedings. The parents reported only 2 wet diapers and 1 bowel movement (normal in color and consistency) on the day of presentation. The infant had a normal birth history and no previous medical or surgical history. There were no significant childhood illnesses in the family history.

Key point

This history (ie, lethargy, sleeping through feedings, persistent vomiting) suggests a potentially seriously ill child. Neonates presenting with such a history require aggressive diagnostic workup and management.

Physical Examination

Vital signs were as follows: rectal temperature, 37.6° C; heart rate, 144 bpm; respiratory rate, 44 breaths/min; blood pressure, 80/49 mm Hg. The infant's weight was 3.33 kg, up 60 g from birth weight. On physical examination, the infant was sleeping comfortably but was easily aroused. No dysmorphisms were noted. Head and neck examination revealed a supple neck, clear breath sounds, and no murmurs. Abdominal examination was significant for active bowel sounds on auscultation. On palpation, the abdomen was soft, nontender, and non-

distended; there was no hepatosplenomegaly and no palpable masses. Rectal examination revealed normal tone and no stool in the vault. The extremities were warm and well perfused, with 2+ femoral pulses and a capillary refill of less than 2 seconds. Neurologic examination showed normal tone and strength and normal Moro and suck reflexes.

Key point

At this point, the results of the physical examination in this patient are normal. Physical examination findings of concern in the neonate include: (a) abnormal vital signs (hyper- or hypothermia, apnea or tachypnea, tachy- or bradycardia, hypotension), (b) signs of dehydration (sunken anterior fontanelle, dry mucous membranes, mottled skin, delayed capillary refill), (c) poor weight gain (must be compared to birth weight; after initial weight loss, should gain 20–30 g/day), and (d) neurologic abnormalities (absence of primitive reflexes, decreased tone, irritability, tremors).

Laboratory Studies

Results of laboratory studies taken in the ED are shown in **Table 1.** Blood, urine, and cerebrospinal fluid (CSF) samples were sent for culture.

Key point

These laboratory findings are essentially normal. However, a negative laboratory profile (ie, normal peripheral leukocyte count and differential, normal CSF findings, normal glucose levels) in a neonate who is symptomatic

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is not as reassuring as it would be in an older child. For example, if this laboratory profile were seen in a child aged 4 months it would not prompt as much worry about sepsis. In a 9-day-old infant, however, sepsis must remain a concern.

- **What is the differential diagnosis for a neonate with persistent vomiting and lethargy?**

DIFFERENTIAL DIAGNOSIS

In formulating the differential diagnosis for this case, one must consider that it is common during the neonatal period for a child to "spit up" and to sleep frequently. However, 8 episodes of vomiting associated with decreased urine output and sleeping through feedings are outside the range of normal neonatal behavior. This child's history of lethargy, poor feeding, and persistent vomiting raises concern about sepsis, despite her normal physical examination and laboratory findings. Infection should be at the top of the list of differential diagnoses because of its prevalence and disastrous consequences if untreated in the neonatal period. Fever is not always seen in association with neonatal infections. Sepsis (causative organisms include *Escherichia coli, Listeria* sp., group B streptococcus), meningitis (*E. coli, Listeria* sp., group B streptococcus, herpes simplex virus, *Candida albicans*), pneumonia (group B streptococcus), urinary tract infections (*E. coli*) and necrotizing enterocolitis should all be considered.[1,2]

Gastrointestinal (GI) pathology must also be considered, given this child's persistent emesis. Vomiting outside of the immediate neonatal period should prompt a work-up for gastrointestinal anatomic anomalies that cause obstruction (eg, malrotation, volvulus, intestinal atresia or stenosis, incarcerated hernia) and those that do not cause obstruction (eg, gastric antral web, gastric duplication, hiatal hernia, hypertrophic pyloric stenosis, pylorospasm, and gastroesophageal reflux disease [GERD]).[1,2]

Other considerations include neurologic causes (eg, increased intracranial pressure secondary to hydrocephalus, hemorrhage, tumor, or trauma), renal insufficiency secondary to infection or obstruction, metabolic causes (eg, hypoglycemia, inborn errors of metabolism, congenital adrenal hyperplasia) or other common etiologies such as overfeeding and milk or soy protein allergy.[1,2]

CLINICAL COURSE
Admission and Initial Management

The patient was admitted to the hospital with a pre-

Table 1. Laboratory Values of Case Patient

Variable	Result
Blood	
Leukocyte count	$12.5 \times 10^3/mm^3$
Differential count	
Segmented neutrophils	15%
Lymphocytes	77%
Platelet count	$482 \times 10^3/mm^3$
Hemoglobin	13.0 g/dL
Electrolytes	
Sodium	139 mEq/L
Potassium	4.5 mEq/L
Chloride	101 mEq/L
Bicarbonate	20 mEq/L
Blood urea nitrogen	2.0 mg/dL
Creatinine	0.60 mg/dL
Glucose	82 mg/dL
Cerebrospinal fluid	
Leukocytes	$5/mm^3$
Erythrocytes	$17/mm^3$
Glucose	44 mg/dL
Protein	77 mg/dL
Gram stain	Rare white blood cells, no bacteria

sumptive diagnosis of neonatal sepsis. She was placed on ampicillin and gentamicin and intravenous fluids.

During hospital days 1 and 2, the patient did not tolerate any oral feedings with clear fluids. She had multiple episodes of clear emesis for 4 to 5 hours after each attempt at oral feeding. All emesis was nonbloody, nonbilious, and small in volume (approximately 15 mL).

The patient's physical examination remained unchanged (ie, afebrile with stable heart rate and blood pressure). Results of blood, CSF, and urine cultures remained negative. The continued lack of fever and the negative cultures ruled out the possibility of sepsis. These factors combined with the vomiting of all oral intake suggested GI pathology, specifically obstruction. Although GERD is common in the neonate, and the emesis was nonbilious, it is important to rule out obstruction first. An obstructive series was ordered and revealed a nonspecific bowel gas pattern but no evidence of obstruction and no air-fluid levels.

With no evidence of obstruction, other GI diagnoses were considered, specifically pyloric stenosis and GERD.
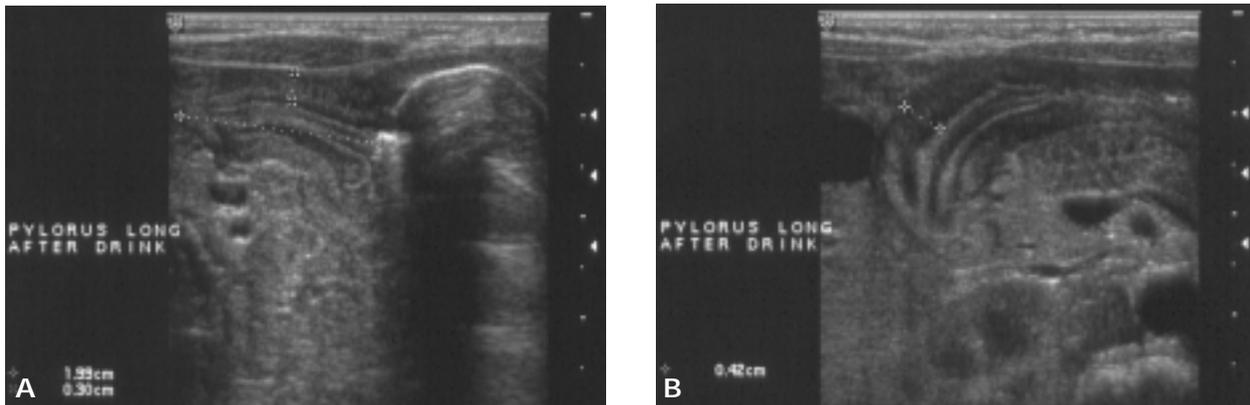
Figure 1. Ultrasound of case patient obtained on hospital day 3. (A) Pyloric length of 19 mm is visible. (B) Pyloric width (ie, muscle thickness) of 4 mm is visible. Both images are consistent with the ultrasound diagnostic criteria for pyloric stenosis.

An abdominal ultrasound was scheduled; an upper GI contrast study also was scheduled in the case of a negative ultrasound. The ultrasound obtained on hospital day 3 was consistent with the diagnosis of hypertrophic pyloric stenosis (**Figure 1**). Given this patient's normal electrolyte levels, she was brought to the operating room immediately for pyloromyotomy. Postoperatively, a feeding protocol was advanced and the patient was discharged on postoperative day 3 (hospital day 6).

- **What are the most important factors to know about the diagnosis and clinical course of pyloric stenosis?**
- **What are the components of the preoperative, operative, and postoperative management of a child with pyloric stenosis?**

HYPERTROPHIC PYLORIC STENOSIS
Definition

Hypertrophic pyloric stenosis is a GI disorder of infancy in which the pyloric musculature is hypertrophied, leading to constriction and obstruction of the gastric outlet. This obstruction leads to nonbilious emesis, dehydration, and alkalosis.[3] On pathologic inspection, the pylorus is grossly enlarged. There is hypertrophy of the circular muscle and increased connective tissue.[4]

Epidemiology

Pyloric stenosis occurs in 3 of every 1,000 live births. It is more common in whites of northern European ancestry, males (usually first-born males), and those whose mother had pyloric stenosis.[5]

Etiology

The cause of pyloric stenosis is unknown. However, there are several hypotheses about the etiology of this disorder, including the role of blood groups (type B and O blood groups), maternal stress (increased stress during the third trimester), and abnormal muscle innervation (causing muscle hypertrophy).[6]

Clinical Presentation

History. Nonbilious vomiting occurs in the first few weeks of life, most commonly between weeks 1 and 10. The vomiting may be projectile and is usually progressive. The vomitus may be bloody from associated gastritis. The infant is hungry after emesis. With progressive and more forceful emesis, the infant loses weight and becomes dehydrated.[4,5]

Physical examination. Assessment of the hydration status is imperative. Abdominal examination may reveal the hypertrophied pylorus (or "olive"). This mass is firm and mobile and is about 2 cm in length. The mass is usually located in the epigastric region, above and to the right of the umbilicus.[4–6]

Experienced examiners can palpate the olive in 85% of cases.[5,7] The olive may be missed if the infant has a distended stomach or is crying. Feeding the child may help relax the stomach musculature, making it easier to palpate the olive. After a feeding, there may also be a peristaltic wave across the abdomen of a child with pyloric stenosis.

Jaundice occurs in 2% to 5% of infants with pyloric stenosis and is likely secondary to decreased levels of glucuronyl transferase associated with the obstruction.[4,6]

Diagnosis

Laboratory examination. The presence and severity of electrolyte abnormalities depends on the duration of vomiting prior to diagnosis. Because of the closed

pylorus, both hydrogen and chloride ions are lost. In an attempt to compensate for volume depletion, potassium and hydrogen ions are excreted in the urine in exchange for sodium. Hypochloremic, hypokalemic metabolic alkalosis is a characteristic laboratory finding in pyloric stenosis. The alkalosis is usually profound, with serum chloride levels in the range of 60 to 75 mEq/L.[5]

Imaging studies. Abdominal radiographs may show a dilated stomach bubble, but this is not diagnostic for pyloric stenosis.

Ultrasound has replaced the upper GI contrast series as the diagnostic study of choice for 2 reasons: there is a risk of aspiration in a neonate who is vomiting and there is increased sensitivity of the ultrasound compared with the upper GI. Ultrasound is 90% sensitive in the diagnosis of pyloric stenosis. Diagnostic criteria include pyloric muscle thickness greater than 4 mm or an overall pyloric length greater than 14 to 16 mm, and a pyloric diameter greater than 14 mm.[4–6]

In the event of a negative ultrasound, an upper GI contrast study can help rule out other etiologies of vomiting (eg, GERD, malrotation). If the infant has pyloric stenosis, the upper GI contrast study will show an elongated pylorus, a bulge of pyloric musculature into the antrum (shoulder sign), and a single-string or double-tract sign (streaks of barium through a narrow pyloric channel).[6,7]

Key point

If evaluated early in the clinical course, pyloric stenosis may present without the "classic" findings of projectile vomiting, "olive" on abdominal examination, weight loss, and hypochloremic, hypokalemic metabolic alkalosis.

Treatment

Conservative medical management (ie, small frequent feeds and administration of atropine) has been previously attempted. However, it has resulted in slower improvement and a higher mortality rate. Surgical treatment after medical stabilization (with a focus on rehydration and correction of electrolyte abnormalities) is curative.[6]

Preoperative. Although not a surgical emergency, pyloric stenosis can be a medical emergency requiring aggressive fluid and electrolyte resuscitation. The patient should be kept without oral intake, and a naso- or orogastric tube can be used to empty stomach contents. Initial fluid boluses may be needed in a severely dehydrated child. Preoperative treatment is focused on rehydration and correction of electrolyte abnormalities. Fluid therapy should be continued until the child

is rehydrated and the alkalosis is corrected. Within 24 hours the patient is usually ready for surgery. The infant's laboratory values should be as follows: pH < 7.5, chloride > 88 mEq/L, bicarbonate < 30 mEq/L, potassium > 3.2 mEq/L.[4] Also, there should be adequate urine output (1 mL/kg body weight per hour).[4,5,7] Correction of alkalosis is imperative to prevent postoperative apnea and other complications.

Operative. First described in 1911, the Ramstedt pyloromyotomy remains the surgical procedure of choice. This procedure has a mortality rate of less than 0.4%.[4] Essentially, the pyloric mass is split without cutting the mucosa. This can be performed laparoscopically or through a small transverse incision. Surgical treatment is curative.

Postoperative. Postoperative vomiting occurs in about 50% of patients. This vomiting is usually secondary to pyloric edema at the site of the incision and should resolve over a 24-hour period. Persistent emesis after pylorotomy for pyloric stenosis is indicative of a second cause of obstruction (ie, GERD, incomplete pylorotomy).[4–6]

Although rare, postoperative complications include duodenal or gastric perforation, bleeding, superficial wound infection, and wound dehiscence.[4,5,7]

Although reestablishment of normal feeding patterns may take several days, initiation (after 24 hours postsurgery) of small, incremental feedings is encouraged. Most centers have a protocol for feeding advancement according to the patient's tolerance of oral feeding. The infant is usually discharged on postoperative day 2, when tolerating at least 4 oz of formula or breast milk every 3 to 4 hours.[5,7]   **HP**

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