2024 PREP® Self-Assessment
Pediatrics Review and Education Program

Nephrology | PREP 2024

Divided by: Dr. Elaf AlShammari, Advanced General Pediatrics Fellow at NGHA, Riyadh

8 Questions | 21 pages

An 8-year-old boy is brought to the office for follow-up. He was seen 2 weeks ago for a sore throat, at which time he was noted to have an elevated blood pressure. Laboratory testing performed at that visit demonstrated normal renal function and an unremarkable urinalysis. Today, the boy's weight is at the 25th percentile and his height is at the 50th percentile for age. His heart rate is 88 beats/min, his respiratory rate is 16 breaths/min, and his blood pressure in the right arm is 128/88 mm Hg. Completion of 4 limb blood pressure measurements shows readings of 126/86 mm Hg in the left arm, 136/94 mm Hg in the left thigh, and 138/96 mm Hg in the right thigh. His physical examination findings are otherwise remarkable only for a bruit heard in the upper abdomen.

Of the following, the BEST next step in this boy's evaluation is

- A. dimercaptosuccinic acid scanning
- B. Doppler renal ultrasonography
- C. echocardiography
- D. magnetic resonance imaging of the adrenal glands

Correct answer is B

PREP Pearl(s)

- Renal disease and renovascular disease are the most common secondary causes of hypertension in children.
- Renal artery stenosis should be suspected in a child with stage 2 hypertension, significant diastolic hypertension, hypokalemia, abdominal bruit, and a kidney size discrepancy on renal ultrasonography.
- Doppler ultrasonography of the renal vessels is used to screen for renal artery stenosis. Computed tomographic angiography or magnetic resonance angiography are noninvasive tests used to diagnose renal artery stenosis.

Critique

The boy in the vignette has stage 2 hypertension, a normal upper and lower limb blood pressure gradient, and an abdominal bruit on physical examination. The best next step in his evaluation is to obtain Doppler renal ultrasonography to evaluate for renal artery stenosis, the most likely cause of his hypertension.

For children and adolescents, normal blood pressure levels are defined based on age, sex, and height. The American Academy of Pediatrics 2017 *Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children And Adolescents* (Flynn et al) defines normal blood pressure and the stages of hypertension as follows:

- Normal: <90th percentile
- Elevated: ≥90th percentile to <95th percentile
- Stage 1 hypertension: ≥95th percentile to <95th percentile + 12 mm Hg
- Stage 2 hypertension: ≥95th percentile + 12 mm Hg

The 95th percentile blood pressure for an 8-year-old boy (50th percentile for height) is 114/74 mm Hg. The boy's blood pressure in the office today is 128/88 mm Hg, which classifies him as having stage 2 hypertension.

Hypertension in children is further classified as primary (no identifiable cause) or secondary (with identifiable cause). Primary hypertension is usually asymptomatic, and often associated with obesity and a family history of hypertension. Children with secondary hypertension are usually younger than 6 years, have an elevated diastolic blood pressure, and exhibit signs and symptoms of the underlying cause. Causes of secondary hypertension in children are shown in Table 1. Renal disease and renovascular disease are the most common secondary causes.

The diagnostic evaluation for children with hypertension (Table 2) includes a detailed history, physical examination, urinalysis, basic metabolic panel (including renal function tests), lipid profile, and renal ultrasonography. Renal artery stenosis (RAS) should be suspected in a child with stage 2 hypertension, significant diastolic hypertension, hypokalemia, abdominal bruit, and a kidney size discrepancy on renal ultrasonography. Doppler ultrasonography of the renal vessels is used to screen for RAS in normal-weight children at least 8 years old who can cooperate with the procedure. Computed tomographic angiography and magnetic resonance angiography are noninvasive tests used to diagnose RAS. Renal arteriography is the reference standard for diagnosis of RAS.

A dimercaptosuccinic acid (DMSA) scan is indicated when renal scarring is suspected as a cause of hypertension. The boy in the vignette has normal renal function, an unremarkable urinalysis finding, and no history of urinary tract infections; therefore, a DMSA scan is not indicated. Coarctation of the aorta is suspected when the systolic blood pressure is 10 to 20 mm Hg higher in the upper extremity than in the lower. The boy in the vignette has a normal blood pressure gradient between his upper and lower extremities. Echocardiography will not identify the cause of this boy's hypertension, because there is no evidence of coarctation of the aorta on his physical examination, so this test is not the best next step in his evaluation. However, echocardiography may be indicated in the future to assess left ventricular mass, geometry, and function, as well as cardiac damage. Magnetic resonance imaging of the adrenal glands is indicated to diagnose pheochromocytoma, which is unlikely for this boy owing to the absence of episodic headache, tachycardia, and sweating.

Suggested Reading(s)

- Adam HM. Hypertension. *Point-of-Care Quick Reference*. *Pediatric Care Online*. American Academy of Pediatrics. April 6, 2020. Accessed September 1, 2023. Pediatric Care Online
- Flynn JT, Kaelber DC, Baker-Smith CM, et al. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. *Pediatrics*. 2017;140(3):e20171904. doi:10.1542/peds.2017-1904
- Weaver DJ Jr. Hypertension in children and adolescents. *Pediatr Rev*. 2017;38(8):369-382. doi:10.1542/pir.2016-0106

Content Domain

- Renal

ABP Content Specification(s) / Content Area(s)

- Recognize the clinical findings associated with hypertension
- Formulate a differential diagnosis of hypertension in patients of various ages
- Plan the initial clinical and diagnostic evaluation of hypertension

The correct answer is: Doppler renal ultrasonography

View Peer Results

Table 1. Common Causes of Secondary Hypertension in Children.

Causes	Examples
Renal	Congenital urologic disorders Glomerulonephritis Reflux nephropathy
Renovascular	Renal artery stenosis
Cardiac	Coarctation of the aorta
Endocrine	Cushing syndrome Hyperaldosteronism Hyperthyroidism Mineralocorticoid excess Pheochromocytoma
Medications	Corticosteroids Decongestants Stimulants
Environmental exposure	Cadmium Mercury
Others	Neuroblastoma Tuberous sclerosis

Courtesy of A. Jain

Table 2. Diagnostic Evaluation of Children With Hypertension.

Etiologic Origin	History	Physical Examination Findings	Studies
Renal parenchymal disease	• Failure to thrive • Family history of renal disease • Gross hematuria • History of oligohydramnios • Muscle weakness • Nocturia • Polyuria • Swelling • Urinary tract infections	• Edema • Pallor • Palpable mass • Short stature	• Blood urea nitrogen level • Complete blood cell count • Electrolyte levels • Genetic testing for monogenetic forms of hypertension (for consideration) • Renal ultrasonography • Serum creatinine level • Urinalysis
Renovascular disease	• Neonatal history of an umbilical artery catheter	• Abdominal mass • Adenoma sebaceum • Ash leaf spots • Café-au-lait spots • Carotid or abdominal bruit • Neurofibromas	• Angiography • Computed tomographic or magnetic resonance imaging • Renal ultrasonography with Doppler • Serum aldosterone level • Serum renin level
Endocrinopathies	• Flushing • Heat intolerance • Muscle weakness • Tremor • Weight loss	• Acne • Goiter • Hirsutism • Moon facies • Striae • Tachycardia • Virilization	• Adrenal imaging • Corticotropin • Cortisol level • Free thyroxine, thyrotropin • Plasma and urine steroids • Serum renin level • Serum aldosterone level
Primary hypertension	• Daytime fatigue • Family history of cardiovascular disease • Sedentary behavior • Smoking • Snoring • Weight gain	• Acanthosis nigricans • Increased body mass index	• Fasting lipids • Hemoglobin A_{1c} • Polysomnography
Iatrogenic	• Contraceptive pills • Decongestants • Immunosuppressants • Medical history • Stimulants		• Drug screen
Cardiac	• History of congenital cardiac disease • Shortness of breath	• Decreased pulses in lower extremity • Leg blood pressure 10 mm Hg lower than arm blood pressure	• Echocardiography

Adapted and reprinted with permission from Weaver DJ Jr. Hypertension in children and adolescents. *Pediatr Rev*. 2017;38(8):373.

A 1-day-old male neonate is evaluated in the nursery. He was born to a 25-year-old primigravida mother at 40 weeks' gestation via vaginal delivery. Prenatal ultrasonographic findings were significant for moderate hydronephrosis of the right kidney. He is breastfeeding well and has good urine output. The neonate's weight is 3.2 kg, and his length is 50 cm. He is vigorous and alert, and his vital signs are normal. His physical examination findings, including those of the genitourinary examination, are unremarkable.

Of the following, the BEST next step in this neonate's evaluation is to perform

- A. diuretic renography 2 days after birth
- B. renal function testing 2 days after birth
- C. renal ultrasonography 2 weeks after birth
- D. voiding cystourethrogram 2 weeks after birth

Correct answer is C

PREP Pearl(s)

- Common causes of prenatal hydronephrosis are transient physiologic hydronephrosis, ureteropelvic junction obstruction, vesicoureteral reflux, and ureterovesical junction obstruction.
- Neonates with prenatal unilateral hydronephrosis should undergo postnatal renal ultrasonography 1 to 2 weeks after birth.
- Neonates with prenatal bilateral hydronephrosis or a solitary kidney with hydronephrosis should undergo postnatal renal ultrasonography within 48 hours after birth.

Critique

The neonate in the vignette has unilateral moderate hydronephrosis demonstrated on prenatal ultrasonography. The postnatal ultrasonographic examination, in a case of unilateral hydronephrosis, should be performed when the neonate has nearly regained birth weight, usually 1 to 2 weeks after birth.

Hydronephrosis noted on prenatal ultrasonography has a reported incidence of 1% to 5%. The most common causes are transient physiologic hydronephrosis, ureteropelvic junction obstruction, vesicoureteral reflux (VUR), and ureterovesical junction obstruction. These and other common etiologies of unilateral and bilateral prenatal hydronephrosis are outlined in the Table. Infants and children with hydronephrosis are commonly asymptomatic but can present with urinary tract infection. Severe hydronephrosis can present with abdominal mass and/or hematuria. Bilateral hydronephrosis, such as that seen with posterior urethral valves, can lead to urinary incontinence, poor urinary stream, failure to thrive, and renal failure.

Renal function testing (eg, serum creatinine level) is not recommended in the first 7 to 10 days after birth for neonates with unilateral hydronephrosis and a normal contralateral kidney, as earlier testing will be reflective of the mother's creatinine level rather than that of the neonate. However, if bilateral hydronephrosis is present, renal function testing should be performed to assess for the presence of renal dysfunction.

Renal and bladder ultrasonography is the preferred initial imaging study in the evaluation of prenatal hydronephrosis. The timing of initial renal ultrasonography depends on whether there is unilateral or bilateral involvement or the presence of solitary kidney on prenatal ultrasonography. Renal ultrasonography obtained within 48 hours after birth can show a false absence or reduction of hydronephrosis because

neonatal urinary flow is reduced in the first 1 to 2 days after birth. For the most accurate results, a neonate with prenatally diagnosed unilateral hydronephrosis with a normal contralateral kidney should undergo renal ultrasonography 1 to 2 weeks after birth. Neonates with bilateral prenatal hydronephrosis or a solitary kidney with hydronephrosis should undergo postnatal renal ultrasonography within 48 hours after birth to prevent renal injury due to a delay in diagnosis. Neonates with bilateral hydronephrosis may have posterior urethral valves, which should be promptly managed via urinary catheter insertion and surgical correction. Neonates with hydronephrosis in a solitary kidney may have severe VUR, and early intervention is required.

The presence of a dilated ureter is helpful in narrowing the differential diagnosis of hydronephrosis. Ureteropelvic junction obstruction, which impairs urinary flow from the renal pelvis to the ureter, results in dilation of the renal pelvis (hydronephrosis without a dilated ureter). Ureterovesical junction obstruction, which impairs urinary flow from the ureter to the bladder, leads to dilation of the ureter and structures up to the renal pelvis (hydroureteronephrosis). Other uncommon causes of obstruction to urine flow that lead to hydroureteronephrosis include ureterocele (cystic dilation of the ureter at the insertion into the bladder) and ectopic ureter (abnormal insertion of the ureter into the bladder). Vesicoureteral reflux can also cause hydronephrosis with a dilated ureter.

The severity and persistence of hydronephrosis in the postnatal period determines the need for further evaluation. Neonates with unilateral mild hydronephrosis are monitored with serial renal ultrasonography. A voiding cystourethrogram is indicated for (a) unilateral moderate-to-severe hydroureteronephrosis to diagnose VUR and (b) bilateral hydronephrosis to diagnose bilateral VUR and posterior urethral valves.

Infants with persistent moderate-to-severe hydronephrosis or hydroureteronephrosis without VUR should undergo diuretic renography (99mTc mercaptoacetyltriglycine [MAG-3] with furosemide) to diagnose obstruction of the ureteropelvic junction or the ureterovesical junction. This test should be performed when the infant is at least 4 to 8 weeks of age; neonatal glomerular filtration is low, which can lead to delayed excretion of MAG-3 and an inaccurate test result. Diuretic renography provides both an assessment of differential function of each kidney and information regarding the degree of obstruction. Severe hydronephrosis causing thinning of renal parenchyma, decreased relative renal function, or a marked degree of obstruction on diuretic renography requires surgical correction.

Suggested Reading(s)

- Campbell DE, Nemerofsky SL, Iyare A, Mauch TJ, Schwend RM. Postnatal assessment of common prenatal sonographic findings. In: McInerny TK, Adam HM, Campbell DE, DeWitt TG, Foy JM, Kamat DM, eds. *American Academy of Pediatrics Textbook of Pediatric Care*. American Academy of Pediatrics; 2017:chap 97. Accessed September 1, 2023. Pediatric Care Online
- Nguyen HT. Obstructive uropathy and vesicoureteral reflux. In: McInerny TK, Adam HM, Campbell DE, DeWitt TG, Foy JM, Kamat DM, eds. *American Academy of Pediatrics Textbook of Pediatric Care*. American Academy of Pediatrics; 2017:chap 299. Accessed September 1, 2023. Pediatric Care Online
- Schlomer BJ, Copp HL. Antenatal hydronephrosis. *NeoReviews*. 2013;14(11):e551-e561. doi:10.1542/neo.14-11-e551

Content Domain

- Renal

ABP Content Specification(s) / Content Area(s)

- Formulate a differential diagnosis of urinary tract obstruction
- Recognize the clinical findings associated with hydronephrosis in patients of various ages
- Understand the various causes of urinary tract obstruction

The correct answer is: renal ultrasonography 2 weeks after birth

View Peer Results

Table. Common Etiologies of Unilateral and Bilateral Hydronephrosis.

Unilateral Hydronephrosis*	Bilateral Hydronephrosis
Ectopic ureter	Megacystis-megaureter syndrome
Extrarenal pelvis	Posterior urethral valves
Polycystic kidney disease	Prune belly syndrome
Transient physiologic	Urethral atresia
Unilateral vesicoureteral reflux	
Ureterocele	
Ureteropelvic junction obstruction	
Ureterovesical junction obstruction	

*Can also present as bilateral hydronephrosis

Courtesy of A. Jain

A 3-year-old with end-stage renal disease secondary to renal dysplasia is seen in the office for immunization before undergoing a kidney transplant. The surgery will occur in 8 weeks; their mother will be the donor. The child will receive long-term immunosuppressive medications (tacrolimus and mycophenolate mofetil) to prevent rejection after the transplant. They have received all of their routine age-appropriate immunizations. The child is at the 5th percentile for height and 25th percentile for weight for their age. Vital signs and physical examination findings are unremarkable.

Of the following, the MOST appropriate immunization to administer to this child today is

- A. *Haemophilus influenzae* type b
- B. inactivated poliovirus
- C. measles, mumps, and rubella
- D. pneumococcal conjugate

Correct answer is C

PREP Pearl(s)

- Live virus vaccines should be given at least 4 weeks and inactivated vaccines at least 2 weeks before kidney transplant.
- Live virus vaccines are contraindicated after kidney transplant; the child will receive long-term immunosuppressive medication and will be at risk of experiencing a virulent vaccine strain that causes severe illness.
- Inactivated vaccines can be given 3 to 6 months after kidney transplant, except for the inactivated influenza vaccine, which can be given as early as 1 month after transplant.

Critique

The child in the vignette, whose routine immunizations are up to date, should receive the measles, mumps, and rubella (MMR) vaccine at this visit. The MMR vaccine is routinely recommended at 12 to 15 months and 4 to 6 years of age. Because it is a live virus vaccine, the MMR vaccine is contraindicated after transplantation. Therefore, to enhance the child's immunity, a second dose of the MMR vaccine should be administered at least 4 weeks before the kidney transplant (as long as it has been at least 28 days since their first MMR dose).

Children and adolescents requiring kidney and other solid-organ transplants (eg, heart, liver, and lung) should receive all age-appropriate immunizations before receiving the transplant. Live virus vaccines are contraindicated after transplantation owing to the risk of a virulent vaccine strain causing severe illness in a child with immunosuppression. If indicated, live virus vaccines (eg, MMR, varicella, and live attenuated influenza virus) should be given at least 4 weeks before the transplant. In cases of deceased donor transplant, because the timing of transplantation is not planned, the child is made "inactive" on the transplant list for at least 4 weeks after immunization with a live virus vaccine.

Inactivated vaccines, if indicated, should be given at least 2 weeks before transplantation. The child in the vignette is up to date on their primary series of *Haemophilus influenzae* type b, inactivated poliovirus, and pneumococcal conjugate vaccine; thus, additional doses are not indicated at this time. Pneumococcal polysaccharide vaccine is indicated for transplant candidates older than 2 years for protection against

additional pneumococcal strains. Inactivated virus vaccines can be given 3 to 6 months after transplantation; early administration leads to poor antibody response due to intense immunosuppression. The only exception is inactivated influenza vaccine, which can be given as early as 1 month after transplant and then annually.

Children who are not up to date with their immunizations should receive catch-up vaccinations before the transplant surgery. After transplantation, primary care providers should work closely with a transplant specialist to ensure that immunosuppressed children continue to receive age-appropriate inactivated vaccines.

Suggested Reading(s)

- American Academy of Pediatrics. Immunization and other considerations in immunocompromised children. In: Kimberlin DW, Barnett ED, Lynfield R, Sawyer MH, eds. *Red Book: 2021–2024 Report of the Committee on Infectious Diseases, Committee on Infectious Diseases*. 32nd ed. American Academy of Pediatrics; 2021. Accessed September 1, 2023. Red Book Online
- Katz DT, Torres NS, Chatani B, et al. Care of pediatric solid organ transplant recipients: an overview for primary care providers. *Pediatrics*. 2020;146(6):e20200696. doi:10.1542/peds.2020-0696
- Spinner JA, Denfield SW. Immunosuppressant drugs and their effects on children undergoing solid organ transplant. *Pediatr Rev*. 2022;43(2):71-86. doi:10.1542/pir.2020-000620

Content Domain

- Renal

ABP Content Specification(s) / Content Area(s)

- Plan an appropriate immunization regimen for a patient who is about to undergo renal transplantation

The correct answer is: measles, mumps, and rubella

View Peer Results

A 16-year-old boy is seen for follow-up after an emergency department visit 2 weeks earlier for right flank pain and bright red urine with passage of clots. At that time he had no dysuria. His urinalysis revealed 3+ blood, no protein, 50 to 100 red blood cells/high-power field, and <5 white blood cells/high-power field. Renal ultrasonography showed a 5-mm echogenic focus with acoustic shadowing in the right lower pole of the kidney. The boy was treated with pain medication and intravenous fluids and discharged home after his pain improved.

Today, he has no pain and his urine color is yellow. His physical examination findings, including vital signs, are normal.

A 24-hour urine collection is ordered.

Of the following, this boy's MOST likely urinary metabolic abnormality is

- A. hypocalciuria
- B. hypocitraturia
- C. hypooxaluria
- D. hypouricosuria

Correct answer is B

PREP Pearl(s)

- Adolescents with nephrolithiasis usually have the classic symptom of flank pain; children younger than age 5 years may be asymptomatic or have recurrent abdominal pain.
- Renal ultrasonography is the first-line imaging modality to detect nephrolithiasis.
- A 24-hour urine test for metabolic abnormalities predisposing to nephrolithiasis may reveal hypercalciuria, hypocitraturia, hyperoxaluria, hyperuricosuria, or cystinuria.

Critique

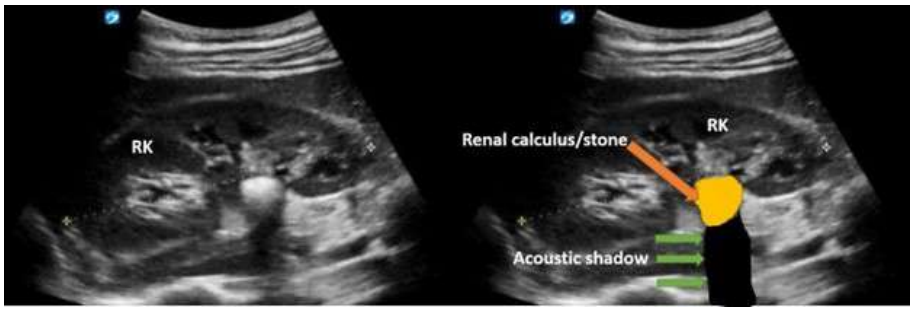
The boy in the vignette has a history of flank pain, hematuria with clots, and renal ultrasonography findings suggestive of nephrolithiasis. A 24-hour urine test for a metabolic abnormality may reveal one or more of the common risk factors for nephrolithiasis including hypercalciuria, hypocitraturia, hyperoxaluria, hyperuricosuria, or cystinuria. Of the response choices, hypocitraturia is the only finding associated with renal stone formation.

Kidney stones are commonly composed of calcium oxalate, calcium phosphate, struvite, cysteine, or uric acid. A renal stone, when retrieved, should be sent for composition analysis. Factors that increase the risk of stone formation include low urine volume, increased solute excretion (eg, calcium, uric acid), a decreased level of stone inhibitors (eg, citrate, magnesium), urinary tract infection, and renal structural abnormalities (eg, renal cyst). A 24-hour urine collection should be obtained after an acute episode to identify treatable urinary metabolic abnormalities such as hypercalciuria, hyperoxaluria, hypocitraturia, hyperuricosuria, or cystinuria. Identification of a specific urinary metabolic abnormality allows for targeted interventions (eg, dietary, medications) to prevent recurrence of nephrolithiasis.

The incidence of nephrolithiasis in children is increasing, most likely due to dietary changes and the rising prevalence of obesity. Adolescents with nephrolithiasis usually demonstrate the classic symptoms of flank pain or renal colic as the stone tries to pass through the ureterovesical junction. Children younger than age 5 years may be asymptomatic or experience recurrent abdominal pain. Other presenting features of nephrolithiasis include gross hematuria, dysuria, urinary urgency, and urinary tract infection.

When evaluating a child with nephrolithiasis, practitioners should obtain a history that includes any urinary tract anomalies (eg, obstruction, renal cyst), recurrent urinary tract infections (eg, *Proteus, Klebsiella*), metabolic conditions (eg, malabsorption syndrome, ketogenic diet), medications (eg, sulfadiazine, indinavir), and family history. The physical examination should include blood pressure measurement, growth parameters, and a thorough abdominal examination. It is important to assess for a mass that could result from a urinary obstruction. The initial laboratory evaluation should include urinalysis with microscopic examination for the presence of crystals; urine culture to rule out urinary tract infection; urine calcium-to-creatinine ratio; and renal function tests.

Renal ultrasonography is the first-line imaging modality recommended to detect nephrolithiasis, which appears as a focal area of echogenicity with acoustic shadowing (**Figure**). However, renal ultrasonography may not detect small stones or ureteral stones. A plain abdominal radiograph detects radiopaque stones. Noncontrast helical computed tomography is the most sensitive test for detection of nephrolithiasis.



Reprinted with permission from POCUS Certification Academy.

Figure. Right kidney (RK) showing hyperechoic calculi (orange arrow) in the renal pelvis with posterior acoustic shadowing (green arrows).

The acute management of nephrolithiasis includes pain control, aggressive fluid administration, and facilitating passage or removal of the stone. Identification of the stone's composition and risk factors for stone recurrence facilitates long-term management with targeted dietary intervention and medications. For children with hypercalciuria, reduced sodium intake and a thiazide diuretic may be indicated. Moderate dietary oxalate restriction and avoidance of excess vitamin C intake are indicated for the management of hyperoxaluria. Citrate supplementation is indicated for those with hypocitraturia to decrease the risk of stone formation.

Suggested Reading(s)

- Gellin CE. Urinary tract stones. *Pediatr Rev*. 2019;40(3):154-156. doi:10.1542/pir.2017-0235
- McKay CP. Renal stone disease. *Pediatr Rev*. 2010;31(5):179-188. doi:10.1542/pir.31-5-179
- Miah T, Kamat D. Pediatric nephrolithiasis: a review. *Pediatr Ann*. 2017;46(6):e242-e244. doi:10.3928/19382359-20170517-02

Content Domain

- Renal

ABP Content Specification(s) / Content Area(s)

- Recognize the signs and symptoms of urinary tract stones in patients of various ages
- Plan the evaluation of urinary tract stones in patients of various ages

The correct answer is: hypocitraturia

A 5-year-old boy is brought to the emergency room for evaluation of body swelling and weight gain over the past 3 days. The swelling started over his eyelids and now involves his legs. He has normal urine output. There is no change in urine color, dysuria, fever, or rash. His vital signs are a blood pressure of 105/65 mm Hg, a heart rate of 92 beats/min, and a respiratory rate of 16 breaths/min.

The boy's weight is at the 85th percentile and height is at the 50th percentile for age. He is alert.

He has swelling of the upper eyelids, abdominal distension, and pitting edema of the lower legs. The remainder of the boy's physical examination findings are unremarkable.

Laboratory results are shown:

Laboratory Test	Result
Sodium	131 mEq/L (131 mmol/L)
Potassium	3.9 mEq/L (3.90 mmol/L)
Chloride	102 mEq/L (102 mmol/L)
Bicarbonate	24 mEq/L (24 mmol/L)
Blood urea nitrogen	14 mg/dL (5.0 mmol/L)
Creatinine	0.3 mg/dL (26.52 µmol/L)
Calcium	7.5 mg/dL (1.88 mmol/L)
Albumin	2.0 g/dL (20.00 g/L)
Urine	
pH	5.7
Specific gravity	1.020
Protein	4+
Blood	Negative
Red blood cells	< 2/high-power field
White blood cells	< 5/high-power field

Of the following, the MOST likely cause of this boy's hyponatremia is

- A. decreased glomerular filtration rate

B. pseudohyponatremia

C. urinary sodium loss

D. water retention

Correct answer is D

PREP Pearl(s)

- Nephrotic syndrome is characterized by heavy proteinuria (urine protein >40 mg/m^2/hr or urine-protein-to-creatinine ratio of >2 mg/mg), hypoalbuminemia (serum albumin <2.5 g/dL [25 g/L]), edema, and hyperlipidemia.
- Children with nephrotic syndrome have mild hyponatremia because of water retention.
- The glomerular filtration rate (indicated by blood urea nitrogen and serum creatinine concentrations) is usually normal in nephrotic syndrome.

Critique

The boy in the vignette has nephrotic syndrome (NS), evidenced by his eyelid and leg edema, hypoalbuminemia, and 4+ proteinuria. His hyponatremia is dilutional secondary to water retention. The low effective circulatory volume secondary to hypoalbuminemia in NS causes a decrease in urine sodium.

Nephrotic syndrome is characterized by the following:

- Heavy proteinuria (urine protein >40 mg/m^2/hr or urine-protein-to-creatinine ratio of >2 mg/mg)
- Hypoalbuminemia (serum albumin <2.5 g/dL [25 g/L])
- Edema
- Hyperlipidemia

Nephrotic syndrome usually presents with periorbital edema and a progressive increase in swelling that leads to pedal edema, ascites, pleural effusion, vulvar or scrotal edema, and anasarca. The typical age of NS onset is between 2 and 10 years. Most cases of NS in children are idiopathic (primary), but some have secondary causes (underlying systemic disease such as vasculitis or systemic lupus erythematosus). Minimal change disease is the most common histology in children with idiopathic NS.

Laboratory evaluation of suspected NS typically begins with a urinalysis that shows proteinuria. Microscopic hematuria (3-5 red blood cells/high-power field) occurs in 25% of children with idiopathic NS. The serum albumin concentration is low because of increased urinary losses. The serum cholesterol concentration is elevated because of low oncotic pressure from loss of serum albumin, increased synthesis, and decreased degradation of products in the cholesterol pathway. The serum calcium concentration may be low in NS as a result of hypoalbuminemia. However, ionized calcium concentrations are normal.

Children with NS have mild dilutional hyponatremia because of water retention, which is a result of lower effective intravascular volume from hypoalbuminemia in addition to inappropriate secretion of antidiuretic hormone. A normal amount of water intake causes edema and hyponatremia in NS. Hyponatremia can also result from diuretic therapy used in the treatment of NS. Hyponatremia in NS is not due to the urinary loss of sodium. Urine sodium excretion is decreased due to low effective intravascular volume. The hyponatremia in NS is true hyponatremia and is not a factitious or pseudohyponatremia.

The glomerular filtration rate (indicated by blood urea nitrogen and serum creatinine concentrations) is usually normal in NS, as seen with the child in the vignette. Mildly increased blood urea nitrogen and serum creatinine concentrations may occur in NS owing to decreased effective circulatory blood volume, but this is not the cause of hyponatremia in NS. A decrease in glomerular filtration rate (azotemia), hypertension, and macroscopic hematuria are commonly seen in nephritic syndrome or acute glomerulonephritis.

Prednisone is the first-line treatment for idiopathic NS. Hyponatremia in NS is treated with water and sodium restriction and by addressing the hypoalbuminemia.

Suggested Reading(s)

- Rodriguez-Ballestas E, Reid-Adam J. Nephrotic syndrome. *Pediatr Rev.* 2022;43(2):87-99. doi:10.1542/pir.2020-001230
- Varade WS. Nephrotic syndrome. In: McInerny TK, Adam HM, Campbell DE, DeWitt TG, Foy JM, Kamat DM, eds. *American Academy of Pediatrics Textbook of Pediatric Care.* 2nd ed. American Academy of Pediatrics; 2016:chap 295. Accessed September 1, 2023. Pediatric Care Online

Content Domain

- Renal

ABP Content Specification(s) / Content Area(s)

- Formulate a differential diagnosis of nephrotic syndrome with and without hematuria
- Identify the etiology of hyponatremia in nephrotic syndrome

The correct answer is: water retention

View Peer Results

A 6-year-old boy is seen in the office for evaluation of a rash over his lower extremities of 7 days' duration and mild, diffuse, colicky abdominal pain of 2 days' duration. He has had no diarrhea or vomiting. The boy's blood pressure is 120/80 mm Hg, his heart rate is 80 beats/min, and his respiratory rate is 14 breaths/min. He has a nonblanching, erythematous, papular rash over the posterior aspects of his bilateral lower limbs (Figure). There is mild pitting edema of his lower extremities without joint swelling. His abdomen is soft, with mild diffuse tenderness on palpation. The remainder of the boy's examination findings are normal.

Reprinted with permission from Quddusi FI, Youssef MJ, Davis DMR. *Pediatr Rev*. 2021;42(12):662.

Figure. Skin findings for the boy described in the vignette.

Laboratory results are shown:

Urine Test	Result
Blood	3+

Urine Test	Result
Red blood cells	50-100/high-power field
Protein	3+

Of the following, the BEST next step in this child's care is to

- A. prescribe a 7-day course of ibuprofen
- B. prescribe a 7-day course of oral prednisone
- C. refer him to a dermatologist for a skin biopsy
- D. refer him to a nephrologist for a kidney biopsy

Correct answer is D

PREP Pearl(s)

- Signs and symptoms of Henoch-Schönlein purpura, the most common vasculitis affecting children, may include palpable purpura over the lower extremities and buttocks, diffuse abdominal pain, arthritis, hematuria, and proteinuria.
- Children with Henoch-Schönlein purpura and mild nephritis (microscopic hematuria, non–nephrotic-range proteinuria) can be closely monitored.
- Children with Henoch-Schönlein purpura and macroscopic hematuria, worsening proteinuria, nephrotic syndrome, or acute kidney injury should be referred to a pediatric nephrologist for a kidney biopsy. Individuals with nephrotic-range proteinuria, nephrotic syndrome, or acute kidney injury are at risk of progressing to chronic kidney disease and end-stage kidney disease.

Critique

The child in the vignette has Henoch-Schönlein purpura (HSP). This diagnosis is supported by the presence of palpable purpura, abdominal pain, and renal involvement (hematuria, proteinuria, edema, and hypertension). The best next step is to refer him to a pediatric nephrologist for a kidney biopsy before starting treatment with prednisone.

Henoch-Schönlein purpura is the most common vasculitis in children, with a peak incidence at age 4 to 6 years. Inflammation of the small vessels of the skin, joints, gastrointestinal tract, and kidneys leads to its classical manifestations. The skin findings of HSP include asymptomatic, erythematous, edematous papules (palpable purpura) over the lower extremities and buttocks. The face, trunk, and forearms may be involved in infants and young children. These skin lesions tend to occur in crops, last up to 10 days, may recur in the first 3 months, and resolve without scarring. Skin biopsy shows leukocytoclastic vasculitis with IgA immune complex deposition. However, skin biopsy is not routinely performed; it is indicated only when the diagnosis is suspected but the rash or clinical presentation is atypical. The child in the vignette has typical skin manifestations of HSP, so referral to a dermatologist for skin biopsy is not indicated.

Arthritis occurs in 60% to 80% of children with HSP; it presents as swelling and tenderness of the ankle, wrist, knee, finger joints, or a combination of these. Gastrointestinal tract manifestations, occurring in 50% to 70% of HSP cases, may include intermittent colicky abdominal pain, vomiting, and blood in the stool. Common complications of HSP are intestinal ischemia, perforation, and small bowel intussusception.

Kidney involvement occurs in 50% to 70% of children with HSP. A urinalysis should be performed at the time of presentation, repeated weekly or biweekly for the first 2 months, and then monthly for 6 months to monitor for hematuria and proteinuria. Children with HSP may have microscopic hematuria, macroscopic

hematuria, proteinuria, nephrotic syndrome, or rapidly progressive acute glomerulonephritis. Kidney function tests and complement levels should be obtained in children with hematuria, proteinuria, and hypertension.

Henoch-Schönlein purpura is a self-limiting illness in the majority of affected children. The symptoms are treated according to their severity. Mild arthritis is managed conservatively, whereas severe arthritis may require treatment with nonsteroidal anti-inflammatory drugs, steroids, or a combination of these. However, nonsteroidal anti-inflammatory drugs should be used cautiously in children with renal involvement to avoid worsening of acute kidney injury. The child in the vignette does not have joint manifestations, so a prescription for ibuprofen is not indicated.

Children with mild abdominal pain may be treated supportively. Children with severe abdominal pain and gastrointestinal bleeding are usually treated with steroids for 2 to 4 weeks. The child in the vignette has mild abdominal pain, so treatment with prednisone is not indicated.

Children with mild HSP nephritis (microscopic hematuria, non-nephrotic–range proteinuria) can be closely monitored. Those with macroscopic hematuria, worsening proteinuria, nephrotic syndrome, or acute kidney injury should be referred to a pediatric nephrologist for a kidney biopsy, which will confirm the diagnosis of IgA nephropathy, reveal the severity of kidney involvement, and guide treatment with prednisone or other immunosuppressive medication. The kidney biopsy should be performed before starting prednisone treatment so as to avoid masking the findings. The duration of prednisone treatment for HSP nephritis is up to 12 weeks to avoid chronic kidney damage. Severe HSP nephritis (including crescentic glomerulonephritis) is managed aggressively with intravenous steroids, cyclophosphamide, intravenous immunoglobulin, plasmapheresis, or a combination of these.

Children with mild HSP have an excellent prognosis. Microscopic hematuria resolves spontaneously. The long-term prognosis depends on the severity of kidney involvement. Children with HSP and nephrotic-range proteinuria, nephrotic syndrome, or acute kidney injury are at risk of progressing to chronic kidney disease and end-stage kidney disease.

Suggested Reading(s)

- Adrogue HE, Hayde NA. Henoch-Schönlein purpura. In: McInerny TK, Adam HM, Campbell DE, DeWitt TG, Foy JM, Kamat DM, eds. *American Academy of Pediatrics Textbook of Pediatric Care*. 2nd ed. American Academy of Pediatrics; 2016:chap 264. Accessed September 1, 2023. Pediatric Care Online
- Kamat DM. Henoch-Schonlein purpura. Henoch-Schönlein purpura. *Point-of-Care. Quick Reference.* American Academy of Pediatrics; 2020. Accessed February 23, 2023. Pediatric Care Online
- Quddusi FI, Youssef MJ, Davis DMR. Dermatologic manifestations of systemic diseases in childhood. *Pediatr Rev*. 2021;42(12):655-671. doi:10.1542/pir.2020-000679
- Reid-Adam J. Henoch-Schönlein purpura. Pediatr Rev. 2014;35(10):447-449 doi:10.1542/pir.35-10-447

Content Domain

- Renal

ABP Content Specification(s) / Content Area(s)

- Recognize the renal findings associated with Henoch-Schönlein purpura
- Understand the prognostic implications when Henoch-Schönlein purpura is associated with nephrotic syndrome
- Recognize the clinical findings associated with IgA nephropathy

The correct answer is:
refer him to a nephrologist for a kidney biopsy

View Peer Results

A 14-year-old boy with type 1 diabetes mellitus is brought to the emergency department having experienced 2 days of abdominal pain, vomiting, and fatigue. He has had decreased urine output for 1 day. He has no fever, cough, diarrhea, or upper respiratory infection symptoms. He was diagnosed with diabetes at age 9 years and currently takes insulin glargine subcutaneously once daily and insulin lispro subcutaneously with meals.

On physical examination, the boy's weight is at the 5th percentile and height is at the 10th percentile for age. His blood pressure is 100/70 mm Hg, heart rate is 102 beats/min, and respiratory rate is 20 breaths/min. He has dry mucous membranes, and his capillary refill time is 3 seconds. His abdomen is soft and nontender. The remainder of the boy's examination findings are unremarkable.

Laboratory results are shown:

Laboratory Test	Result
Sodium	135 mEq/L (135 mmol/L)
Potassium	5.5 mEq/L (5.5 mmol/L)
Chloride	100 mEq/L (100 mmol/L)
Bicarbonate	14 mEq/L (14 mmol/L)
Blood urea nitrogen	56 mg/dL (19.99 mmol/L)
Creatinine	2 mg/dL (176.80 µmol/L)
Glucose	360 mg/dL (19.98 mmol/L)
Osmolality	316 mOsm/Kg
β-Hydroxybutyrate	32 mg/dL (reference range, 0.2-2.8 mg/dL)
Urine	
Specific gravity	1.030
pH	5.5
Ketones	Present
Leukocyte esterase	Negative
Blood	Negative
Protein	Negative

Laboratory Test	Result
Red blood cells	<2/high power field
White blood cells	<5/high power field

Of the following, the additional laboratory finding MOST likely to be seen in this adolescent is

- A. a fractional excretion of sodium <1%
- B. a fractional excretion of urea >50%
- C. red blood cell casts on urine microscopy
- D. a urine-to-serum osmolality ratio <1.5

Correct answer is A

PREP Pearl(s)

- The etiology of acute kidney injury can be divided into prerenal (decreased effective renal blood flow), renal (intrinsic renal damage), and postrenal (obstruction to the flow of urine).
- The fractional excretion of sodium is less than 1% in prerenal acute kidney injury.
- The initial step in the management of prerenal acute kidney injury is to restore intravascular volume using a 10 to 20 mL/kg intravenous bolus of 0.9% saline solution.

Critique

The adolescent in the vignette has diabetic ketoacidosis (DKA) (hyperglycemia, metabolic acidosis, high serum β-hydroxybutyrate concentration, and ketonuria) and acute kidney injury (AKI) (elevated blood urea nitrogen and serum creatinine concentrations) secondary to dehydration from osmotic diuresis and vomiting. He most likely has prerenal AKI (urinalysis with no blood, protein, or casts; high urine specific gravity; and blood urea nitrogen–to–serum creatinine ratio >20). In the setting of prerenal AKI, his fractional excretion of sodium would be <1%, indicating reabsorption of the filtered sodium and water in response to renal hypoperfusion.

In acute kidney injury, there is a decrease in urine output or an increase in serum creatinine, leading to a decrease in glomerular filtration rate. The etiology of AKI (Table) can be divided into the following categories:

- Prerenal: decreased effective renal blood flow
- Renal: intrinsic renal damage
- Postrenal: obstruction to the urine flow

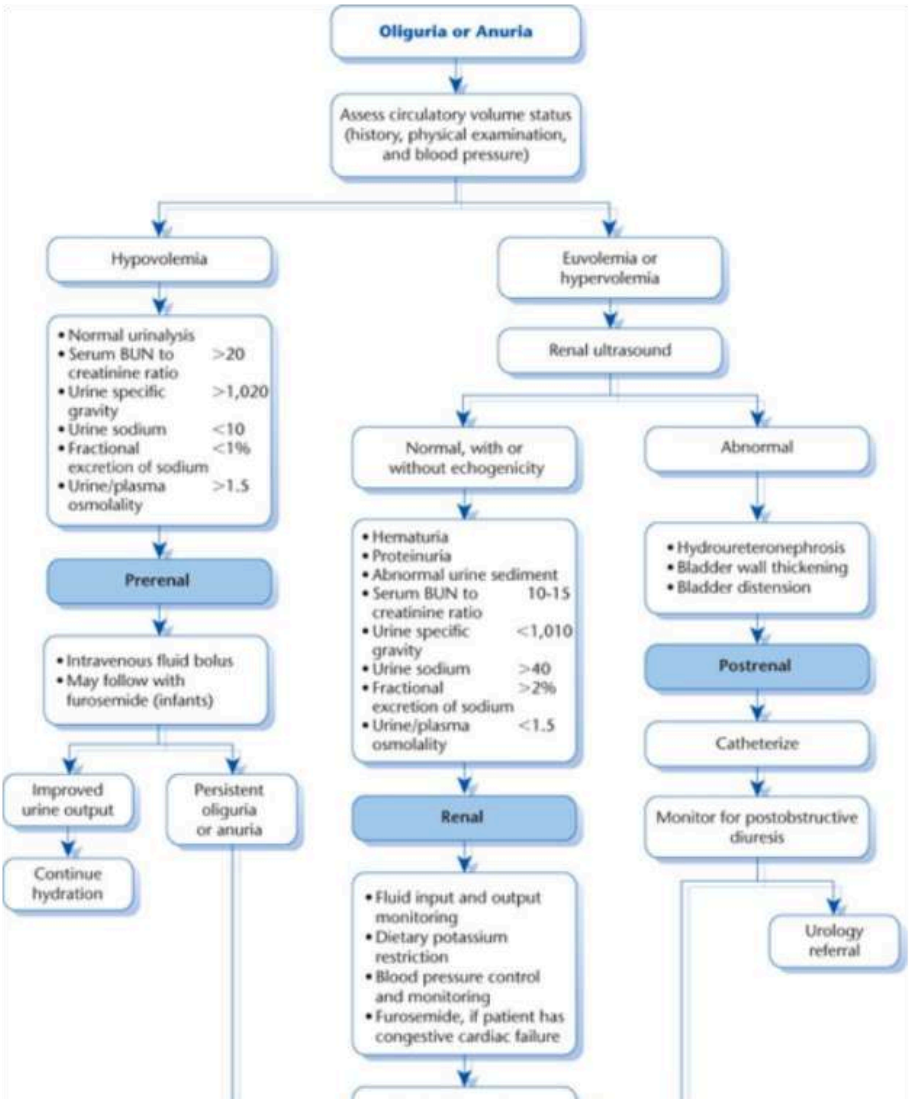
Findings on urinalysis with microscopic examination and urinary indices (fractional excretion of sodium, renal failure index) are useful to differentiate the cause of AKI. Prerenal AKI is associated with a normal urinalysis result, with no blood, protein, cells, or casts. The presence of granular or epithelial cell casts on urinalysis is suggestive of acute tubular necrosis and red blood cell casts are associated with acute glomerulonephritis; both conditions are renal causes of AKI. A blood urea nitrogen–to–serum creatinine ratio <20 and a urine-to-serum osmolality ratio <1.5 support a diagnosis of renal AKI.

The fractional excretion of sodium (FENa) differentiates prerenal AKI from renal AKI and is calculated by using the following formula:

(urine sodium × serum creatinine) ÷ (serum sodium × urine creatinine) × 100

A FENa less than 1% is suggestive of prerenal AKI, and a FENa of greater than 2% favors renal AKI (eg, acute tubular necrosis). The fractional excretion of urea (FEUrea) is also used to differentiate prerenal AKI from renal AKI. The FEUrea is a more accurate test than FENa in children who are receiving diuretic therapy. A

FEUrea of less than 35% is suggestive of prerenal AKI and a FEUrea greater than 50% is seen in renal AKI. The Figure defines an algorithm to differentiate the types of AKI and their initial management.



Abbreviation: BUN, blood urea nitrogen.

Reprinted with permission from Jain A, Mattoo TK. Oliguria and anuria. In: McInerny TK, Adam HM, Campbell DE, DeWitt TG, Foy JM, Kamat DM, eds. *American Academy of Pediatrics Textbook of Pediatric Care*. 2nd ed. American Academy of Pediatrics; 2017:chap 301.

Figure. Algorithm for determination of the type and management of acute kidney injury.

The management of DKA with its associated dehydration and prerenal AKI is challenging. The goal is to correct the dehydration and acidosis with simultaneous slow correction of hyperglycemia to minimize the risk of cerebral edema. The initial step is to restore intravascular volume using an intravenous isotonic crystalloid fluid bolus (eg, 10-20 mL/kg of 0.9% saline). This fluid is administered before insulin therapy begins. The need for further fluid boluses is determined according to the child's vital signs, urine output, and fluid status. Maintenance and deficit fluid calculations should be performed. The goal is to correct the fluid deficit over 24 to 48 hours using 0.45% to 0.9% saline solution.

After the initial fluid resuscitation, a continuous insulin infusion should be started, with dextrose added to the intravenous fluid to maintain stable serum glucose concentrations. Bicarbonate administration is not routinely recommended for DKA management. Laboratory data (eg, glucose, sodium, potassium concentrations) should be carefully monitored. Correction of the fluid deficit and hyperglycemia improves the DKA and AKI. Once the acidosis is resolved and the child is tolerating oral intake, transition to subcutaneous insulin is indicated.

Suggested Reading(s)

- Cashen K, Petersen T. Diabetic ketoacidosis. *Pediatr Rev*. 2019;40(8):412-420. doi:10.1542/pir.2018-0231
- Glaser N, Fritsch M, Priyambada L, et al. ISPAD clinical practice consensus guidelines 2022: diabetic ketoacidosis and hyperglycemic hyperosmolar state. *Pediatr Diabetes*. 2022;23(7):835-856. doi:10.1111/pedi.13406
- Jain A, Mattoo TK. Oliguria and anuria. In: McInerny TK, Adam HM, Campbell DE, DeWitt TG, Foy JM, Kamat DM, eds. *American Academy of Pediatrics Textbook of Pediatric Care*. 2nd ed. American Academy of Pediatrics; 2017:chap 301. Accessed September 1, 2022. Pediatric Care Online
- Selewski DT, Symons JM. Acute kidney injury. *Pediatr Rev*. 2014;35(1):30-41. doi:10.1542/pir.35-1-30

Content Domain

- Renal

ABP Content Specification(s) / Content Area(s)

- Plan the appropriate diagnostic evaluation of oliguria
- Plan the appropriate initial management of acute renal failure, while considering the effects of various therapies on associated physiologic abnormalities

The correct answer is: a fractional excretion of sodium <1%

View Peer Results

Table. Etiology of Acute Kidney Injury.

Age	Prerenal	Renal	Postrenal
Neonates	• Congenital heart disease • Dehydration • Drugs (indomethicin, maternal use of ACE inhibitor or NSAIDs) • Hemorrhage • Perinatal asphyxia • Respiratory distress syndrome • Sepsis or shock	• ATN • Congenital kidney disease • Endogenous toxins (hemoglobin, myoglobin, uric acid) • Exogenous toxins (aminoglycosides, amphotericin B) • Vascular (renal vein thrombosis, renal artery thrombosis)	• Bilateral ureteral obstruction • Meatal stenosis • Neurogenic bladder • Posterior urethral valves
Children	• Burns • Decreased cardiac output • Dehydration • Hemorrhage • Renal loss (diabetes mellitus, diabetes insipidus, diuretics) • Shock • Third-space loss (surgery, trauma, nephrotic syndrome)	• ATN • Endogenous toxins (hemoglobin, myoglobin, uric acid) • Exogenous toxins (aminoglycosides, amphotericin B) • Glomerulonephritis • Interstitial nephritis • Vascular (hemolytic uremic syndrome, vasculitis)	• Bilateral ureteral obstruction • Meatal stenosis • Neurogenic bladder • Obstructive urinary tract stones or sludge • Posterior urethral valves

ACE, angiotensin-converting enzyme; ATN, acute tubular necrosis; NSAIDs, nonsteroidal anti-inflammatory drugs.

A 5-day-old neonate is undergoing a routine health supervision visit. He was born to a 30-year-old primigravida mother at 39 weeks' gestation via normal vaginal delivery. Prenatal ultrasonography showed an absent left kidney and a normal amount of amniotic fluid. Renal ultrasonography performed 2 days after birth showed a normal right kidney with normal corticomedullary differentiation and no hydronephrosis. The left kidney was not visualized.

The neonate is breastfed and voids multiple times per day. He passed the newborn hearing screening test. His vital signs and physical examination findings are normal. He is circumcised.

Of the following, the condition MOST likely to be seen in this neonate is

- A. compensatory renal hypertrophy
- B. hypertension secondary to renal artery stenosis
- C. pulmonary hypoplasia secondary to Potter sequence
- D. ureteropelvic junction obstruction

Correct answer is A

PREP Pearl(s)

- Unilateral renal agenesis is usually detected on prenatal ultrasonography and occasionally during the evaluation of a urinary tract infection.
- Children with a solitary kidney should undergo regular monitoring for compensatory hypertrophy (via renal ultrasonography), proteinuria, and hypertension.

Critique

The neonate in the vignette has unilateral renal agenesis and a normal contralateral kidney. In this scenario, the contralateral kidney is expected to undergo compensatory hypertrophy.

Renal agenesis is the congenital absence of a kidney due to lack of initiation of embryonic development. The reported incidence of unilateral renal agenesis is 1 in 1,000 to 3,000 live births. It is more common in males and occurs predominantly on the left side. Unilateral renal agenesis is usually detected on prenatal ultrasonography and occasionally during the evaluation of a urinary tract infection. One-third of children with renal agenesis have associated extrarenal malformations of the heart, genitals, bone, or gastrointestinal tract. The associated findings of hearing loss or abnormal hearing test result, branchial cyst, preauricular pits, and renal agenesis is suggestive of branchiootorenal syndrome.

Ultrasonography is the preferred initial imaging modality to confirm the absence of one kidney, detect associated abnormalities of the contralateral kidney and urinary tract, and measure the size of the solitary kidney. Abnormal echogenicity and/or decreased size of the solitary kidney may suggest renal dysplasia.

Vesicoureteral reflux can be associated with a solitary kidney, but it is usually low grade. Hydronephrosis of a solitary kidney warrants a voiding cystourethrogram to evaluate for vesicoureteral reflux or a renal scan to detect ureteropelvic junction obstruction. When the contralateral kidney is normal, it is expected to undergo compensatory renal hypertrophy (kidney size at or above the 50th percentile for age) to account for the reduced nephron number.

Children with a solitary kidney should undergo regular monitoring for the following:

- Compensatory hypertrophy (via renal ultrasonography)
- Proteinuria
- Hypertension

The solitary kidney can experience hyperfiltration injury, resulting in proteinuria, hypertension, and reduced glomerular filtration rate. Children who have obesity, hypertension, or proteinuria, as well as those without compensatory renal hypertrophy, are at risk of experiencing progression to chronic kidney disease with solitary kidney.

Hypertension associated with a solitary kidney can occur in adulthood (not in the neonate) owing to hyperfiltration injury and obesity; this is not caused by renal artery stenosis. Potter syndrome or sequence (oligohydramnios, facial and limb abnormalities, and pulmonary hypoplasia) is associated with bilateral renal agenesis. Bilateral renal agenesis is almost universally fatal at or shortly after birth owing to severe pulmonary hypoplasia. The neonate in the vignette has no hydronephrosis of the contralateral kidney; therefore, he is unlikely to have ureteropelvic junction obstruction.

Suggested Reading(s)

- Janjua HS, Lam SK, Gupta V, Krishna S. Congenital anomalies of the kidneys, collecting system, bladder, and urethra. *Pediatr Rev*. 2019;40(12):619-626. doi:10.1542/pir.2018-0242
- Wolfe DS, Suskin B. Prenatal diagnosis. In: McInerny TK, Adam HM, Campbell DE, DeWitt TG, Foy JM, Kamat DM, eds. *American Academy of Pediatrics Textbook of Pediatric Care*. 2nd ed. American Academy of Pediatrics; 2016:chap 82. Accessed November 17, 2023. Pediatric Care Online

Content Domain

- Physiology

ABP Content Specification(s) / Content Area(s)

- Recognize the clinical findings associated with various anomalies of the kidneys, urinary collecting system, and urinary excretion system
- CPlan the diagnostic evaluation of abnormalities of the kidneys, urinary collecting system, bladder, and urethra

The correct answer is: compensatory renal hypertrophy

View Peer Results