

Vomiting in a Neonate

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Citation

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Abstract

At 22 days of life, a girl presented with one day of vomiting after every feed. The vomit was milk colored, without bile or blood. Since birth, she had intermittent vomiting. She had no diarrhea, cough, congestion or fever. Her urine output was decreased. Case and diagnosis are discussed in the case report.

CASE

At 22 days of life, a girl presented with one day of vomiting after every feed. The vomit was milk colored, without bile or blood. Since birth, she had intermittent vomiting. She had no diarrhea, cough, congestion or fever. Her urine output was decreased. Also, her parents felt that her eyelids were puffy.

During pregnancy, her mother's alpha-fetoprotein was elevated. She was born at 38 weeks gestation by normal spontaneous vaginal delivery. The cord was wrapped around her torso, the amniotic fluid was meconium stained, and the placenta was enlarged. She had polycythemia which required an exchange transfusion, and her newborn thyroid screen was abnormal. The patient had no surgeries, was not taking any medications and had no allergies. She was of Caucasian background and had an older sibling.

On physical exam, her temperature was 98.8°F, heart rate 157 beats per minute, respiratory rate 47 per minute, 99% oxygen saturation on room air and weight 3.2 kilograms. She was interactive with her parents and the medical staff. She was normocephalic, and her anterior fontanel was enlarged and flat. Her eyes had red reflexes bilaterally, and her pupils were equal and reactive. Her mouth was moist and her neck supple. Her heart had a regular rate and rhythm with normal S₁S₂ and no murmurs. Her lungs were clear to auscultation with no crackles, rhonchi, wheeze or retractions. Her abdomen was soft, nondistended and without hepatosplenomegaly. Her extremities were warm, well perfused, and her capillary refill was less than 2 seconds. Neurologically, she was interactive with her surroundings and moved all extremities equally. No family members had pyloric stenosis.

This neonate's treatment included bloodwork, urine analysis

and an abdominal ultrasound. WBC was 11.0 tho/cmm, hematocrit 46.2% and platelets 637 tho/cmm. Her chloride was 106 mequiv/l, HCO₃ 28 mmol/l, BUN 2mg/dl and creatinine 0.1 mg/dl. Due to the hemolysis of several blood samples, her potassium level was unknown. Urine analysis showed 3+ protein and 3+ hemoglobin. Leukocyte esterase, nitrites and ketones were negative. The urine had 0-5 wbc's per hpf and 10-20 rbc's per hpf. PT was 10.7 seconds, APTT 35.1 seconds and INR 1.0. Total bilirubin was 0.4 mg/dl, AST 23 IU/l and ALT 14 IU/l. Protein was 3.2 gm/dl. Her T₄ was 1.07 mg/dl and TSH 6.15 IU/ml. Ammonia was 34 umol/l and lactate 2.4 mmol/l. Ultrasound showed that the pylorus was 19mm long with a wall thickness of 3.4mm. Also, the left kidney had moderate dilation of the renal pelvis and trace hydronephrosis.

DISCUSSION

This neonate presented with one day of vomiting after all feeds, and ultrasound diagnosed pyloric stenosis. Also, proteinuria and hypoproteinemia diagnosed her congenital nephrotic syndrome. If an abdominal ultrasound found a normal sized pylorus, then other causes of vomiting would have been considered including: infection, gastroesophageal reflux, overfeeding, formula allergy, structural abnormalities causing partial GI obstruction, inborn errors of metabolism, renal tubular acidosis, hydrocephalus, subdural hemorrhage and brain tumor. Some other causes of edema in a neonate include: heart failure, nephrotic syndrome, glomerulonephritis, renal failure, medications, liver failure, protein malnutrition and a protein losing enteropathy.

Pyloric stenosis (PS) is hypertrophy of the pylorus which causes nonbilious vomiting. It typically presents in infants 4-6 weeks of life beginning with vomiting after some feeds

which progresses to vomiting after all feeds. It occurs in 1 to 4 live births per 1,000 and has a male predominance of 5 males to 1 female [1]. There is a familial predisposition and a greater incidence in first born children. It is more common in children of mothers with PS than children of fathers with PS. [2] It more commonly affects Caucasians than Hispanics or African Americans. The incidence of PS is increasing in the US and other countries. [1] Also, erythromycin treatment has been linked with developing PS. [3] This Caucasian infant had nonbilious vomiting since birth and after all feeds for one day.

In an infant with PS, a palpable olive may be appreciated. The vomiting can cause alkalosis, hypochloremia and hypokalemia. Ultrasound is most commonly used to diagnose PS, with a criteria of muscle thickness of >4mm and pylorus length >16mm. [1] Premature or smaller weight infants with PS have been found to have similar muscle thickness but smaller pylorus length. [4] Surgical treatment is most common, and PS is the most common surgery in infants. [2] Prior to surgery, the acid-base balance, chloride and urine output are normalized. One theory about the cause of PS is hyperacidity. By reducing gastric acidity with atropine, relative underfeeding and gastric lavage, medical treatment as opposed to surgical treatment has been possible. There are other theories about the cause of PS including: diet, innervation to the pyloric muscle, molecular causes, immature ganglion function, increased gastrin levels and neurotransmitter Substance P. [1] In this infant an olive was not appreciated, and she did not have alkalosis or hypochloremia. Compared to a standard, her pyloric length was increased, 19mm vs. 16mm, while her muscle thickness was not, 3.4mm vs. 4mm. [1]

With the diagnosis of PS as the cause of her vomiting, the work-up might seem complete. However, her complicated birth history including polycythemia requiring an exchange transfusion and her facial swelling were also notable. So, another medical illness was considered. Proteinuria and hypoproteinemia diagnosed CNS in this infant. Two adults cases with nephrotic syndrome and polycythemia were found.[5,6] However, no infants or children were found with this association. Polycythemia does seem possible with nephrotic syndrome due to hemoconcentration caused by hypoproteinemia and edema. Since polycythemia occurs in newborns, another cause of polycythemia in a newborn might be overlooked. In a series of 41 patients with CNS, 12% presented with PS. The reason for this is unknown. [7]

CNS is a rare disorder presenting within the first three

months of life. It has a worse prognosis than nephrotic syndrome occurring after 1 year of life. [8] It is characterized by proteinuria, hypoalbuminemia, edema and hypercoagulability. CNS includes a heterogeneous group of renal diseases that cause increased glomerular permeability. The most common cause of primary CNS is the Finnish type (CNS-F). Other primary causes of CNS include diffuse mesangial sclerosis, minimal change nephrotic syndrome and focal segmental glomerulosclerosis. Secondary causes include syphilis, toxoplasmosis, cytomegalovirus, hepatitis B and C, human immunodeficiency virus, mercury intoxication, genetic malformation syndromes such as XY gonadal dysgenesis, systemic lupus erythematosus and amyloidosis. [8,9]

CNS-F is an autosomal recessive disorder with a defect in chromosome 19, the nephrin gene. This protein is associated with the glomerular filtration barrier. [8,9] Neonates with CNS-F often have large placentas, are born premature and are small for gestational age. Proteinuria occurs prenatally, and maternal serum alpha-fetoprotein is elevated. In families with CNS, maternal alpha-fetoprotein may prove helpful for prenatal diagnosis in future pregnancies. Edema often develops within the first week of life. Infants have wide cranial sutures, large open fontanelles, small noses, wide low-set ears and umbilical hernias. They feed and grow poorly secondary to protein loss, edema, pyloric stenosis and gastroesophageal reflux (GER). [9] Alpha-fetoprotein was elevated, and this infant had an enlarged placenta and an enlarged anterior fontanel. She may have had GER and had PS. However, she was not premature and was growing well.

Since most infants with CNS develop end stage renal disease, transplantation is necessary. Bilateral nephrectomy, peritoneal dialysis, a high-calorie / high-protein diet, vitamins, albumin and diuretics are utilized to reach a recommended weight for renal transplantation. [9,10] Treatment and observation help prevent and diagnose infections and thromboembolism early. Bilateral nephrectomy, prior to transplantation, has been standard treatment. [9,10] However, in a series of seven patients, transplantation at >2 years was possible with unilateral nephrectomy while transplantation at age 1 with bilateral nephrectomy. Unfortunately, CNS-F recurs in about 25% of patients after transplantation. [8] This infant had a bilateral nephrectomy at age 6 months and received peritoneal dialysis, albumin, immunoglobulin, diuretics, thyroid replacement and fluid restricted concentrated feeds to enable her to reach an age at which time renal transplantation would

be more successful. At 14 months of age, she received a deceased renal transplant and subsequently was treated with cellcept, prograf, valcyte, mepron and prilosec and had a minimum fluid requirement of 1 liter per day.

This infant's vomiting was due to PS. However, without consideration of her complicated newborn course and her facial edema, the diagnosis of CNS could have been missed. While children generally have only one illness, more than one can occur. When it is unlikely that the disease diagnosed could cause all of the abnormalities found, further work-up for another cause is necessary. In the end, it is hoped that an earlier diagnosis might lead to prompt treatment, decreased complications and improved outcomes.

References

1. Letton, Jr. RW. Pyloric Stenosis. *Pediatr Ann* 2001;30:745-50.
2. Hernanz-Schulman M. Infantile hypertrophic pylorus stenosis. *Radiology* 2003;227(2):319-31.
3. Patole S, Rao S, Doherty D. Erythromycin as a prokinetic agent in preterm neonates: a systemic review. *Arch Dis Child Fetal Neonatal Ed* 2005;90(4):F301-6.
4. Haider N, Spicer R, Grier D. Ultrasound diagnosis of infantile hypertrophic pyloric stenosis: determinants of pyloric length and the effect of prematurity. *Clin Radiol* 2002;57(2):136-9.
5. Balal M, Seyrek N, Karayaylali I, Paydas S. A unique form of polycythemia associated with minimal change disease. *Med Princ Pract* 2004;13(6):366-8.
6. Sonneborn R, Perez GO, Epstein M, Martelo O, Pardo V. Erythrocytosis associated with the nephritic syndrome. *Arch Intern Med* 1977;137(8):1068-72.
7. Mahan JD, Mauer SM, Sibley RK, Vernier RL. Congenital nephrotic syndrome: evolution of medical management and results of renal transplantation. *J Pediatr* 1984;105:549-57.
8. Papez KE, Smoyer WE. Recent advances in congenital nephritic syndrome. *Curr Opin Pediatr* 2004;16:165-70.
9. Penaflo G, Cheng T. Congenital nephrotic syndrome *Pediatr Rev* 2001;22:32.
10. Badoe EV, Kumoji R. Congenital nephritic syndrome of the Finnish type. *Ghana Med J* 2008;42(1):42-4.

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