A 3-day-old term infant presented to the emergency department for evaluation of bloody stools. The infant was born after an uncomplicated pregnancy followed by a normal spontaneous vaginal delivery. The mother was group B Streptococcus colonized, and received antenatal penicillin prophylaxis. The infant received routine delivery room care, and was given ophthalmic erythromycin and intramuscular vitamin K. Circumcision was performed without bleeding and he was discharged from the newborn nursery and the hospital after 48 hours. On the day of presentation, he had streaky bright red blood in 4 consecutive stools. After discussion with the infant’s pediatrician, the parents took him to the emergency department. The infant was afebrile, nursing well without emesis, and had made ~10 wet diapers that day. The physical examination revealed a fussy infant with mild tachycardia, tachypnea, and scleral icterus. The complete blood count was unremarkable. Serum total bilirubin was 11.9 mg/dL, sodium 156 mmol/L, chloride 120 mmol/L, potassium 4.7 mmol/L, and bicarbonate 16 mmol/L. International normalized ratio was prolonged at 2.7, prothrombin time 26.6 seconds, partial thromboplastin time 38.9 seconds. The stool showed a nonobstructed gas pattern. Official radiology interpretation the following day reported possible pneumatosis intestinalis in the left and right colon. Our multidisciplinary panel will discuss the assessment of bloody stools in the term newborn, evaluation of electrolyte abnormalities, the diagnosis, and patient management.

**CASE HISTORY WITH SUBSPECIALTY INPUT**

A 3-day-old term, male infant was referred to the emergency department by his pediatrician for evaluation of bloody stools. The infant was born via spontaneous vaginal delivery to a 26-year-old gravida 3 para 3 mother at 39 weeks. Pregnancy was complicated by group B Streptococcus colonization, with adequate antenatal prophylaxis. He was 3700 g at birth, and received routine care in the delivery room. Ophthalmic erythromycin and intramuscular vitamin K were given. He was exclusively breastfed, and voided and stools within 24 hours after birth. Circumcision was performed without bleeding and he was discharged from the hospital after 48 hours.

On the day of presentation, his parents noted bright red blood in 4 consecutive stools. The infant was afebrile, nursing for 10 to 15 minutes on each breast every 3 hours without emesis, and had made ~10 wet diapers in the previous 24 hours. His parents called his pediatrician, who referred him to the emergency department. Physical examination at the time of presentation revealed mild tachycardia and tachypnea: temperature 98.8°F, heart rate 164 bpm, blood pressure 109/68.

Dr. Josephsen led the initial conceptualization and design of this manuscript, recruited the other authors, and participated in the care of this patient; Dr. Bray-Aschenbrenner participated in this patient’s care, and conceptualized and drafted the initial manuscript; Drs. Feldenberg, Kirby, and Fitzpatrick participated in care of this patient, and contributed their subspecialty expertise during initial draft conceptualization; and all authors reviewed and revised the manuscript and approved the final manuscript in the form it was submitted.

**DOI:** https://doi.org/10.1542/peds.2017-0073

Accepted for publication Apr 28, 2017

Address correspondence to Justin B. Josephsen, MD, Department of Pediatrics, Saint Louis University, 1485 S Grand Blvd, St Louis, MO 63104. E-mail: jjosephs@slu.edu

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

Copyright © 2017 by the American Academy of Pediatrics

**FINANCIAL DISCLOSURE:** The authors have indicated they have no financial relationships relevant to this article to disclose.

**FUNDING:** No external funding.

**POTENTIAL CONFLICT OF INTEREST:** The authors have indicated they have no potential conflicts of interest to disclose.

90/60, respiratory rate 78, and blood oxygen saturation level 99%. His weight was 3000 g, 19% (700 g) below his birth weight. He was fussy but consolable. His fontanelle was soft and flat; his mucous membranes were moist, and there was mild scleral icterus. His heart sounds were normal without murmur. The abdomen was soft, non-distended, and did not appear tender to palpation. There was no organomegaly. He was mildly jaundiced with no petechiae. No anal fissures were seen. The complete blood count was unremarkable, with hemoglobin 20 g/dL, hematocrit 60%, platelets 210,000/L, and white blood cell count 12,600/L. Immature to total neutrophil ratio was 0.22. Total serum bilirubin was 11.9 mg/dL, sodium 156 mmol/L, chloride 120 mmol/L, potassium 4.7 mmol/L, creatinine 0.69 mg/dL, blood urea nitrogen (BUN) 15.6 mg/dL, and bicarbonate 16 mmol/L. Coagulation studies were abnormal: international normalized ratio (INR) 2.7, prothrombin time 26.6 seconds, and partial thromboplastin time 38.9 seconds. Blood, urine, and stool bacterial cultures were obtained. Lumbar puncture was deferred until the coagulopathy resolved. The stool was hemeoccult positive, however no Apt test was performed. An obstructive radiograph series of the abdomen showed a nonobstructed gas pattern without portal venous gas (Fig 1). The emergency department physicians read the radiograph as unremarkable, however formal radiology interpretation the following morning reported possible pneumatosis in the upper left and lower right quadrants (arrows).

The evaluation starts with a thorough history and physical examination. If breastfeeding, obtain a history of the frequency and duration of nursing and any evidence of cracked or bleeding areola. Stooling history in the newborn nursery may suggest the sequelae of GI anomalies like Hirschprung’s disease, which can be life-threatening if identification and treatment are delayed. The examination should be focused on vital signs, overall appearance of the infant, cardiovascular examination to evaluate congenital heart disease, abdominal examination, and genitourinary examination, looking for any anal abnormality.

Laboratories and imaging should target evaluation of the common and the deadly. A stool guaiac test will confirm the presence of blood. In young neonates, an Apt test may be performed to determine if blood (in stool or in emesis) is

---

**Dr Justin B. Josephsen (Neonatologist)**

Etiology of bloody stools in the neonate can range from benign to severely life-threatening conditions (Fig 2). Benign etiologies include swallowed maternal blood (from delivery or because of maternal nipple abrasions), anal fissures, and milk-protein allergy in the older infant. More severe conditions include infections such as bacterial or viral sepsis, and infectious colitis, gastrointestinal (GI) malformations, and necrotizing enterocolitis (NEC).

---

### Allergy
- Milk protein allergy

### Gastrointestinal atonic anomaly
- Anal fissure
- Hirschsprung’s disease with enterocolitis
- Malrotation with volvulus
- Intussusception
- Meckel’s diverticulum
- Vascular malformation
  - Arteriovenous malformation
  - Hemangioma

### Hematologic disorder
- Thrombocytopenia
- Vitamin K deficiency
- Inherited clotting factor deficiency
- Disseminated intravascular coagulopathy

### Exogenous source
- Swallowed maternal blood
  - Intrapartum
  - Cracked and/or bleeding nipples with breastfeeding

### Infection
- Neonatal sepsis
  - Bacterial (Group B Streptococcus, E. coli, Listeria)
  - Viral (herpes simplex virus, cytomegalovirus)
- Infectious enterocolitis
  - Bacterial (Salmonella, Shigella, E. coli)
  - Viral (cytomegalovirus, rotavirus, norovirus)

### Gut hypoperfusion
- Necrotizing enterocolitis
- Vasoactive drugs
- Congenital heart disease

---

**Dr Amelia Bray-Aschenbrenner (Pediatrics, Resident)**

Hematochezia in a newborn can be dramatic. When a newborn nursery or an emergency department calls for consultation, how do you guide the evaluation?

---

*Figures 1 and 2*
maternal or the infant’s. In this test, sodium hydroxide exploits the differing oxygen-binding affinities between adult and fetal hemoglobin. A complete blood count with differential, a complete metabolic panel, and blood, urine, and possibly spinal fluid cultures can evaluate organ function and infectious etiologies. Coagulation studies should be considered, but may be low yield. An abdominal obstructive series radiograph should be obtained to evaluate for evidence of pneumatosis intestinalis, portal venous air, or free abdominal air suggestive of NEC or bowel perforation.\(^3\) If an obstructive gas pattern is present, additional testing, such as an upper or lower GI series, would be indicated to determine the etiology of the obstruction.

This patient was nontoxic, however examination performed by the admitting attending physician the morning after initial presentation revealed a sunken fontanelle, continued mild tachycardia and tachypnea, and a capillary refill of 3 seconds despite fluid bolus in the emergency department. The serum chemistries and coagulation testing were highly abnormal and abdominal imaging demonstrated pneumatosis suggestive of NEC. He was made nil per os with intravenous (IV) fluids, given antibiotics, and pediatric surgery was consulted. Arterial blood gas at this time showed a mixed mild respiratory alkalosis with metabolic acidosis (pH 7.46, PaCO\(_2\) 27, PaO\(_2\) 55, base excess −4.1).

Dr Bray-Aschenbrenner

NEC is not typically a diagnosis associated with term infants. How does your approach to NEC differ in term versus premature neonates?

Dr Colleen Fitzpatrick (Pediatric Surgeon)

Term infants account for <10% of all NEC diagnoses.\(^3\)–\(^6\) The presentation is similar across gestational ages with bloody stools, feeding intolerance, and possibly an abnormal abdominal examination. The age of presentation is inversely related to the infant’s gestational age, so term babies tend to present within the first week of life, whereas preterm infants may present much later.\(^5\) The pathogenesis is thought to be related to gut mucosal dysfunction leading to bacterial translocation, injury, and ultimately bowel necrosis resulting in perforation, sepsis, and potentially death. In preterm infants, the etiology is thought to be immaturity of the gut mucosa and gut hypoperfusion related to prematurity. In term infants, however, NEC is associated with conditions that cause reduced mesenteric blood flow, such as sepsis, congenital heart disease, dehydration,\(^7,\)\(^8\) maternal drug exposure to vasoactive drugs (such as cocaine), or perinatal asphyxia.\(^5,\)\(^9\)

The treatment of NEC and the indications for surgery do not differ between the term and preterm populations. Pediatric surgery should be consulted and involved in the medical management, which includes extended bowel rest (10–14 days), antibiotics, and total parenteral nutrition with serial radiographic and physical examinations.\(^5\) Indications for surgical interventions include intestinal perforation and free air.

In this patient, pediatric surgery was consulted once the concern for pneumatosis was raised. He had a reassuring examination without evidence of free air, so he was medically managed. The coagulopathy raised a suspicion for herpes sepsis, although transaminases, white blood cell count, and platelet count were normal. Broad coverage IV antimicrobial agents were initiated with ampicillin, cefotaxime, metronidazole, and acyclovir. He was also given an additional dose of IV vitamin K and 10 mL/kg of fresh-frozen plasma with normalization of his INR. Lumbar puncture was then performed and did not suggest meningitis or herpes simplex virus infection. Pneumatosis resolved in ~24 hours, but the primary etiology of his NEC was still under investigation.

Dr Bray-Aschenbrenner

Infants with NEC can have electrolyte abnormalities including hyponatremia. However, this infant had an elevated sodium of 156 mmol/dL. In general, how should the general pediatrician approach the evaluation of hyponatremia?

Dr L. Richard Feldenberg, (Pediatric Nephrology)

The first step is to assess the volume status of the patient: is he hypovolemic, euvolectic, or hypervolemic? From there, one can investigate each of the differential diagnoses. Hypovolemic hyponatremia is relatively common in pediatrics due to inadequate water intake or free water loss from GI, renal, or insensible losses. These patients will have signs of dehydration, such as weight loss, tachycardia, and possibly low blood pressure. Hypervolemic hyponatremia may be because of salt poisoning from a high solute enteral formula, receiving a large volume of concentrated IV saline solution, or hyperaldosteronism. They will often have weight gain or high blood pressure. Euvolemia is the most difficult to assess because it is not as obvious as the other 2. Essential hyponatremia falls into this category.\(^9,\)\(^10\)

Our patient had several clues to etiology of his hyponatremia. First, he was breastfed. Breast milk has a low renal solute load compared with other mammalian milk. For example, mature human breast milk contains ~15 mg/dL of sodium, whereas cow’s milk has 3 times that.\(^11\) Therefore, it would be impossible for him to become sodium overloaded from this alone. His 19% weight loss
from birth suggests hypovolemia. Although minor fluctuations in weight may be attributed to differences in scales used for measurement, a 700 g weight loss over 3 days warrants additional investigation. At presentation, he was tachycardic and had developed more physical findings of hypovolemia with a sunken fontanelle and a capillary refill time of ~3 seconds when I examined him early the next morning, although he was being treated with IV fluids. Now the question is why is he hypovolemic?

**Dr Bray-Aschenbrenner**

Hypernatremia combined with his significant weight loss could suggest dehydration from breastfeeding failure. This is fairly common in breastfeeding neonates whose mother’s milk is not yet “in,” correct?

**Dr Feldenberg**

Yes, but he was making 10 wet diapers each day. An infant with dehydration from poor feeding would have significantly decreased urine output, and should have a rapid improvement in hypernatremia with IV rehydration. After this infant was given IV fluids, his sodium continued to trend upward. The fact that the infant did not respond to IV fluids suggests there could be renal water loss, such as in diabetes insipidus (DI), or extrarenal water losses as in children with severe diarrhea. A nephrologist can be helpful in recommending additional evaluations.

Serum BUN and creatinine can identify intrinsic renal disease. If these are normal, determining accurate urine output, along with additional blood and urine testing, can aid in determining the ultimate diagnosis. Our patient’s BUN and creatinine were high for his age with a BUN to creatinine ratio of 24, which is elevated. This suggests a prerenal etiology of decreased kidney function, such as dehydration.

The next step in our evaluation was to look at his urine electrolytes and osmolality and compare these with his serum osmolality, looking specifically for evidence of renal tubular dysfunction.8 Osmolality reflects how much solute is in a given fluid (eg, blood, serum, etc) and can be reported as a chemistry value or can be calculated by using the patient’s BUN, glucose, and sodium levels. In a patient with dehydration as a result of extrarenal water losses with otherwise normal kidneys, the renal tubules reabsorb as much water as possible. Therefore, the urine should be noticeably concentrated with a high osmolality, usually >600 mOsm/kg, whereas serum osmolality should be normal or slightly elevated.

**Dr Bray-Aschenbrenner**

Our patient was producing 17 mL/kg per hour of dilute urine with a specific gravity of <1.005. Additional studies revealed an inappropriately low urine osmolality of 243 mOsm/kg and an elevated serum osmolality of 339 mOsm/kg. This would support renal free water loss as the source of his hypernatremia and dehydration (Table 1), likely secondary to DI.

**Dr Feldenberg**

DI is diagnosed based on urine output of >40 mL/kg per day, a urine osmolality of <300 mOsm/kg, and a serum osmolality of >300 mOsm/kg.12 Patients with DI cannot reabsorb water in response to rising serum sodium values because of a loss of antidiuretic hormone (ADH). ADH is released from the posterior pituitary gland and acts on the collecting tubule of the nephron to increase free water uptake by binding to the V2 vasopressin receptor (V2R). This stimulates the insertion of aquaporin channels on the apical membrane in the tubule, resulting in the reabsorption of free water. If the hormone is absent, free water is lost in the collecting tubule, resulting in a triad of polydipsia, polyuria, and nocturia. Free water loss causes elevated serum sodium and osmolality with inappropriately dilute (low osmolality) urine. This is in contrast to polyuric patients without water loss, such as those with primary polydipsia, who have normal serum osmolality and appropriately low urine osmolality, as their body is excreting the excess water they consume.

Once the diagnosis is made, it is important to differentiate the type of DI: central or nephrogenic. The overall incidence of DI is 1:25 000, and the central form is far more common, accounting for ~90% of all DI.12 It occurs when the production or release of ADH is disrupted. This is usually secondary to some other insult to the brain, such as an inflammatory process like meningitis, neurosurgery, head trauma, tumor,

---

**TABLE 1 Pertinent Laboratory Values**

<table>
<thead>
<tr>
<th>Laboratory Values</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Admission laboratory work</td>
<td></td>
</tr>
<tr>
<td>Serum sodium</td>
<td>156 mEq/L</td>
</tr>
<tr>
<td>Serum creatinine</td>
<td>0.69 mg/dL</td>
</tr>
<tr>
<td>Serum BUN</td>
<td>15.9 mg/dL</td>
</tr>
<tr>
<td>Serum osmolality</td>
<td>339 mOsm/kg</td>
</tr>
<tr>
<td>Urine sodium</td>
<td>23 mmol/L</td>
</tr>
<tr>
<td>Urine osmolality</td>
<td>243 mOsm/kg</td>
</tr>
<tr>
<td>Urine specific gravity</td>
<td>&lt;1.005</td>
</tr>
<tr>
<td>Secondary laboratory work</td>
<td></td>
</tr>
<tr>
<td>Pre-DDAVP serum sodium</td>
<td>171 mEq/L</td>
</tr>
<tr>
<td>Post-DDAVP serum sodium</td>
<td>162 mEq/L</td>
</tr>
<tr>
<td>Pre-DDAVP urine osmolality</td>
<td>143 mOsm/kg</td>
</tr>
<tr>
<td>Post-DDAVP urine osmolality</td>
<td>127 mOsm/kg</td>
</tr>
</tbody>
</table>
or infiltrative process. Rarely, congenital DI can occur.

Nephrogenic DI (NDI) is much less common, accounting for ~10% of all DI, and is caused by an inability of the collecting tubule to respond to circulating ADH.\(^{12}\) It is most often a congenital disorder, presenting early in infancy with failure to thrive, emesis, and dehydration.\(^{13,14}\) Presentation in breastfed infants may be slightly delayed because of breast milk’s low solute load. NDI may also be secondary to certain medications such as lithium, haloperidol, or amphotericin B, as well as electrolyte abnormalities such as hypokalemia and hypercalcemia.\(^{12}\) It is important to carefully investigate and rule out these reversible causes, especially in patients with NDI without a family history.

**Dr Bray-Aschenbrenner**

How are the diagnoses of central and nephrogenic DI differentiated?

**Dr Feldenberg**

A water deprivation test can be performed in a controlled environment once sodium has normalized and the patient is well hydrated. The patient is not allowed any fluids while urine and serum osmolality and sodium are collected hourly. Patients with DI will have dilute urine despite rise in serum osmolality and sodium levels. Desmopressin (DDAVP) is then administered subcutaneously, and then urine and serum osmolality are measured again. Patients with central DI will display a decrease in urinary output, a rise in urine osmolality, and a fall in serum osmolality in response to DDAVP administration. Patients with NDI will change neither their urine output nor their serum or urine osmolality in response to DDAVP.

The water deprivation test is not recommended in infants because of the danger of severe dehydration and electrolyte derangements. Instead, our patient underwent a DDAVP challenge without water deprivation, to which he did not respond (Table 1). Additional questioning of the family revealed the diagnosis of NDI in a 4-year-old half-brother who presented at 6 months of age with emesis and dehydration. The diagnosis was certain at this point.

**Dr Bray-Aschenbrenner**

There are 3 ways NDI could be inherited. How do we know which form our patient and his half-brother have?

**Dr Amelia Kirby (Pediatric Genetics)**

There are multiple genetic inheritance patterns for NDI, each associated with different mutation genetic mutations. The X-linked mutation is the most common, accounting for 90% of NDI with an incidence of 4 to 8 per 1 million male live births.\(^{12,13}\) It causes misfolding of the V2R, which prevents ADH binding. Autosomal recessive mutations can also occur, which affect the Aquaporin 2 gene, the aquaporin channel which inserts into the apical membrane in response to vasopressin. The autosomal dominant form of NDI is associated with inability to shuttle the Aquaporin 2 gene to the apical surface and tends to have a milder phenotype than the X-linked and autosomal recessive forms.\(^{13,14}\)

Looking back over the family tree, his maternal half-brother is also affected; however, his maternal half-sister is not. Based on this information, we can be fairly confident that the mutation is inherited from the mother, an asymptomatic carrier. Genetic testing is the only way to be certain of this.

**Dr Bray-Aschenbrenner**

What is the implication for this family? Is genetic testing indicated and for whom?

**Dr Kirby**

This mother should be counseled that her male offspring have a 50% chance of being affected with NDI. Female carriers are typically asymptomatic, but in extreme stress situations such as marathon running, they may also become significantly dehydrated. When age appropriate, his half-sister should also be counseled that she may be an NDI carrier; however, testing is not generally offered until she is old enough to give informed consent.

Genetic testing begins with sequencing for the X-linked mutation in the V2R, as this is the most common defect.\(^{13,14}\) The patient’s half-brother was diagnosed with NDI 3 years before this patient presented; however, neither he nor his mother underwent genetic testing because of a denial of coverage by his insurance company. Genetic testing will not change how a patient is treated, but it may give families more insight into risks to future children and may lead to faster recognition of disease in siblings. Currently, no member of the family has been tested.

**Dr Bray-Aschenbrenner**

Based on his lack of response to DDAVP challenge, we can conclude our patient has nephrogenic DI, likely X-linked, given the family history. How do you treat this?

**Dr Feldenberg**

The treatment of central DI is straightforward because it is a deficiency of ADH. Exogenous DDAVP is typically the treatment strategy of choice. Doses are titrated to reduce the polyuria and associated nocturia to a manageable level. For NDI this strategy will not work, as the renal tubules cannot respond to DDAVP. Instead, treatment consists of thiazide diuretics and nonsteroidal antiinflammatory drugs, typically indomethacin. A low-sodium diet is also recommended to reduce the renal solute load. There are newer...
drugs in development, which act as protein chaperones to help correct the misfolding of V2R, thus allowing them to respond appropriately to vasopressin and recruit aquaporin channels. These drugs, however, are not yet available for clinical use.13,14

Dr Bray-Aschenbrenner
Managing water loss with a diuretic seems counterintuitive. How does that work?

Dr Feldenberg
Thiazides block sodium reabsorption in the distal convoluted tubule, so water is lost resulting in a relative hypovolemia (Fig 3). The nephron responds to this by increasing sodium reabsorption, and therefore water reabsorption, in the proximal convoluted tubule, resulting in a net positive uptake.15 It is unlikely to lower the urinary output to a normal level, but can decrease it enough that the child can keep up with his fluid intake.

As a second-line therapy, indomethacin is thought to increase the aquaporin 2 channels in the collecting tubule independent of vasopressin.14 Currently, our patient is being managed with just a thiazide diuretic, however he may need additional therapy in the future to control his water losses.

CASE WRAP-UP
In conclusion, we had a 3-day-old infant who presented with 1 day of bloody stools, found to have significant hypoperfusion secondary to dehydration as well as radiologic findings suggestive of NEC. Extensive evaluation for other potential causes of NEC, including full sepsis evaluation, was unrevealing. He was polyuric; urine and serum studies revealed inappropriately dilute urine and concentrated serum suggestive of free water loss. This suggests that dehydration, resulting from both free water loss and potential breastfeeding failure, may have led to mesenteric hypoperfusion, resulting in NEC. He was successfully treated with bowel rest, IV antimicrobial agents, and total parental nutrition. Family history was positive for presumed X-linked nephrogenic DI, which was ultimately diagnosed in him as well. His free water losses improved with thiazide monotherapy; indomethacin has been avoided because of potential GI side effects. He went home on a low-solute renal formula, as his mother did not wish to continue breastfeeding. He is followed as an outpatient with nephrology for management of his NDI and has had no additional issues with his GI system. NEC has been linked to dehydration in term infants in previous case reports; however, to our knowledge this is the first reported case of NDI presenting with NEC.

Dr Bray-Aschenbrenner
What additional information should his pediatrician know about his care?

Dr Feldenberg
He is now 10 months old and his growth is a major concern. Despite treatment for his DI and normalized electrolytes, he is falling off his growth curve and is now less than the third percentile for weight (75th percentile at birth). Children with DI would rather drink than eat because of extreme thirst, so failure to thrive and poor weight gain can be significant for them. He was recently changed from a low-solute formula to normal infant formula in an effort to improve his caloric intake. Often, a gastrostomy tube is needed to give them additional nutrition and fluids. These children are highly sensitive to any extra fluid losses so even febrile illnesses or viral gastroenteritis can result in severe dehydration and hospitalization. This patient will likely need a gastrostomy tube in the future, however, it may be removed once the child can eat enough on his own to grow.

Other complications include megaureter and megacystis secondary to copious urine production. Intellectual disability is sometimes present, likely from the effects of repeated episodes of dehydration of neurons and potentially rapid fluid shifts in the brain.14,16 The risk of neurodevelopmental delays underscores the importance of close monitoring by his general pediatrician and prompt management of even minor illness in these children.

ABBREVIATIONS
ADH: antidiuretic hormone
BUN: blood urea nitrogen
DDAVP: desmopressin
DI: diabetes insipidus
GI: gastrointestinal
INR: international normalized ratio
IV: intravenous
NDI: nephrogenic diabetes insipidus
NEC: necrotizing enterocolitis
V2R: V2 vasopressin receptor

FIGURE 3
Thiazide diuretics in the treatment of nephrogenic DI. Increased delivery of water and sodium to the proximal tubules after thiazide administration results in a net increase in overall water absorption.15

![Figure 3 Diagram]
REFERENCES

Bloody Stools in a 3-Day-Old Term Infant
Amelia Bray-Aschenbrenner, L. Richard Feldenberg, Amelia Kirby, Colleen M. Fitzpatrick and Justin B. Josephsen
Pediatrics 2017;140;
DOI: 10.1542/peds.2017-0073 originally published online August 10, 2017;

Updated Information & Services
including high resolution figures, can be found at:
http://pediatrics.aappublications.org/content/140/3/e20170073

References
This article cites 16 articles, 3 of which you can access for free at:
http://pediatrics.aappublications.org/content/140/3/e20170073#BIBL

Subspecialty Collections
This article, along with others on similar topics, appears in the following collection(s):
Fetus/Newborn Infant
http://www.aappublications.org/cgi/collection/fetus:newborn_infant_sub
Neonatology
http://www.aappublications.org/cgi/collection/neonatology_sub
Nephrology
http://www.aappublications.org/cgi/collection/nephrology_sub

Permissions & Licensing
Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
http://www.aappublications.org/site/misc/Permissions.xhtml

Reprints
Information about ordering reprints can be found online:
http://www.aappublications.org/site/misc/reprints.xhtml
Bloody Stools in a 3-Day-Old Term Infant
Amelia Bray-Aschenbrenner, L. Richard Feldenberg, Amelia Kirby, Colleen M.
Fitzpatrick and Justin B. Josephsen
Pediatrics 2017;140;
DOI: 10.1542/peds.2017-0073 originally published online August 10, 2017;

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://pediatrics.aappublications.org/content/140/3/e20170073