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Successful management of pylorospasm with atropine in a failure-to-thrive neonate case report

Eric T Mendenhall, Lauren Rakes, Jamie Vernor, Madison Anderson and Susan Flesher

Abstract

Pylorospasm is an elusive diagnosis that can mimic the presentation of pyloric stenosis. There is limited discussion regarding its management in neonates with few case reports describing the use of antispasmodic agents. The following case reviews this management in a unique neonate. A 2-month-old female presented with persistent nonbilious, nonbloody emesis and failure-to-thrive. A thorough workup was performed due to its pronounced persistence while inpatient. Pyloric ultrasounds remained negative for pyloric stenosis; however, an upper gastrointestinal (GI) study was significant for pylorospasm. The workup also revealed hypothyroidism. Antispasmodic therapy with atropine was pursued as she was not a surgical candidate. Patient tolerated IV atropine therapy well with quick resolution of emesis and successfully transitioned to oral atropine therapy, displaying continued weight gain with exclusive oral feeds. This case displays a unique presentation of pylorospasm with successful management utilizing IV and oral atropine therapy in a neonate with failure-to-thrive and concomitant hypothyroidism.

Keywords

Pylorospasm, atropine, failure-to-thrive, neonatal, pyloric stenosis

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Introduction

Infantile hypertrophic pyloric stenosis (IHPS) has been a long-understood diagnosis with its classic clinical presentation of persistent non-bilious, non-bloody emesis, laboratory findings of hypochloremic, hypokalemic, metabolic alkalosis and distinct radiographic ultrasound diagnosis. In cases where these classic radiographic and/or laboratory findings are not evident, providers must remain diligent in assuring a proper diagnosis to pursue appropriate treatment modalities. One such diagnosis, pylorospasm, can appear with a near identical clinical presentation to pyloric stenosis (PS), however, does not typically align with the radiographic findings of PS. While the gold standard for diagnosis of PS is with a pyloric ultrasound, the diagnosis of pylorospasm is better observed radiographically with upper GI studies, as ultrasounds can be inconsistent and overlap within the diagnostic measurements of PS while never meeting all measurement criteria.[1,2] This has implications for treatment, especially in very ill patients, as the one presented here. With PS, surgical pylorotomy is the standard of care, whereas with pylorospasm, surgical intervention is not indicated. Treatment for pylorospasm, therefore, defaults to non-surgical options. There is a large amount of literature surrounding the usage of antispasmodic agents such as IV or oral atropine administration, as well as botulinum toxin injections; however, these were largely utilized in adult patients with a mixture of pylorospasm or gastroparesis diagnoses. Atropine has only been utilized in very few cases of distinct pylorospasm in neonates.[3] None have done so in patients with concurrent hypothyroidism, which is unique to our patient.

Department of Pediatrics, Marshall University, Joan C Edwards School of Medicine, Huntington, WV, USA

Corresponding Author:
Eric T Mendenhall, Department of Pediatrics, Marshall University, Joan C Edwards School of Medicine, 1600 Medical Center Drive, Suite 3500, Huntington, WV 25701-3655, USA.
Email: mendenhall8@marshall.edu

Case report

Presenting concerns

An otherwise healthy 2-month-old female initially presented to the Emergency Department with concerns of malnutrition and feeding difficulty at the request of their primary care provider (PCP). Patient presented with a history of a 0.5 kg weight loss over the previous 2 weeks, with an average weight gain of 22 g/day since birth accompanied by persistent projectile vomiting.

Medical history

The patient's birth history was significant for vacuum-assisted repeat C-section at 38.6 weeks to a 23-year-old G3P3. The patient's birth weight was 2.98 kg (32nd percentile). There were no adverse events noted during labor and delivery. Maternal medications included Flexeril, Synthroid, Diamox, Buspar, Cymbalta, and aspirin. Of note, the patient's state newborn screen was normal.

The mother reported continuous non-bloody and nonbilious emesis with feeds since birth that were at times projectile. Mother reported vomiting occurred immediately following every feed, a minimum of eight times daily. Mother attempted multiple formula changes during this time, listed chronologically: Similac advance [Abbott Nutrition], Similac sensitive [Abbott Nutrition], Enfamil Enspire, and Soy formula [Mead Johnson], with no improvement in symptoms. Additionally, the patient was trialed on famotidine (0.5 mg/kg daily) for 1 month prior to presentation without improvement. Thickened feeds or hydrolyzed formulas had not been attempted. The patient continued to have adequate daily wet diapers and one non-bloody bowel movement every 2 days with appropriate stool consistency. Two weeks prior to presentation, symptoms worsened, and the patient developed persistent projectile vomiting consistent with the full volume of feeds provided. Per mother, the patient had been taking 6oz every 1–2 h of soy formula for 2 weeks prior to presentation. Mother reported appropriate ratio of formula mixing without dilution. The patient underwent an initial pyloric ultrasound with their PCP 2 weeks prior to presentation, which was negative for PS with a pyloric length of 10 mm and wall thickness of 2 mm. A review of systems was negative for lethargy, fever, diarrhea, dark urine, bruising, extremity swelling, or seizure activity, although the mother did report no stools 3 days prior to initial presentation.

Initial assessment

Vital signs on presentation included temperature of 37.2°C rectally with a heart rate, respiratory rate, and O_2 saturation on room air, all within normal limits. Her weight was 4.225 kg (2.5 percentile for age on the WHO growth chart with a z score of −1.96).[4] Constitutionally, the patient was alert and in no acute distress. The patient was observed to have projectile emesis during physical exam, and volume was consistent with the full volume of formula given by parents. Ocularly, the pupils were equal and reactive to light with red reflex present bilaterally. The anterior fontanelle was open, soft, and flat; palate was intact, and oral mucosa was pink and moist. Capillary refill was brisk without delay. Gastrointestinally, the abdomen was found to be soft, nondistended, with normal bowel sounds. No abdominal masses noted. Neurological examination did not reveal any deficits.

Diagnostic assessment

Initial lab work demonstrated mild hyponatremia of 134 mEq/L. The remainder of labs were within normal limits, including a venous blood gas showing pH 7.44, pCO2 39 mmHg, HCO3 26.2 mmol/L, and base excess 2.2 mmol/L. A pyloric ultrasound was repeated and was negative for PS with pyloric length of 11 mm and wall thickness of 2.6 mm. Stool pH and stool occult blood were obtained to assess for nutritional losses or malabsorption problems in the GI tract as recommended for part of the failure-to-thrive workup per the American Academy of Pediatrics.[5]

The patient was started on volume-limited thickened feeds upon admission, however, continued to display significant amounts of vomiting with each feed. Head ultrasound was obtained due to the persistent vomiting, which showed an abnormal echogenicity with the sulci along the left parietal lobe; however, follow-up MRI of the head was normal. Initial presenting lab finding of hyponatremia resolved after initial fluid hydration, indicating dehydration as the expected cause. Patient received an upper GI swallow study, which revealed normal anatomy, negative for malrotation, volvulus, or atresias, and displayed a finding of pylorospasm. Additionally, the patient had not stooled for 3–4 days, and a glycerin chip was administered which yielded a large bowel movement, and lactulose was recommended. Multiple failed feeding attempts led to concerns of dehydration, and the patient received 24 h of IV maintenance fluids of D5 Normal Saline with 20MeQ KCL. A rapid increase in weight from fluids can be observed on inpatient growth chart per Figure 1. Additionally, given maternal hypothyroid status and history of constipation, Thyroid-Stimulating Hormone (TSH) and Free T4 were obtained, which showed an abnormally elevated TSH with normal free T4. Endocrine was consulted and recommended starting 25 mcg Synthroid daily. Patient continued to display persistent vomiting, including vomiting after taking small amount of enteral medications, therefore, not truly receiving these therapies. Speech evaluation was performed, and the patient displayed appropriate suck/swallow mechanisms with continued projectile vomiting.

Therapeutic intervention

Nasoduodenal tube was placed for meds and feeds in an attempt to evaluate pylorospasm as the root cause of symptoms. It was noted by nursing staff that ND tube was passed,

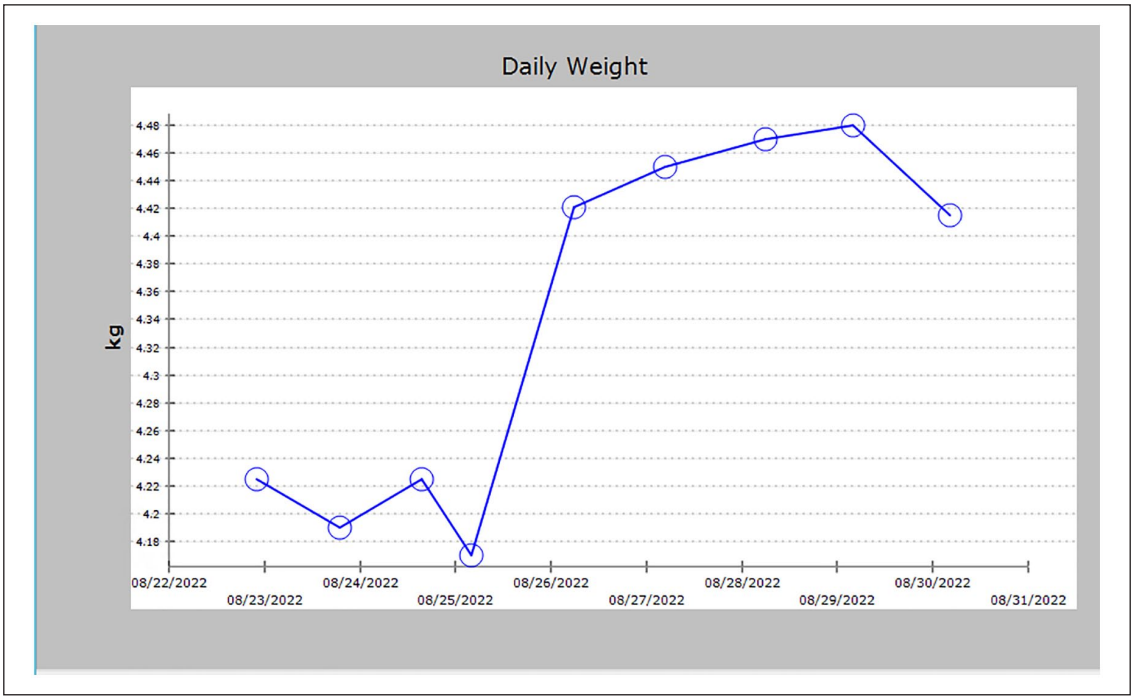

Figure 1. Daily weight curve of the patient starting from initial admission to the pediatric floor.

however, with a great deal of resistance. The patient tolerated feeds well through her ND tube without any regurgitation. An additional repeat ultrasound to assess for PS was again within normal limits, with pyloric length measuring 12 mm and muscle thickness 3 mm. Ultrasound was repeated as literature shows initial US for PS can be normal as hypertrophy can worsen with time. Pyloric thickness again increased, but only slightly by 0.4 mm on this repeat US, and again the patient still did not meet diagnostic criteria for PS. However, due to this slight increase, an additional US was obtained a few days later showing consistent measurements of 12 mm in length and 3 mm in thickness, making further progression unlikely. It is possible these initial discrepancies in size were secondary to differences in radiology reads and inner reviewer/imager variability.

Pediatric surgery was consulted regarding pylorospasm, and stated the case was not a surgical indication. Primary team subsequently pursued conservative management with an antispasmodic medication and started the patient on IV atropine for pylorospam, and switched to exclusively oral feeds. This was dosed and administered starting with 0.08 mg/kg/day IV, divided eight times daily before feeds. The patient continued on 3.5oz every 3 h of Sim Sensitive [Abbott Nutrition], as recommended by dietary for catch-up growth. The patient tolerated oral feeds well on the IV atropine with minimal spit-ups for 24 h and was subsequently transitioned to oral atropine. Atropine was given 5 min before each feed and dosed 0.08mg/kg/day divided eight times daily. The patient continued to feed well, however, incidentally acquired COVID during her hospital course, which caused her to have fevers with increased volume of spit-ups. She defervesced quickly after 12 h, and feedings again improved with minimal-to-no spit-ups on oral atropine. The patient tolerated this frequency and amount well on the oral atropine and gained nearly 200 g over the totality of their hospital stay with sustained daily growth on atropine, as per Figure 1. The patient was assessed as feeding, voiding, and stooling appropriately and was appropriate for discharge with continued atropine and Synthroid to be monitored and managed by PCP.

Patient follow-up

Patient's PCP was outside of our facility's network. However, endocrinology follow-up was established within our health network, and the patient had seen our endocrinologist regarding her management of hypothyroidism. The patient was 4 months removed from inpatient course during this encounter. She had gained almost 2 kg in the 4 months removed from hospitalization and her weight percentile per WHO was 8.8.[4] The patient still had slightly elevated TSH and was being continued on Synthroid 25 mcg at that time. Additionally, it was reported that she was still continuing her oral Atropine therapy, tolerating the medication and her feeds well.

Discussion

Given the infant's presentation the differential included PS, pylorospasm, gastroparesis, overfeeding, gastroesophageal

reflux (GER), and malrotation. Repeat ultrasounds as well as normal venous blood gas excluded PS, and observed feeds excluded overfeeding. Patient's symptoms persisted with reflux precautions and famotidine therapy, making GER less likely. An upper GI study excluded malrotation or volvulus, however, did note pylorospasm during the study. Pylorospasm remained the confirmed source of feeding difficulties after the implementation of atropine therapy resolved the patient's symptoms, whereas this would have exacerbated an underlying gastroparesis.

Persistent vomiting in infanthood is a relatively common phenomenon with a wide differential, some of which can be more immediately life-threatening, including anatomical causes, infectious causes, metabolic syndromes, and dietary intolerances. Forceful nonbilious vomiting with each feed in an infant around 2–3 months is suggestive of PS. This can typically be investigated efficiently with a thorough history, laboratory markers, and imaging modalities. Infantile hypertrophic pyloric stenosis (IHPS) may present as an infant who is a "hungry vomiter," clinically appearing emaciated and dehydrated, with failure to thrive and classically a palpable olive-shaped mass on examination. In differentiating pylorospasm from PS, the fact that our patient did not have the classic PS laboratory picture of hypokalemic, hypochloremic metabolic alkalosis is consistent with the less severe effects of pylorospasm despite presenting with dehydration and failure to thrive.

Further criteria leading to the diagnosis of pylorospasm in our patient was that the ultrasounds did not meet the criteria for PS. Diagnostic criteria include a pyloric muscle length of 15–19 mm or greater and a pyloric muscle thickness of 3–4 mm or greater on pyloric ultrasound, and our patient's thickness continued to measure between 2–3 mm and length never exceeded 12 mm. It has been observed with pylorospasm patients that a level of variability in muscle thickness can range from 2 to 3 mm, overlapping with IHPS thickness, while never reaching the full combination of measurements required for IHPS diagnosis.[1,2] The fact that there was variation in the measurements between our patient's three ultrasounds and yet none met criteria is consistent with a diagnosis of pylorospasm, and was also confirmed on upper GI.[6,7]

Differentiating pylorospasm from IHPS can be done using serial gastrointestinal fluoroscopy or ultrasonography, or an older method that can be used is upper GI.[3,8] Ultimately, differentiating between IHPS and pylorospasm focuses on a trial of atropine, which will be effective in pylorospasm, but not IHPS.[9] It could be argued that upper GI followed by a trial of atropine, as we did in our case is more cost-effective than serial fluoroscopy or ultrasound.

The condition of pylorospasm is when the pylorus experiences phasic contractions of increased tone with failure of sphincter relaxation, in contrast to IHPS in which the sphincter remains persistently hypertrophied with contractions, as has been observed on manometric studies.[10] Conservative management with medications have targeted this mechanism and have even been considered in cases of IHPS. This management includes nasoduodenal feedings, anticholinergics, famotidine, and atropine sulfate.[11–21] While targeting hyperacidity with famotidine to reduce pyloric contractility as a mode of treatment has been studied, our patient had been on famotidine for 1 month with no benefit.[11]

Atropine sulfate use in PS has recently been described and examined in small case series, small nonrandomized studies, and meta-analysis. Atropine use in pylorospasm, while widely used in adults with motility issues, has rarely been used in neonates.[2] It is believed that atropine sulfate relaxes the pyloric musculature acting as a competitive inhibitor of acetylcholine at muscarinic receptors, and has been observed with manometry inducing pyloric relaxation.[5,10] Starting IV dosing recommendations in one study ranged from 0.04 to 0.11 mg/kg/day and aided in the selection of the starting dose for our patient.[6] In areas that are resource starved, conservative management of IHPS with atropine has been used with success rates as high as 91.6% with minimal side effects, however, this medical management for PS could possibly only be temporarily effective. For this reason, our patient treated with atropine for pylorospasm must continue to be monitored on therapy outpatient.[12]

Conclusion

Negative ultrasounds in a persistently, projectile vomiting neonate can miss underlying dysfunction of the pylorus, particularly pylorospasm. Literature has shown atropine to be somewhat useful in IHPS; however, there are only rare studies utilizing atropine in infantile pylorospasm. Nowhere in the literature has there been a patient with diagnosed hypothyroidism and successful treatment of pylorospasm with atropine. This case report brings forth the possibility of using atropine sulfate as a treatment in a patient who presented with failure to thrive due to pylorospasm, also with concurrent hypothyroidism.

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Author contributions

E.T.M., L.R., J.V., M.A., and S.F. were responsible for the workup and management of this patient. J.V. was responsible for obtaining parental consent. E.T.M., L.R., and S.F. were responsible for the literature review. All authors were responsible for the production of the manuscript and contributed equally to reviewing as well as editing the manuscript.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

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ORCID iD

Eric T Mendenhall https://orcid.org/0009-0007-2133-0673

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