Management of Pediatric Lower Limb Length Inequality

by Michael C. Albert, MD
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The answer sheet and program evaluation must be received by August 1, 2018, for the credit to be awarded.

target audience

This education activity is designed for pediatricians, family physicians and related child health care providers.

author information

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educational objectives

- Identify the four pediatric issues covered in this journal and develop appropriate intervention.

- Appropriately use the resources of Dayton Children’s Hospital to improve patient care.
Management of Pediatric Lower Limb Length Inequality

by Michael C. Albert, MD
Following the completion of this article, the reader should be able to:
1. Understand various treatment options for lower limb length inequality.
2. Discuss general etiology and categories of limb length inequality.
3. Understand examination techniques and management principles with regard to limb length inequality.

Limb length inequality

Limb length inequality (LLI) is very common in the lower extremities. Small degrees of LLI occur in over half the population of the United States. It has been estimated that LLI of greater than 2.0 cm occurs in seven percent of the population age 8 to 12 years old. It is well known that gait patterns can be altered with discrepancies of over 2.0 cm.

Finite element models show increased sacroiliac joint loading with peak stresses increasing as the discrepancy increases from one to three centimeters, thus serving as a model demonstrating that LLI may lead to low back pain. Although no long-term studies associate osteoarthritis with limb length inequality, adults with knee osteoarthritis report pain more commonly in the short leg.

Parents perceive that the happiness of their children is adversely affected by increasing limb length inequality.

etiology

There are numerous etiologies of limb length inequality that can be categorized in three general groups:
1) direct change in length,
2) growth inhibition and
3) growth stimulation.

Examples of direct change in length are developmentally dislocated hips causing relative shortening of the lower extremity and fractures causing limb length inequality. Growth inhibition can be caused by numerous congenital diagnoses such as proximal femoral focal deficiency (PFFD) (Figure 1), fibular hemimelia, congenital short femur, posterior medial bowing, clubfoot or hemiatrophy. Other causes of growth inhibition include physeal injury from trauma, infection or irradiation as well as paralysis and tumors.

Growth stimulation occurs in congenital vascular malformations, vascular tumors, trauma and chronic inflammation (Figure 2). Table 1 provides a summary of categories resulting in limb length inequality.

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<thead>
<tr>
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<td>Aplasia</td>
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<td></td>
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<td>Disuse</td>
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<td>Vascular</td>
<td>Ischemia</td>
<td>AV fistula</td>
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<td></td>
<td>Perthes disease</td>
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<tr>
<td>Infection</td>
<td>Physeal injury</td>
<td>Stimulation</td>
</tr>
<tr>
<td>Tumors</td>
<td>Physeal involvement</td>
<td>Vascular lesions</td>
</tr>
<tr>
<td>Trauma</td>
<td>Physeal injury</td>
<td>Fracture stimulation</td>
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<tr>
<td></td>
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natural history

Shapiro et al studied the natural history of LLI, publishing developmental patterns in lower extremity length discrepancies in the Journal of Bone and Joint Surgery in 1982. This landmark paper demonstrates estimated discrepancies in various conditions (Table 2).

secondary effects of LLI

Gait abnormalities are dependent on the magnitude of inequality. In general, the long leg compensates by flexing the knee and circumducting or vaulting the long leg to clear the floor. The short leg compensates by standing in equinus. Energy consumption has shown to be increased secondary to significant LLI. Other secondary effects of LLI include possible increase in low back pain and association of osteoarthritis in the short leg.

evaluation

In obtaining the history, it is important to determine the etiology and if this is going to be a static or progressive deformity. Document areas of pain and disability. During the physical exam, it is essential to differentiate between a “true” LLI versus an apparent LLI secondary to joint contractures or joint instability. It is also important to perform a thorough neurologic and vascular examination (Figure 3).

Table 2.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Average # of Patients</th>
<th>Discrepancy</th>
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<tbody>
<tr>
<td>PFFD</td>
<td>18</td>
<td>27 cm</td>
</tr>
<tr>
<td>Olliers disease</td>
<td>17</td>
<td>10 cm</td>
</tr>
<tr>
<td>Congenital short femur</td>
<td>102</td>
<td>6 cm</td>
</tr>
<tr>
<td>Neurofibromatosis</td>
<td>21</td>
<td>4 cm</td>
</tr>
<tr>
<td>Destroyed growth plate</td>
<td>21</td>
<td>3 cm</td>
</tr>
<tr>
<td>Hemihypertrophy</td>
<td>86</td>
<td>3 cm</td>
</tr>
<tr>
<td>Hemiatrophy</td>
<td>27</td>
<td>3 cm</td>
</tr>
<tr>
<td>Hemangiomas</td>
<td>28</td>
<td>3 cm</td>
</tr>
<tr>
<td>Cerebral palsy</td>
<td>46</td>
<td>2 cm</td>
</tr>
<tr>
<td>JRA</td>
<td>36</td>
<td>2 cm</td>
</tr>
<tr>
<td>Perthes disease</td>
<td>140</td>
<td>2 cm</td>
</tr>
<tr>
<td>Fracture femur</td>
<td>116</td>
<td>1 cm</td>
</tr>
</tbody>
</table>

Figure 2.
Growth Stimulation Klippel-Trenaunay-Weber Syndrome

Figure 3A. Measurement by tape measure from anterior superior iliac spine to medial malleolus

Figure 3B and C. Use of wooden blocks to level the pelvis to equalize limb lengths
Radiographic techniques include orthoradiography (Figure 4A), scanography (Figure 4B) and computed tomography (CT). A scanogram measures only limb lengths, whereas orthoradiograph and CT scan can measure both length and alignment. Dayton Children’s Orthopaedic Center is transitioning to low-dose radiation exposure upright biplanar radiographic imaging called EOS. This new technology allows three-dimensional imaging of the spine and lower extremities with a significant decrease in radiation exposure (Figure 4C).

Management principles

Treatment decisions about limb equalization are based on the projected limb length discrepancy at maturity. There are numerous methods to predict discrepancy at skeletal maturity. The Arithmetic Method uses the rate of growth of the distal femur (1.0 cm/year) and proximal tibia (0.6 cm/year) with growth ending at maturity in boys at age 16 and girls at age 14.

The Growth Remaining Method uses growth remaining graphs and skeletal age, while the Straight Line Graph Method simplifies the Growth Remaining Method. Finally, the Multiplier Method uses several limb length databases and chronologic age. Sander’s Digital Skeletal Age has shown to be superior to using chronologic age in the Multiplier Method.

Discrepancies 2.0 cm or less can be treated with observation or shoe lift. Discrepancies from 2 to 5 cm can be treated with epiphysiodesis prior to maturity or femoral/tibial shortening after maturity. Limb lengthening is reserved for discrepancies over 5 cm in length. Patients with severe limb length inequality of over 20 cm with poor functioning joints who cannot tolerate limb shortening are candidates for amputation and prosthetic fitting.

The above are general guidelines used in clinical decision making for treatment of limb length inequality. Many other factors should be considered such as overall stature, joint stability, joint contractures, neurovascular problems, and emotional stability of patient and family before undergoing limb lengthening.

State-of-the-art treatment methods

Dayton Children’s Orthopaedic Center has been at the forefront of limb deformity correction, having done the first Ilizarov Distraction Osteogenesis Limb Lengthening procedure on a child in our region in 1989.

Since then, there have been numerous innovations in circular external fixator limb lengthening devices. Most recently, we have performed the first magnetic limb lengthening using the PRECICE nail in our region. This is an intramedullary rod that has a magnetic actuator drive that uses an external electromagnetic actuator to control rate and direction of lengthening, thus eliminating the need of external fixation (Figure 5).
case study: distraction osteogenesis

S.D. is a 2-year-old female who presented to the Orthopaedic Center at Dayton Children’s for an LLI noticed by the family. Her birth and developmental history were unremarkable and there was no family history of limb deformity.

Examination revealed an LLI by tape measure and blocks of 2.5 cm left longer than right. She had hyperpigmentation involving her trunk, abdomen and left leg. Her neurovascular exam was normal, and she had hemi-hypertrophy on her left calf with 2.5 cm increase in growth compared to the contralateral leg. A scanogram was obtained which showed a 3 cm overall limb length inequality with 2.0 cm difference in the tibias. Genetics was consulted verifying diagnosis of Klippel-Trenaunay-Weber syndrome. MRI of her brain and cervical, thoracic lumbar spine revealed no abnormalities.

By age 6, her exam revealed a progressive discrepancy showing a 7.0 cm LLI; thus she had her first limb lengthening using a spatial frame external fixator and obtaining 5 cm of length, 20 percent of the length of her tibia (Figure 6).

Figure 5A. PRECICE intramedullary limb lengthening of a femur

Figure 5B. Internal magnet with complex gearbox system using lead screw to lengthen the intramedullary nail

Figure 5C. Daily lengthening using actuator performed by the patient at home

Figure 6A. Two weeks after lengthening with spatial frame

Figure 6B. Two months after lengthening with spatial frame

Figure 6C. Six months after lengthening with spatial frame

Figure 6. By age 6, her exam revealed a progressive discrepancy showing a 7.0 cm LLI; thus she had her first limb lengthening using a spatial frame external fixator and obtaining 5 cm of length, 20 percent of the length of her tibia (Figure 6).
During her lengthening of the tibia, she developed an equinus contracture on the lengthened leg requiring a percutaneous heel cord lengthening upon removal of the frame.

Despite a 5.0 cm lengthening, her LL continued to progress and by age 10 years, 2 months, she had a discrepancy of 8.0 cm. We then did a PRECICE Femoral Nail, lengthening her magnetically an additional 6.0 cm (Figure 7).

She has regained full motion and function from her intramedullary limb lengthening having lengthened her right leg a total of 11.0 cm. We used a combination of two limb lengthening techniques, using an external fixation done on her tibia thus avoiding her growth plates. Magnetic intramedullary lengthening can only be used in children over 10 years as the width and length of the rods are limiting factors.

**conclusion**

Limb length discrepancy is very common. It is important to determine the etiology and predict the discrepancy at maturity in order to determine best treatment options. Limb lengthening continues to evolve with game-changing technology on the horizon.

**references**


CME questions

1. Common causes of limb length inequality include:
   A. Growth stimulation (vascular tumors or infection)
   B. Direct change in length (trauma, dislocated hip)
   C. Growth inhibition (posterior medial bowing, PFFD)
   D. None of the above
   E. All of the above

2. A 13-year-old male has a static limb length inequality of 3.5 cm predominantly on the femur. His bone age is the same as calendar age. The most appropriate treatment is:
   A. Observation
   B. Shoe lift
   C. Percutaneous epiphysiodesis of long leg distal femur
   D. Limb lengthening with spatial frame (external fixation)
   E. Limb lengthening with PRECICE Magnetic Femoral Nail

3. The following statement is false concerning EOS:
   A. EOS is an upright biplanar imaging technique.
   B. The main disadvantage of EOS is increased radiation exposure.
   C. EOS can include the entire spine and lower extremities.
   D. EOS imaging allows studying of rotational and torsional lower extremity deformities.
Pediatric Urinary Tract Infections: Evaluation, Diagnosis and Management

by V. Rama Jayanthi, MD, and Christopher Brown, MD
Following the completion of this article, the reader should be able to:
1. Understand the appropriate evaluation of a child presenting with possible UTI, including proper method of obtaining a urine sample in a given patient.
2. Identify which patients presenting with a possible UTI should receive antibiotic treatment.
3. Recognize when imaging should be obtained, and be able to order appropriate imaging for the clinical situation.

Objectives

Pediatric Urinary Tract Infections

Urinary tract infections (UTIs) are relatively common infections during childhood that may affect any portion of the urinary tract, from the kidneys to the urethra. Pediatric UTIs are estimated to affect 2.4 to 3 percent of all U.S. children each year, and result in over 1.1 million office visits annually and an estimated inpatient cost of over $180 million.¹,²

UTIs have a range of presentations, from uncomplicated cystitis to severe febrile infections, which have the potential to lead to renal scarring and chronic kidney disease. In the first year of life, boys are more likely than girls to develop a UTI. Uncircumcised boys in this age range have 10 times the risk of UTI than circumcised boys. After the first year of life, girls are much more likely to develop a UTI, with the overall childhood risk of UTI 2 percent for boys and 8 percent for girls.²

A recent study showed that up to one-third of providers may delay testing urine in patients that are high risk for UTI.³ Bladder infections do not typically result in long-term sequelae, but upper urinary tract infections may result in permanent renal scarring with inherent risk of subsequent development of hypertension or impaired renal function. Because of the possibilities of renal damage and scarring that may occur after febrile UTIs, providers should think of the possibility of a UTI in a febrile patient without an obvious source.

Evaluation

While older children and adolescents can easily describe symptoms that point to a UTI as a likely diagnosis, one must have a high index of suspicion for UTI in children younger than 2 to 3 years of age as they cannot accurately describe their symptoms. When evaluating a child with a possible UTI, it is important to consider and note the following points in the history:⁴
- Patient age and gender
- First or recurring infection
- Febrile (>100.4°F) or nonfebrile UTIs
- Any known abnormalities of the genitourinary tract (identified on prenatal or postnatal renal and bladder ultrasounds)
- Prior surgeries
- Family history of UTI
- Sexual history in adolescents
- Drinking and voiding habits
- Bowel habits (especially constipation and infrequent bowel movements)
- Associated nausea or vomiting
- Urinary urgency, frequency or dysuria
- Poor appetite or failure to thrive
- Lethargy
- Hematuria

Physical examination should include palpation of the abdomen, suprapubic region and costovertebral angles to determine if tenderness is present. Focused examination of the external genitalia should make note of any lesions, discharge, foreign bodies, tenderness, and labial adhesions in girls, or phimosis or meatal stenosis in boys.¹

Diagnosis

There is an association between delay in treatment of febrile UTIs and permanent renal scarring. In febrile children without an apparent source for their infection, clinicians should not delay testing for UTI.⁵ In these children, before administering any antibiotics, a urine specimen should be obtained and sent for both urinalysis and culture.⁴, ⁶, ⁷ A urinalysis is suggestive of a UTI when it shows positive leukocyte esterase or nitrite test results, or leukocytes or bacteria are seen on microscopy. These findings should be confirmed by urine culture.⁴, ⁶, ⁷

In toilet-trained children, a voided urine specimen after properly cleaning the genitals should be sufficient to evaluate for a possible UTI. A diagnosis of UTI is made in the presence of ≥100,000 cfu/mL of a single uropathogen. Contamination of the urine sample is suggested when there are fewer than 50,000 colonies or in the presence of multiple organisms.
In toddlers and infants not yet toilet trained, a specimen can be obtained in one of three ways.\textsuperscript{4, 6, 7}

1. **Bagged urine specimen**

   Bagged specimens are convenient, but in general they are only helpful if they are negative because they are often contaminated by perineal flora. When a urinalysis from a bagged specimen suggests a UTI, then a second urine specimen needs to be collected through catheterization or suprapubic aspiration to be sent for urine culture.

2. **Catheterization**

   While this is more invasive, there is a significantly smaller risk of contamination of the specimen. Diagnosis of UTI requires both a urinalysis suggestive of infection and \( \geq 50,000 \) cfu/mL of a single uropathogen.

3. **Suprapubic aspiration (SPA)**

   This is the most invasive test, but also has the lowest risk of contamination. Diagnosis of UTI requires both a urinalysis suggestive of infection and \( \geq 50,000 \) cfu/mL of a single uropathogen.

Asymptomatic bacteriuria is defined as 1) no pyuria and 2) the growth of a significant number of a single organism (traditionally \( > 100,000 \) cfu/mL) in the urine sample of an asymptomatic child. This is commonly seen in children with neurogenic bladders, or those performing clean intermittent catheterization (CIC) and should not be treated with antibiotics.\textsuperscript{8}

Just as asymptomatic bacteriuria should not be treated with antibiotics, one should also avoid treating symptoms in the absence of a positive urine culture. Symptoms such as urinary frequency, urgency, dysuria, abdominal, suprapubic, or genital pain often accompany a UTI, but they are not specific for UTIs. When evaluating a child with these or other vague urinary symptoms, antibiotic administration is inappropriate in the absence of laboratory evidence of a UTI.

**treatment**

In general, oral and parenteral antibiotic administration is equally efficacious for UTIs. When administering empiric antibiotics, the choice of antibiotic should be based on local antimicrobial sensitivity/resistance patterns (if available), and adjusted (if needed) according to the culture sensitivities.\textsuperscript{6, 7}

Parenteral empiric antibiotics recommended by the American Academy of Pediatrics (AAP) for UTIs include third-generation cephalosporins, gentamicin, tobramycin and piperacillin. Recommended oral agents include cephalosporins, sulfonamides and amoxicillin-clavulanate.\textsuperscript{6} Escherichia coli is the most common organism causing UTIs in pediatric patients, and any empiric antibiotics given should cover this bacterium. Antibiotic resistance is a growing concern, as E. coli isolates nationwide have shown growing resistance to trimethoprim-sulfamethoxazole (21.3-24 percent), \( \beta \)-lactams (37.7 percent), and ampicillin (45 percent). There is a less than 1 percent resistance to nitrofurantoin.\textsuperscript{9, 10}

For uncomplicated episodes of cystitis, it is recommended to treat with three to seven days of oral antibiotics. A post-treatment urine culture is not needed if the symptoms have resolved.

For episodes of pyelonephritis, some patients may be treated on an outpatient basis, while others require inpatient antibiotic administration and close observation. Inpatient treatment is recommended for toxic appearing patients, those under 2 months of age, those unable to tolerate oral medications, or those in whom there is a concern for compliance. Once a patient is afebrile for 24 to 48 hours, a seven- to 14-day course of oral antibiotics is appropriate.\textsuperscript{4, 6, 7}

**bladder and bowel management**

Any signs of bowel and bladder dysfunction (BBD) should be addressed, as these may contribute to increased risk of UTIs. Children should have regular, soft bowel movement. Treatment of constipation has been shown to decrease UTI recurrence.\textsuperscript{11}

Primary care physicians can evaluate for BBD and initiate measures to address these problems.\textsuperscript{12}

**imaging**

Imaging is not always required in children with UTIs, and is typically obtained in patients with febrile or recurrent UTIs. The AAP has issued evidence-based guidelines regarding the appropriate use of imaging.\textsuperscript{6, 7} In order to avoid the expense and risks associated with invasive studies (those requiring catheterization or injection of radiotracer), they should be ordered appropriately and judiciously. There are three common imaging studies used in the evaluation of children with UTIs.

1. **Renal and Bladder Ultrasound (RBUS)**

   RBUS is safe and appropriate to obtain while a UTI is being treated. It is important to obtain images of both kidneys and the bladder. Any febrile infant with a UTI should undergo an RBUS (especially those who have no documentation of a normal postnatal RBUS). Children 2 to 24 months should undergo RBUS as the only imaging study after a first febrile UTI. Children older than 2 years with recurrent UTI should undergo an RBUS.\textsuperscript{4, 6, 7}
2. Voiding Cystourethrogram (VCUG) (Figures 1 and 3)
A VCUG is helpful in identifying bladder emptying, vesicoureteral reflux (VUR) or urethral obstruction. It should not be obtained until after a child is afebrile for at least 24 hours and is no longer symptomatic. It should be obtained in children less than 2 months with a febrile UTI, in the setting of abnormal anatomy on RBUS (hydronephrosis, renal scarring, etc.), or in other atypical or complex situations. It should be obtained in children 2 to 24 months following a second febrile UTI.6-7

3. Dimercaptosuccinic Acid (DMSA) Renal Scan (Figure 2)
This is a nuclear medicine test, and should not be used routinely after a first febrile UTI, but may be helpful in identifying renal scarring later on. This test will show both renal scarring and active pyelonephritis, but cannot differentiate between the two entities. When evaluating for renal scarring, DMSA scan should not be obtained until four to six months from the time of acute pyelonephritis.8

Figure 1. VCUG from 7-year-old female with history of three febrile UTIs demonstrating right grade 2 vesicoureteral reflux (VUR). Prior imaging showed left grade 3 and right grade 4 VUR.

Figure 2. DMSA scan demonstrating asymmetric renal function and scarring of the upper pole of the right kidney. These findings are stable compared to prior DMSA scans. The child is currently being observed off of antibiotics.

Figure 3. VCUG from 22-month-old female with history of febrile UTI demonstrating bilateral grade 5 VUR. She recently underwent bilateral ureteral reimplantation because of recurrent breakthrough infections despite antibiotic prophylaxis.
antibiotic prophylaxis

Bowel and bladder dysfunction is a major risk factor for recurrent UTIs. The question of whether or not to place a child on prophylactic antibiotics has no universally applicable answer, and there is some controversy regarding the use of antibiotic prophylaxis in children with VUR. It is most commonly used in children with VUR, and more specifically is generally beneficial in children less than 1 year with VUR, or children over 1 year with high-grade VUR. When looking at all children, antibiotic prophylaxis has been shown to substantially reduce the risk of UTI recurrence in patients with VUR, but does not alter the development of renal scarring. A Swedish study has shown that in girls between 1 to 2 years of age with high-grade VUR, prophylaxis decreases both UTI recurrence and new renal scarring. Surgical management of VUR

Multiple options are available for the surgical correction of VUR, including endoscopic, laparoscopic and open techniques. Resolution of VUR is expected in 98.1 percent for those undergoing open surgery and 83 percent for endoscopic therapy. Surgical intervention is considered for higher grades of VUR, breakthrough febrile UTIs or recurrent infections while on antibiotic prophylaxis, or in children with evidence of renal scarring. Prospective randomized controlled trials have shown a decreased occurrence of febrile UTIs in patients after open surgical repair when compared to patients receiving antibiotic prophylaxis.15

when to refer

In some cases, referral to a pediatric urologist should be considered. These situations include patients with VUR (especially high-grade), abnormal RBUS results, congenital genito-urinary tract anomalies, recurrent or severe UTIs, febrile UTI in an infant, or symptoms of urinary urgency, frequency or enuresis in the absence of UTI.

references

Christopher Brown, MD, is currently completing his final year of fellowship in pediatric urology at Nationwide Children’s Hospital. Dr. Brown received his medical degree from St. Louis University School of Medicine, and completed his urology residency at the University of Arizona in Tucson, Arizona. He is excited to be joining the pediatric urology team at Dayton Children’s in the summer of 2018.

CME questions

4. Which of the following is true regarding urine sample collection in children?
   A. Clean catch urine samples are appropriate for all children.
   B. Bagged urine specimens in infants regularly provide reliable urine culture results.
   C. Any amount of bacterial growth on a catheterized specimen is considered significant.
   D. Infants should undergo catheterization or suprapubic aspiration in order to confirm any positive urinalysis.

5. Which of the following is most suggestive of UTI by dipstick?
   A. Nitrite alone
   B. LE alone
   C. Blood alone
   D. Nitrite and LE
   E. Blood and nitrite
   F. LE and blood

6. When treating cystitis, which of the following is true?
   A. A post-treatment urine culture should be obtained to confirm adequate treatment.
   B. Bactrim is effective against the most common urinary pathogens and should be used regularly as first-line therapy for presumed UTI.
   C. Antibiotic choice should be based on local antibiotic resistance patterns when patient-specific culture results are not yet available.
   D. A seven- to 10-day course of antibiotics is typically required.

7. In an 18-month-old female experiencing her first febrile UTI, what imaging study or studies should be obtained?
   A. No imaging is necessary
   B. RBUS only
   C. VCUG only
   D. RBUS and VCUG
   E. RBUS, VCUG and DMSA scan
Antibiotic Stewardship
at Dayton Children’s:

Preventing Vancomycin-Induced Acute Kidney Injury in Hospitalized Pediatric Patients

by Jon Woltmann, MD, and Patricia Christoff, RpH, PharmD
Following the completion of this article, the reader should be able to:

1. Review evidence regarding nephrotoxicity caused by the combination of vancomycin and piperacillin/tazobactam.
2. Identify potential risk factors for drug-induced nephrotoxicity.
3. Understand strategies to mitigate the severity and incidence of nephrotoxicity secondary to the use of the combination of vancomycin and piperacillin/tazobactam.

The pairing of vancomycin with piperacillin-tazobactam has recently been reported as a suspected nephrotoxic combination. A meta-analysis of ten observational cohort studies, including 2,688 adult patients, reported a significant association of the development of AKI with the combined use of vancomycin and piperacillin-tazobactam versus vancomycin alone. In the same meta-analysis, four studies involving a total of 570 adult patients compared the association for the development of AKI with combination of vancomycin plus piperacillin-tazobactam versus the combination of vancomycin plus another beta lactam. No significant association was noted in the latter group, suggesting vancomycin may be paired with other extended spectrum beta lactams without increased risk for AKI.

In 2014, Pratt et al reported the first case series of piperacillin-tazobactam induced AKI in children. Four pediatric patients being treated for febrile neutropenia developed AKI shortly after piperacillin-tazobactam administration, with only one patient with biopsy evidence of interstitial nephritis. The authors, although attributing all four cases of AKI to possible interstitial nephritis, theorized that the severity of AKI was exacerbated by the addition of vancomycin. Goal vancomycin serum concentrations were not reported.

A young patient admitted to the pediatric intensive care unit for concerns of potential pneumonia was the subject of the first published case report of a child experiencing AKI while receiving both vancomycin and piperacillin-tazobactam in 2016. On the third hospital day, the piperacillin-tazobactam was maximized at 100 mg/kg/dose IV every six hours and the vancomycin dose was increased to 18.5 mg/kg/dose IV every six hours following a subtherapeutic trough.
On hospital day four, her serum creatinine had increased from a baseline of 0.4 mg/dl to 1.16 mg/dl, with a corresponding serum vancomycin trough concentration of 37 mcg/ml. No other nephrotoxins were identified in her drug regimen, so both the piperacillin-tazobactam and vancomycin were discontinued and replaced by other antibiotics. Her follow-up vancomycin trough 36 hours after her last dose was 5 mcg/ml and the serum creatinine declined to 0.49 mg/dl on hospital day ten.

Nephrotoxicity occurred in 3.8 percent of patients receiving vancomycin alone and 23.6 percent of patients receiving the combination therapy (P=0.0001) in a retrospective single center cohort study of 79 pediatric patients treated with vancomycin and 106 patients treated with vancomycin and piperacillin-tazobactam. The authors noted that concomitant nephrotoxic medication, critical illness warranting intensive care admission, and vancomycin troughs of >15 mcg/ml could not be excluded as additional risk factors. Limitations of the study included short duration of treatment, lack of inclusion of intravenous contrast as a potential nephrotoxin, and unequal treatment group sizes.10

A retrospective cohort study of children aged 6 months to 18 years who were hospitalized for more than three days and received vancomycin in addition to one other anti-pseudomonal beta lactam antibiotic was just published in October 2017. Among this cohort of 1,915 patients from six pediatric hospitals, 157 patients developed antibiotic associated AKI. After adjusting for age, level of care, receipt of other nephrotoxins and hospital, the antibiotic combination of vancomycin plus piperacillin-tazobactam was associated with a higher odds ratio of AKI each day compared with vancomycin plus one other antipseudomonal beta lactam combination.11

### Antibiotic stewardship team response

Shortly after the stewardship team was developed in 2011, an evidence-based guideline for dosing and monitoring of vancomycin was implemented. The Infectious Disease Society of America (IDSA) recommended that goal troughs of 15-20 mcg/ml must be set not only for severe infections but for all infections caused by methicillin resistant Staphylococcus aureus (MRSA).12 The rationale is based on the

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**Table 1. Expanded vancomycin monitoring**

1. Serum creatinine monitoring of patients on vancomycin should be obtained every 48 hours. This will not apply to pre-op or one-time doses.

2. A vancomycin trough should be obtained if serum creatinine has risen ≥ 1.5 x in past 48 hours.

3. A Best Practice Alert will fire if a patient has a vancomycin order and no basic metabolic panel or serum creatinine ordered within the past 48 hours.

4. Pharmacists will be provided the ability to order vancomycin levels, especially in circumstances where levels are lacking and the provider cannot be contacted, in order to make appropriate dosing recommendations.

5. No recommendations will be made for routine monitoring of vancomycin levels other than what already exists in EPIC, i.e. after the fourth dose.

6. In circumstances where the following exists, daily vancomycin and serum creatinine may be recommended by pharmacy:
   a. Patients with serum creatinine increased by ≥ 1.5 times within past 48 hours or baseline or > 0.3 mg/dl or in patients with baseline serum creatinine > 1.0 mg/dl.
   b. Patients receiving concurrent:
      i. Vasopressors
      ii. Loop diuretics
      iii. Piperacillin-tazobactam
      iv. NSAIDS
      v. Aminoglycosides

7. For all vancomycin doses > 4 grams/day or 25 mg/kg every six hours, an ID Consult will be recommended.

8. PRIOR TO RECOMMENDING A DOSAGE INCREASE
   a. Assess patient’s clinical status by reviewing current vital signs. Additional information may be found in recent progress notes and/or by asking the resident their assessment of the patient’s current clinical status.
   b. If recommended dose exceeds 4 grams/day and resident has not called infectious disease, please confirm dose increase with infectious disease.
fact that the vancomycin minimum inhibitory concentration (MIC) for MRSA at Dayton Children’s is ≥ 1 mcg/ml for greater than 50 percent of our isolates. In order to achieve the recommended area under the serum concentration time curve to MIC ratio (AUC:MIC) of 400, goal troughs of 10-20 mcg/ml must be achieved for adequate efficacy. A standard dose of 15 mg/kg total body weight intravenously every six hours in children less than 16 years of age, and 15 mg/kg total body weight every eight hours in children age 16 years or older was recommended to comply with the IDSA MRSA guidelines for pediatric patients.13

Using these initial recommendations, we encountered three cases of vancomycin-induced nephrotoxicity, two of which required acute hemodialysis in the summer of 2015. A multidisciplinary team was developed to devise new recommendations for improvement in vancomycin monitoring and dosing. All patients who had received vancomycin from 2013 to 2015 were reviewed. Out of 564 patients meeting criteria, a total of 15 patients met the definition of vancomycin-induced AKI according to the Kidney Disease Improving Global Outcomes Group (KDIGO) criteria.14 The criteria adopted by the Dayton Children’s AST were that of KDIGO, which defines AKI as greater than or equal to 1.5 times the baseline serum creatinine or an absolute increase in serum creatinine of 0.3 mg/dl. The records of these 15 patients were then reviewed in depth for all possible risk factors.

In September 2015, expanded monitoring criteria were approved by the ad hoc multidisciplinary committee and implemented by the AST (Table 1). A quality improvement project was then undertaken to determine if the intervention resulted in reduced incidence or severity of vancomycin-induced AKI. The results of that project can be found in Figure 1. Implementation of the expanded vancomycin monitoring criteria reduced the severity of vancomycin-induced AKI as measured by the ratio of the maximum serum creatinine to baseline creatinine in patients who had vancomycin troughs of > 20 mcg/ml. A significant decrease in the median maximum serum creatinine to baseline creatinine ratio from 6.0 to 1.80 was noted in the following two years from implementation of the expanded monitoring tool. The incidence, however, has remained fairly constant at nine cases per year prior to the intervention to an average of eight (10.6) per year after the intervention.

The most common risk factors for vancomycin-induced AKI both before and after implementation of the expanded monitoring criteria are listed in Table 2. Following implementation, four of the most common risk factors declined in incidence; however, concurrent use of piperacillin-tazobactam emerged from the last to the number one risk factor for vancomycin-induced AKI. All six patients experiencing this adverse effect in 2017 had been receiving the combination. In five patients, the AKI developed within 24 to 48 hours of initiating the combination. Although the addition of piperacillin-tazobactam to vancomycin was originally recognized as a potential risk factor, the rapidity with which the nephrotoxicity can occur was not previously appreciated.

The mechanism by which the combination of piperacillin-tazobactam and vancomycin appear to increase nephrotoxicity is unknown. Piperacillin has been shown to decrease tubular secretion of flucloxacillin by competitive inhibition of the tubular secretion site by piperacillin in healthy volunteers. This inhibition appears to be greater at higher piperacillin doses.15 A similar phenomenon has been reported for methotrexate and piperacillin in the rabbit model. The renal clearance of methotrexate was significantly reduced in the presence of piperacillin.16 If similar renal clearance inhibition occurs with vancomycin, increased nephrotoxicity may result.

### Table 2. Risk factors

<table>
<thead>
<tr>
<th>Risk factors before implementation of 2015 enhanced monitoring guideline, in order of frequency</th>
<th>Risk factors after implementation of 2015 enhanced monitoring guideline, in order of frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medically complex (&gt;3 concurrent diagnoses)</td>
<td>Concurrent piperacillin-tazobactam</td>
</tr>
<tr>
<td>Initial trough &gt;15 mcg/ml</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Duration &gt; one week</td>
<td>Medically complex (&gt; three concurrent diagnoses)</td>
</tr>
<tr>
<td>Concurrent NSAIDS</td>
<td>Initial serum concentration &gt;15 mcg/ml</td>
</tr>
<tr>
<td>Furosemide</td>
<td>Concurrent NSAIDs</td>
</tr>
<tr>
<td>Vasopressors</td>
<td>Contrast within 24 hours</td>
</tr>
<tr>
<td>Concurrent piperacillin-tazobactam</td>
<td>Vasopressors</td>
</tr>
</tbody>
</table>
Next steps for the Dayton Children’s AST include identifying and promoting via clinical practice guidelines safe and effective alternative combinations of antibiotics for empiric gram-negative and gram-positive infections, which could reduce both the incidence and severity of vancomycin-induced AKI.

References


**Figure 1. Vancomycin - induced kidney injury**
Jon Woltmann, MD, is a pediatric infectious disease physician and is involved with the antibiotic stewardship team at Dayton Children’s. He obtained his doctorate of medicine at Northeast Ohio Medical University. He completed his pediatric residency training at the Children’s Hospital of the King’s Daughters in Norfolk, Virginia, and his fellowship training at Cincinnati Children’s Hospital Medical Center.

Patricia Christoff, RPh, PharmD, BCPPS, AE-C, is a clinical pharmacist who serves on the Antibiotic Stewardship Team and serves as coach for the Intermediate Quality Improvement Course at Dayton Children’s. In addition, she helps provide the investigational drug service for clinical trials conducted at Dayton Children’s. She completed her doctor of pharmacy degree at the State University of New York at Buffalo where she also completed a fellowship in pharmacokinetics.

CME questions

8. Early attempts to reduce vancomycin induced-AKI were successful in:
   A. Reducing the incidence of AKI
   B. Reducing the severity of AKI
   C. Providing alternatives to vancomycin use
   D. Reducing both the severity and incidence of AKI

9. What is the rationale for attaining initial goal vancomycin troughs of 15 to 20 mcg/ml at Dayton Children’s?
   A. MRSA treatment recommendations published by the Infectious Diseases Society of America
   B. The fact that more than 50 percent of the MRSA vancomycin MICs at Dayton Children’s are ≥ 1 mcg/ml
   C. The fact that the goal vancomycin AUC:MIC ratio for the treatment of MRSA is < 400
   D. Answers A and B are correct
   E. Answers B and C are correct

10. Expanded vancomycin monitoring criteria at Dayton Children’s includes:
   A. Obtaining vancomycin troughs earlier in the course of therapy, i.e. after the second dose
   B. Restricting the use of piperacillin to the Infectious Disease Service
   C. A best practice alert if vancomycin has been ordered and no basic metabolic panel or serum creatinine has been ordered in the past 48 hours
   D. A maximum dose of 6 grams per day
Primary Dysmenorrhea in Adolescents

by Heather Stewart, MD
Menstrual cramps are the most common gynecologic symptom reported across all ages and ethnicities. Dysmenorrhea may affect upwards of 90 percent of women during their lifetime.\(^1\) It can result in school absenteeism, decreased social interactions with peers and withdrawal from sports participation, yet less than 15 percent of dysmenorrhea sufferers seek care from a medical provider for their symptoms. Most people have attempted home remedies and over-the-counter treatment options, which studies show have variable results.\(^2\)

Primary dysmenorrhea is defined as pain with menstruation not attributable to pelvic pathology or abnormality. The onset of routine ovulation is thought to trigger the development of primary dysmenorrhea in adolescence. Secondary dysmenorrhea is due to a pelvic abnormality, such as endometriosis. Initiation of medical therapy should not be delayed while establishing a precise diagnosis as it is likely to alleviate symptoms for both primary and secondary dysmenorrhea.\(^3\)

**Diagnosis**

The first step to treating primary dysmenorrhea is screening for menstrual health. The American Academy of Pediatrics (AAP) endorses the recommendations of the American College of Obstetrics and Gynecology (ACOG) found in Committee Opinion No. 651, “Menstruation in Girls and Adolescents: Using the Menstrual Cycle as a Vital Sign.”

By engaging the patient and family in questions about menses, we open the discussion for when and what to expect with puberty and menarche. These conversations should start around age 7 or 8 years to be preparatory for the changes that come with puberty.

The median age for menarche in the United States is still 12.4 years. Typically, first menses occurs within two years of breast budding, and should be evaluated if not present within three years. Breast development is the first stage of puberty for females and can occur as young as 8 years old. On physical exam, menarche typically occurs once a patient reaches a sexual maturity rating (SMR) of four. Patients who have not started breast development by age 13 years, or have not reached menarche by age 15, should be referred for further evaluation.\(^4\)

When patients present for a variety of concerns, incorporate questions about their menstrual history into your routine screening questions. Ask for the first day of the last menstrual period (LMP) with a year-at-a-glance calendar nearby when obtaining vital signs. A more thorough history should be obtained at least once a year to screen for the presence or absence of dysmenorrhea, any impact on school or lifestyle, the interval between periods, duration of bleeding and the amount of daily menstrual product use (Table 1). Any identified abnormalities can then be addressed in followup or referred for further investigation and treatment. Routine monitoring for menstrual health helps reinforce its importance to patients and families.

### Table 1. Reference for normal menstruation in adolescents

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age of onset</td>
<td>12.43 years</td>
</tr>
<tr>
<td>Average day cycle length</td>
<td>32.2 days the first year following menarche</td>
</tr>
<tr>
<td>Cycle length normal range</td>
<td>21 to 45 days</td>
</tr>
<tr>
<td>Duration of bleeding</td>
<td>7 days or less</td>
</tr>
<tr>
<td>Number of menstrual products used per day</td>
<td>3 to 6 pads or tampons</td>
</tr>
</tbody>
</table>
Menstrual cramps typically present between 12 to 18 months after the first menstrual cycle with primary dysmenorrhea. While primary dysmenorrhea can present earlier than 12 months, the presence of dysmenorrhea at the onset of menarche should raise suspicion for an obstructing genital tract malformation. Primary dysmenorrhea accounts for 90 percent of menstrual pain in adolescents. It is due to the physiologic response to progesterone withdrawal seen after involution of the corpus luteum.

After progesterone levels decline, phospholipids are released from cell membranes. The fatty acids are converted to arachidonic acid (AA) by enzyme phospholipase A2. AA is then converted by cyclooxygenase into prostaglandins, then into leukotrienes via lipoxigenase. Prostaglandin F2-a (PGF2a) acts locally on the myometrium causing hypercontractility and vasoconstriction of the arterioles, leading to uterine ischemia and pain. Leukotrienes C4 and D4 have also been correlated with the severity and occurrence of menstrual pain. The actions of prostaglandins and leukotrienes are also responsible for the systemic symptoms of primary dysmenorrhea.5,6

Patients presenting with primary dysmenorrhea often complain of a colicky, lower abdominal pain starting with or close to the onset of menstrual bleeding and lasting up to 48 to 72 hours. Some individuals may report pain that starts one to two days before their period. Additionally, they may complain of associated headaches, nausea, vomiting, diarrhea, back pain, fatigue and dizziness.

Factors associated with the development of primary dysmenorrhea include age of menarche less than 12 years; first-hand and second-hand tobacco smoke exposure; a body mass index of less than 20; a history of long, heavy or irregular periods; and the presence of premenstrual symptoms.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Presentation</th>
<th>Evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary Dysmenorrhea</strong></td>
<td>Recurrent, cramping lower abdominal pain with onset of menses, generally starting 12 to 18 months after menarche</td>
<td>Clinical history, consider urine pregnancy and STI tests</td>
</tr>
<tr>
<td><strong>Secondary Dysmenorrhea</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endometriosis</td>
<td>Cyclic menstrual pain; pelvic pain may be associated with dyspareunia, dyschezia or dysuria; fails to respond to first-line therapy</td>
<td>Treat with first-line therapy; if fails to respond; pelvic ultrasound and referral to gynecology</td>
</tr>
<tr>
<td>Adenomyosis</td>
<td>Menorrhagia, possible intermenstrual bleeding; boggy, tender, enlarged uterus on exam</td>
<td>Referral; transvaginal* ultrasound or magnetic resonance imaging</td>
</tr>
<tr>
<td>Uterine myomas</td>
<td>Cyclic pelvic pain with menorrhagia, occasional dyspareunia</td>
<td>Referral; transvaginal* ultrasound for fibroids</td>
</tr>
<tr>
<td>Obstructive lesions of the genital tract</td>
<td>Pain with onset of first menstrual cycles; known renal tract anomaly</td>
<td>External genital exam, pelvic ultrasound, MRI; referral</td>
</tr>
<tr>
<td><strong>Other Causes of Menstrual Pain</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pelvic inflammatory disease</td>
<td>Lower abdominal pain in sexually experienced patient; pelvic exam findings of cervical motion tenderness, uterine tenderness, and/or adnexal tenderness; possible fever greater than 101°F, mucopurulent cervical or vaginal discharge, nausea, vomiting</td>
<td>Pelvic examination; elevated CRP or ESR; testing for Neisseria gonorrhoeae and Chlamydia trachomatis is confirmatory</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>Severe, sharp lower abdominal pain, history of missed period, abnormal uterine bleeding, possibly localized to one side; signs of shock/complications</td>
<td>Pregnancy test; emergent ultrasound to demonstrate extrauterine gestation</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td>Dyschezia, melena, hematochezia, pain not associated with menstrual cycles</td>
<td>Gastroenterology referral; screen for rectal STI if exposure</td>
</tr>
<tr>
<td>Interstitial cystitis</td>
<td>Suprapubic pain, noncyclic, associated with urinary symptoms; pain radiates toward groin and is relieved by voiding</td>
<td>Urinalysis (rule out infection) and urology referral</td>
</tr>
</tbody>
</table>

**Table 2. Differential diagnosis of dysmenorrhea**

(*Transvaginal approach is not recommended for younger patients, consider MRI*)
The impact of dysmenorrhea on involvement in activities, such as school, sports and social events, is an important indicator of severity and need for intervention. School absenteeism is more common with moderate to severe dysmenorrhea. Approximately 14 percent of adolescents with dysmenorrhea report missing two or more days a month due to pain. The use of analgesics should also be determined, with emphasis on dose and frequency, as many people do not use over-the-counter medications effectively. Individuals may have tried a variety of options, including supplements and complementary medicine, in an attempt to treat the pain.

The differential for dysmenorrhea includes gynecologic causes of secondary dysmenorrhea and non-gynecologic causes (Table 2). Endometriosis is the most common gynecologic cause of secondary dysmenorrhea and occurs more frequently in adolescents with a positive family history in a first-degree relative. Additional clues to differentiating primary and secondary dysmenorrhea include symptoms that are unresponsive to first-line therapy with nonsteroidal anti-inflammatory drugs (NSAIDs) and hormonal therapy; new onset of symptoms in a sexually active adolescent; atypical pain symptoms of dysuria, dyschezia or dyspareunia; or pelvic pain unrelated to the menstrual cycle. Patients with known renal tract abnormalities may have underlying Mullerian anomalies that present as dysmenorrhea with the first menstrual cycle. Part of a thorough history for dysmenorrhea includes inquiry into prior sexual behavior and safe sex or contraceptive practices. Ensuring confidentiality practices in your office can help youth feel more comfortable with disclosing their history. For some youth, dysmenorrhea may be an acceptable reason to obtain needed contraception. Screen for signs and symptoms of sexually transmitted infections or pelvic inflammatory disease, and perform a pelvic exam if necessary to rule out this cause of new onset pelvic pain.

The physical examination for primary dysmenorrhea should include an abdominal exam for any palpable abnormalities. For sexually naive adolescents with a typical primary dysmenorrhea history, pelvic examination is not necessary for diagnosis. Consideration of an external genital exam should be made to rule out the presence of an abnormality of the hymen. However, if the history is suggestive of causes other than primary dysmenorrhea, or the patient is not responding to first-line therapy for primary dysmenorrhea, a pelvic examination is indicated.

Imaging and laboratory testing are not recommend
ded for primary dysmenorrhea. Testing for pregnan
cy, N. gonorrhoeae and Chlamydia trachomatis are important in sexually active adolescents. Pelvic ultrasound should be used first when evaluating for pelvic anomalies as a cause of secondary dysmenorrhea, or if the patient has failed to respond to adequate therapy over three to six months. A transvaginal ultrasound is generally not recommend
ed for patients with an intact hymen or prior to coitarche. In such cases, if the images cannot be obtained via transabdominal ultrasound, then magnetic resonance imaging may be recommended to avoid traumatizing the patient. A referral should be made to adolescent gynecology for patients suspected of secondary dysmenorrhea.

### treatment

Treatment for dysmenorrhea should take into consideration what methods the patient has previously tried since many strategies are available over the counter. Non-pharmacologic methods including exercise, yoga, dietary modification and acupuncture have been investigated, but the results are conflicting from small studies. Topical heat has been shown to provide symptomatic relief of pain and is an inexpensive, easily accessed option. A variety of supplemental herbs and vitamins such as ginger, fish oil, vitamin B1 or rosehips may be used by some patients. A Cochrane review from 2016 of dietary supplements for dysmenorrhea (27 studies, 3,101 women) found a lack of high-quality evidence to support recommendations for use.

Pharmacologic management of dysmenorrhea is step-wise and should consider additional benefits and contraceptive needs.
First-line treatment options include NSAIDs, combined hormonal therapy and long-acting reversible contraceptives (LARCs). The goal of pharmacologic treatment is to reduce prostaglandin production and action, as well as menstrual flow.

NSAIDs work to inhibit the cyclooxygenase pathway preventing the production of PGF2α from arachidonic acid. A recent Cochrane review of 80 randomized controlled trials concluded that NSAIDs are superior to placebo in the treatment of dysmenorrhea. There is not enough evidence to recommend one NSAID over another. When choosing NSAIDs, consideration should be made for ease of use and adherence to dosing recommendations. NSAIDs should be started one to two days prior to the onset of menses if the cycle is predictable, or immediately with onset if it is not predictable. Scheduled dosing should continue for the first two to three days of the menstrual cycle. Use of a loading dose has been shown to have some improvement over pain control (Table 3).

Combined hormonal therapy (CHC) in the form of the contraceptive vaginal ring, the transdermal contraceptive patch and oral contraceptive pills (OCP), provide benefit by suppressing monthly ovulation and limiting endometrial growth. These actions thereby reduce the production and release of prostaglandins and leukotrienes. Low-dose OCP (20 mcg ethinyl estradiol and 100 mg levonorgestrel) has been found to effectively reduce dysmenorrhea symptoms in adolescents. Small studies, mostly in adult women, have shown more improvement in pain symptom reduction with OCPs or the vaginal ring (ethinyl estradiol and etonogestrel) than the transdermal patch (20 mcg ethinyl estradiol and 150 mcg norelgestromin daily). Additionally, the use of extended cycling to reduce the number of hormone-free days has been shown to decrease the frequency of dysmenorrhea.

Depot medroxyprogesterone acetate (DMPA) is another option available, but requires injections every 11-13 weeks and has a potential side effect of weight gain most adolescents find unacceptable. Concerns about reduction in bone mineral density accumulation during adolescence with prolonged use of DMPA should be reviewed with the patient and/or family.

LARCs include the levonorgestrel intrauterine device (LNG-IUD) and the subdermal single rod etonogestrel implant. The mechanism of benefit for dysmenorrhea with the LNG-IUD is mainly local action on the endometrium to reduce endometrial development and growth. The implant reduces dysmenorrhea through inhibiting ovulation with sufficiently elevated progestin levels. These methods are effective for years and provide a reliable, highly effective contraceptive benefit. Training to provide these services in your office is available, or referral to a skilled provider is needed for placement and surveillance of LARCs.
CME questions

11. Primary dysmenorrhea typically starts 12 to 18 months after the first menstrual period.
   A. True  
   B. False

12. Older age of menarche (more than 13 years) and short, light periods are factors associated with dysmenorrhea.
   A. True  
   B. False

13. Which of the following are treatment options for primary dysmenorrhea?
   A. Naproxen 500 mg every 12 hours  
   B. Ibuprofen 200-600 mg every 6 hours  
   C. Oral contraceptive pill with 20 mcg ethinyl estradiol and 100 mg levonorgestrel  
   D. Levonorgestrel intrauterine device  
   E. All of the above

author

Heather Stewart, MD, FAAP

Heather Stewart, MD, FAAP, is the medical director for the adolescent young adult medicine department at Dayton Children’s and assistant professor of Pediatrics at Wright State University Boonshoft School of Medicine. She completed her fellowship at San Antonio Uniformed Services Health Education Consortium in Texas during her 12-year tenure in the United States Air Force. She collaborates several times a year with medical students through Boonshoft PRIDE to improve knowledge of LGBTQ-unique health care needs and delivery. She is also a registered LARC provider.

text

Screening for the presence of dysmenorrhea provides an opportunity to improve the quality of life and level of functioning for your adolescent patients. The prevalence of dysmenorrhea in adolescents is reported as high as 93 percent (range 48 percent to 93 percent), causing many people to believe it is an inevitable part of menstruation. Incorporating a menstrual history into office visits opens the discussion for health prevention and treatment before unnecessary negative effects on sports, school and home. A step-wise approach to dysmenorrhea treatment makes it manageable for both patient and provider.

references

Dayton Children’s physicians have been peer-selected to the Best Doctors in America® List for 2017-2018. Approximately four percent of doctors in America earn this prestigious honor, as a result of the biennial Best Doctors Poll. Each listed physician has received peer consensus as a physician from whom other physicians would seek care and is verified as being clinically active with an active medical license free from disqualifying disciplinary actions.

A selected Best Doctor doesn’t remain in the database forever. The peer-review process requires every listed physician to be re-evaluated by their peers in each poll. Best Doctors does not pay physicians to be included in the database, nor can physicians or their organizations pay Best Doctors. A physician cannot apply to become a Best Doctors physician. The only way for a physician to be selected is to be nominated by and then receive voting consensus from current Best Doctors physicians.

As a result, Best Doctors has a proprietary, global database of physicians—free from commercial and financial bias—numbering more than 50,000 in over 450 specialties and subspecialties worldwide. There are close to 40,000 Best Doctors physicians in the U.S.
Dayton Children’s Hospital received the 2018 Women’s Choice Award® as a Best Children’s Hospital

Dayton Children’s Hospital is named as a Best Children’s Hospital by the Women’s Choice Award®, America’s trusted referral source for the best in health care, as well as Best Children’s Hospital for Emergency Care.

The list of 67 award winners, including Dayton Children’s Hospital, represents hospitals that have met the highest standards for childcare.

“At Dayton Children’s, moms know we treat their kids like they are our own,” says Deborah A. Feldman, president and CEO. “We are honored that the Women’s Choice Award proves that trust.”

The designation of Best Children’s Hospitals is based on a point system and self-reported data from almost 100 children’s hospitals in the nation.

Hospitals are judged according to the availability of specific services and capabilities, including:

- Family centered care
- Family sleeping/living accommodations
- Dedicated pediatric emergency department
- Pediatric ICU
- Neonatal ICU
- Child life specialists
- Use of telehealth technologies
- Participation in pediatric health research
- Accreditation by the Joint Commission
- Pediatric Trauma Center accreditation
The State of our Children’s Health

Survey of parents, pediatricians and partners outlines state of children’s health in the Dayton region and sets community health agenda.

Mental health and addiction, chronic disease and maternal and infant health are the top pediatric health concerns in the Dayton region, according to the findings of the 2017 Community Health Needs Assessment. Conducted every three years, the health assessment is provided by the hospital and will be used to develop new programs and strategies to impact these important pediatric health issues.

“We are proud to be a part of this process and have funded the study for the past 15 years,” says Patti Scheer, Dayton Children’s Foundation board chair. “This research is important to ensure Dayton Children’s has the data necessary to identify and support efforts to address the region’s most critical pediatric health concerns.”

Among the findings in the 2017 Assessment:

**Mental Health and Addiction** (includes emotional wellbeing, mental illness conditions and substance abuse disorders)

- 9% of children experienced 2 or more adverse childhood experiences.

- 19% of mothers and 11% of fathers of 0 to 5 year olds rated their mental and emotional health as fair or poor.

**Chronic Disease** (includes conditions such as heart disease, diabetes and asthma, and related clinical risk factors-obesity, hypertension and high cholesterol, as well as behaviors closely associated with these conditions and risk factors: nutrition, physical activity and tobacco use)

- 50% of children ages 0 to 11 were classified as overweight (14%) or obese (36%) by Body Mass Index (BMI) calculations.

- 9% of parents were told by a doctor that their child had asthma.

- 13% of parents reported experiencing food insecurity, increasing to 19% of parents with a child age 0 to 5 and 60% of parents with incomes less than $25,000.

- 50% of children ages 0 to 11 were classified as overweight (14%) or obese (36%) by Body Mass Index (BMI) calculations.

- 9% of parents were told by a doctor that their child had asthma.

- 13% of parents reported experiencing food insecurity, increasing to 19% of parents with a child age 0 to 5 and 60% of parents with incomes less than $25,000.

- Maternal and Infant Health (includes infant and maternal mortality, birth outcomes and related risk and protective factors impacting preconception, pregnancy and infancy, including family and community contexts)

- 11% parents reported their child was born premature, increasing to 21% of African American parents.

- 30% of parents of 0 to 5 year olds reported their child was never breastfed.

- 41% of parents of 0 to 5 year olds reported their child slept in bed with a parent or another person as an infant.

“With 20 percent of a child’s health determined by his or her environment and 40 percent determined by behavior – there is a great need to ensure children have healthy and safe places to live, learn and play,” says Deborah A. Feldman, president and CEO at Dayton Children’s. “We use our community health needs assessment to build a children’s health agenda which focuses on wellness, access to care and unmet community needs – elements impacting children’s health beyond the walls of a hospital.”
program evaluation

1. The material presented in this publication met the mission to enhance health care delivery in our region through education based on the essentials and policies of the Accreditation Council for Continuing Medical Education.
   - Strongly agree
   - Agree
   - Neutral
   - Disagree
   - Strongly disagree

2. Did the material presented in this publication meet the educational objectives stated?
   - Yes
   - No

3. Did the material presented in this publication have a commercial bias?
   - Yes
   - No

4. Please rate the contents of this issue using the following scale:
   1 = Poor, 2 = Fair, 3 = Good, 4 = Very good, 5 = Excellent
   (Circle one response for each.)

<table>
<thead>
<tr>
<th>Poor</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<th>Excellent</th>
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</thead>
<tbody>
<tr>
<td>Timely, up-to-date?</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>Practical?</td>
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<td>Relevant to your practice?</td>
<td>1</td>
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5. Please describe any changes you plan to make in your clinical practice based on the information presented in this program.

   _______________________________________________

6. Are there any other topics you would like to have addressed in this publication or future educational programs for health care providers?
   - Yes
   - No  If yes, please describe:

   _______________________________________________

7. Please describe how you will incorporate information obtained from this publication into your practice.

   _______________________________________________

8. Letter to the editor — Letter to the editor may be emailed to alters@childrensdayton.org or attached to this evaluation and may be published in the next issue.

program test

to obtain CME credit you must:

Read and reflect on each article.
Answer the questions from each article and complete this test. 70 percent correct answers are needed to obtain the full 4.0 AMA PRA Category 1 Credits™.
Complete the program evaluation.

Return your completed test and program evaluation by email, mail or fax to:

Sue Strader, coordinator
Department of Continuing Medical Education
Dayton Children’s
One Children's Plaza,
Dayton, OH 45404-1815
Fax: 937-641-5931
E-mail: straders@childrensdayton.org

Take test online: childrensdayton.org/providers

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your answers to CME questions
(Please circle the BEST answer.)

1. a b c d e
2. a b c d e
3. a b c d
4. a b c d
5. a b c d e f
6. a b c d
7. a b c d e
8. a b c d
9. a b c d e
10. a b c d
11. true false
12. true false
13. a b c d e

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