URGENT CARING

A Peer Reviewed Publication from the College of Urgent Care Medicine

THIRD QUARTER, 2023

Volume 7, Issue 3

Table of Contents

Contents

A Message from the CUCM President, Chris Chao, MD

September is Concussion Awareness Month

Heads up! Did you know that September is Concussion Awareness Month and September 15, 2023 is Concussion Awareness Day? And did you know that the Urgent Care Association (UCA) and the College of Urgent Care Medicine (CUCM) have joined the Concussion Awareness Now (CAN) coalition? CAN has initiated a public service campaign to educate the public on the dangers of head injuries and the importance of seeking a medical evaluation following a head injury. Concussions affect more than 3.8 million people a year and patients with head injuries frequently present to Urgent Care centers for treatment. In the future, we will be developing resources to help clinicians improve management of head injuries in the Urgent Care setting. In the meantime, check out the Melon Family at Concussion Awareness Now, a site that provides an entertaining but informative spin on the dangers of head injuries.

Become a Champion for Concussion Awareness

The CAN Coalition is asking all of us to go to their website to join their cause by becoming a concussion Awareness Now Champion. Join here.

As summer fades into fall, the College is busy preparing for our 2024 Urgent Care Association convention. Several of the CUCM board of directors will rotate off the board due to term limits. Thus, **the College is looking for leaders who are passionate about advocating and growing Urgent Care as a specialty.** If this is you, we encourage you to run for a CUCM board position! Additionally, we are looking for dynamic speakers who want to share their clinical knowledge at the 2024 Convention. If you are interested, please let us know by filling out a Session Proposal.

This year, the College published an Urgent Care competency list which lists the skills we believe are necessary to be an effective Urgent Care provider. Later this year, we plan to release a clinical competency exam in partnership with Hippo Education to assist providers with knowledge gap analysis.

Finally - hang in there! - many of us are facing high volumes due to yet another COVID-19 surge. With cold and flu season around the corner as well as continued staff shortages and burnout, this may be a challenging couple of months. Please do not hesitate to reach out to your fellow College members for support or advice! We are all here for each other.

Chris Chao, MD
President, College of Urgent Care Medicine Board of Directors

From the Editors-in-Chief

Tracey Davidoff, MD, FCUCM

Cesar Mora Jaramillo, MD, FAAFP, FCUCM

Embracing Pioneering Progress in Urgent Care

Welcome to this issue of Urgent Caring, where we proudly celebrate the relentless pursuit of Urgent Care clinicians to pioneer progress within the Urgent Care field. In the continuously evolving healthcare system, Urgent Care centers have demonstrated not only resilience but how crucial our services are for the communities we serve, especially during times of crisis – in addition to helping overwhelmed Emergency Departments.

Urgent Care centers have expanded rapidly because of longer hours, quick access, location convenience, gaps in primary care, and high costs of emergency room visits. The growth highlights the crisis in the U.S. primary care system. According to the Association of American Medical Colleges, a shortage of up to 55,000 primary care physicians is expected in the next decade.

Healthcare challenges are shaping the way we deliver essential medical services to our communities. As we embark on this journey together, let's not forget how Urgent Care Centers are pioneering efforts to achieve high-quality care and increase accessibility to meet the growing demand, all while navigating the challenges to care like staffing shortages, burnout and limited access to primary care services.

Urgent Care leaders continuously try to find ways to overcome these challenges, solidifying the importance of UCCs within the healthcare system.

With this in mind, we want to celebrate the remarkable experience that was the recent meeting of the Clinical Consortium, which brought together Urgent Care medical thought leaders to discuss important topics in Urgent Care: Antibiotic Stewardship, leadership in Urgent Care, forging a path to excellence, staffing shortages (a constant challenge), and the role of Urgent Care with primary care services. We want to thank everyone who attended and the presenters who made the event such an impactful experience. We cannot wait for next year's meeting!

Urgent Caring continues to expand and serve as a reliable source addressing unique clinical cases and high-yield educational topics to help you during your day-to-day clinical challenges at work. We want to thank the new members of our Editorial Board and our section editors for their continued support! If you want to join the team, do not hesitate to reach out to us. We are advancing the specialty together and need to support each other in this journey.

Last but not least, we want to thank our peer reviewers for this issue: Sean McNeeley, MD, FCUCM, Joseph Toscano, MD, FCUCM, Ivan Koay, MD, and Brigham Temple, MD. We appreciate your expertise and commitment to Urgent Care.

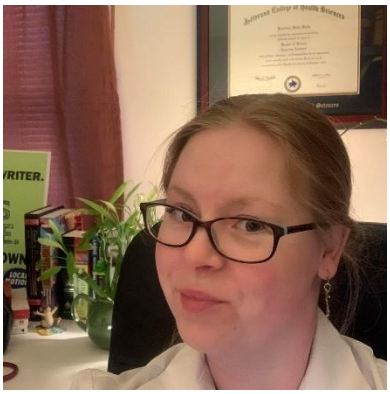
Thank you all for your continued support in our collective pursuit of advancing the specialty of Urgent Care medicine and making a positive difference in the lives of those we serve.

Tracey Q. Davidoff, MD, FCUCM

Cesar Mora Jaramillo, MD, FAAFP, FCUCM

Editorials and Opinions—When Clinicians Call Out

Kateland Kelly, PA-C: The Write Assistant



My fingers hovered anxiously over the screen, and I closed my eyes tapping the fatal message, "I can't come in to work today."

I held my breath, waiting for the inevitable response from my manager or regional manager in our group text before one finally floated through cyberspace, "Is there any chance you can come in later today? If you don't, we'll have to close both clinics."

My eyebrows shot up but before I could respond to the text, another wave of nausea gripped me, and I vomited mucus onto the towel lining my bathroom floor. Her text was filled with subtext: If you don't show up, no one will get paid and patients will suffer. This is not an uncommon dilemma for medical providers: If we get sick, who looks after our patients? I looked up at the sky, praying for an answer and asked myself what would I tell a patient in a similar situation…How would I counsel them?

I would counsel them to take a sick day, curl up in the bedroom with a hot pad, take their prescribed medication, and hydrate. I would counsel them to enforce boundaries with their employer because sick people can't heal sick people.

While it is true, I am an Urgent Care/emergency Physician Assistant, I'm also a human, and I live with multiple chronic health conditions. My employer doesn't need to know the specifics of my health conditions but in America, we often sacrifice our medical privacy for a modicum of understanding. The sad thing is, the chronically ill are rarely understood, and unless we are productive (like a clinician that can rake in high numbers on an outpatient shift), then we are dismissed as "bad employees."

The implicit suggestion in the text from my boss suggested I take additional ownership in the clinic for support staff, and that it is my responsibility to keep it staffed no matter what.

Except, it's not.

Three years ago, I would have literally killed myself working through pain, illness, or injury but things have changed since the pandemic. I value my own body more since realizing I'm only a diagnosis away from death. Sounds dramatic, but when you've lived through a few active gunmen, you start to realize what is and isn't important anymore while on the job.

The concept of quiet quitting has been floating around as the topic du jour but I like to think that calling out sick when you need to is simply being a human. Quiet quitting is defined as, "a response to the burdensome demands of employment. It is often linked to employee burnout and involves deliberate

disengagement from work. Employees are performing the absolute least necessary and establishing clear limits rather than quitting their employment," according to Sociology Plus.

The reason I push back against this concept is because we aren't products and we aren't robots; sometimes we need the tincture of time to heal. That shouldn't be labeled as a deficiency or a withdrawal of any kind, but rather be recognized as a factor in working with other people. Life happens, or at least, that's the lip service our human resource officers spout whenever they hold a company meeting. However, as a clinician, tincture of time is one potion we've never got enough of. But we can start setting healthy examples for our patients when we start acting in our own best interests.

Let me illuminate just how difficult it is for those of us being choked by white collars to call out sick.

Have you ever considered what happens when your doctor calls out sick? Your physician assistant? Your psychiatrist?

We feel immense guilt even considering a day off.

I pushed through my guilt, and this time and I responded: "I wouldn't call out if I didn't need to; I'm sorry but I can't come in today."

I curled up in bed, but not without missing a text and two phone calls from the clinic asking for clarification on various patients. I stared at my phone blinking confused — didn't I just call out sick?

Even though I called out sick and even though I'm paid hourly, I was still contacted during my sick time to manage other patients. I made a conscious decision to not answer any of the calls as I had already taken some pain medication and once I pull that trigger, I don't practice medicine. I spent the day praying to the porcelain gods, but I pulled myself through the fog and came to work the next day. Regrettably, I saw fifty-three Urgent Care patients all desperate for someone to listen to them and while I did my best, by the next morning I was up at 5 a.m. vomiting profusely and doubled over with diarrhea.

My period was vengeful.

I texted my manager and regional once more, déjà vu washing over me as I felt like a little bit of a failure, "I can't believe I'm doing this again and I'm so sorry, but I need to call out sick. I've been vomiting since 5:00 a.m. and the diarrhea won't stop. I can't come in."

This time the response was more intimidating, "I hope you feel better but this time we need a doctor's note."

Aren't I the person people come to for a doctor's note? What the what?

I responded quickly, referencing Connecticut state law which states employers can only ask for a note explaining three consecutive days off of work in a row. I had not called out multiple in a row, but I did realize it was a holiday weekend, Labor Day to be exact. I understood she was suspicious, but I wasn't

trying to score a day down by the lake, I was just trying to get my symptoms under control so I could get off the toilet.

My regional responded, “It’s the weekend.”

So, what? My internal dialogue screamed at myself. The irony of the weekend was not lost on me, but I don’t think she appreciated it considering how chronically short staffed our Urgent Care centers were. I couldn’t help but reflect on the dark origins of Labor Day and how it began after American laborers were tired of being taken advantage of by their employers. Labor Day, at its very core, is a celebration of the achievements and contributions of American laborers. Sadly, there is no rest for the clinically inclined and apparently, I needed an excuse to take the day off even though there was nothing in my contract to suggest that kind of documentation was needed.

I imagined myself as a medical assistant making a fraction of what the clinician makes, and I realized just how scary that text was to a sense of job security.

Staring back at the black mirror I refused to back down: “Is that company policy or law?”

My heart pounded waiting for a response, but the funny thing is, she never responded. I called her bluff. And I didn’t drag my tired self to go sit in a waiting room where someone would nod, agree with me that I was ill and needed the day off, and write me a performative note wasting their time and mine. What I did was curl back under the covers once my prescriptions kicked in and I realized why I was so upset about the subtext in our conversations about calling out sick:

Are we not adults?

What happened to the sanctity of someone's word?

I’ve seen support staff, medical assistants and receptionists, blue collar workers that lack the clout to call out, nurse their own illnesses in shame while on shift because the pressure to keep working is so high. When people scorn us saying, “No one wants to work anymore,” I can’t help but laugh, and then cry, when I realize it’s because no one wants to work in these conditions anymore. We deserve better. We deserve to be treated like adults, not children trying to play hooky.

When I call out sick as a clinician, I am doing so because I know my body and I know the risks that I can and can’t safely take. I will never put my patients at risk and now that I know my worth, I won’t continue to put myself at risk.

So, consider this my white coat endorsed professional opinion: If you think you need to take a day off to recover from an illness or injury, take the day off and let the corporate overlords figure out how to staff your absence. That’s their job, after all, not yours, even on Labor Day.

U.S. Antibiotic Awareness Week Webinar: Common Myths & New Successes in Urgent Care Stewardship

Kick off U.S. Antibiotic Awareness week by joining Guillermo (Memo) Sanchez, PA-C from the Centers for Disease Control and Prevention (CDC) and Patrick Dolan, MD, for a one-hour presentation brought to you by the College of Urgent Care Medicine. Dr. Dolan serves as the principal investigator in an Urgent Care based study on ABS where Urgent Care centers around the country have moved the needle on appropriate prescribing practices. Memo Sanchez, PA-C recently presented to a group of your peers at the UCA Assembly in Nashville, TN with a great response from the attendees as he identified myths and misconceptions around ABS. His presentation is uniquely targeted to the nuances of the Urgent Care setting with updated data points and industry trends. Both presenters are focused on solutions and successes that have been realized across a diverse group of Urgent Care settings. As Urgent Care medicine has returned to normalcy following the COVID-19 pandemic, it is time to refocus our energy on combatting antimicrobial resistance and fulfilling the role we all have in becoming part of the solution. Clinical and administrative Urgent Care leadership have an opportunity to collaborate to make a difference—and there is something for everyone in the information that will be provided by our speakers.

This webinar is provided as part of the College's commitment to stewardship in collaboration with the CDC. There is no cost to attend.

You don't want to miss what's new and what's working.
Register Here: https://us02web.zoom.us/webinar/register/WN_VnQTtk6_TmCsQG3ntrioCg

Memo Sanchez is an emergency medicine PA and epidemiologist with CDC's Office of Antibiotic Stewardship. Memo is the first author on CDC's Core Elements of Outpatient Antibiotic Stewardship, which was published in 2016. His areas of concentration include creating CDC stewardship guidance, leveraging partnerships to promote appropriate antibiotic use, and supporting implementation of outpatient antibiotic stewardship, including among advanced practice providers and within Urgent Care and telemedicine settings.

Patrick Dolan, MD is an experienced Pediatrician with a demonstrated history of working in the hospital & health care industry. Skilled in Healthcare Consulting, MOC, Pediatric Advanced Life Support (PALS), Emergency Medicine, and Advanced Cardiac Life Support (ACLS). He is a strong healthcare services professional with a Bachelor of Science (B.Sc.) focused in Biology, General from Saint John's University.

History of a Painful Penis - A Case Report

Ivan Koay, MBChB, FRNZCUC, MD

A 40-year-old man walks into the Urgent Care center complaining of a painful penis, having woken up a few hours earlier with the pain. He initially woke up with a painful erection. The pain continued but the erection has subsided a little. He denies any trauma, there was no sexual activity in the preceding hours or previous evening. He had taken some Tylenol a couple of hours prior to presenting to the UCC. He had passed urine a couple of times since the pain started; he denied noticing any blood in the urine during those times. There was no abdominal pain and no fever. There was no other past medical history of note.

Clinical examination noted no fever, pulse of 78 beats per minute and regular, oxygen saturations of 98% on air. He had a normal cardiovascular examination, abdominal examination was unremarkable, genitalia examination showed a semi-tumescent penis that was slightly painful to palpate, non-tender and normal lying testes. Urine dip revealed 1+ blood and 1+ leukocytes, negative nitrates, negative glucose, and ketones.

What are the plausible differentials:

A. Lower urinary tract infection
B. Sexual transmitted infection
C. Cellulitis
D. Priapism
E. More information needed

On further direct questioning, he denied any previous history of STIs and he has one female partner with whom he has been with for the last 10 years and never been unfaithful. He also denies using any other recreational substances. He admits to having a similar episode about a year ago which resolved after going for a walk.

Answer D: Priapism

Priapism is defined as a prolonged and persistent penile erection, unassociated with sexual interest or stimulation, lasting longer than 4 hours. It is divided into three main categories based on the aetiology and pathophysiology of the condition: ischemic, non-ischemic and stuttering priapism.

Ischemic priapism (the most common with 95% of presentations), also termed veno-occlusive or low flow priapism, is a persistent erection marked by rigidity of the corpora cavernosa and little or no cavernous arterial inflow. It consists of an imbalance in vaso-regulatory mechanisms, predisposing the penis to an ischemic environment. The tissue ischemia and increased pressure generated within the corporal bodies lead to pain and rigidity, classically seen with ischemic priapism. This constitutes a true urological emergency.

Non-ischemic priapism, also termed arterial or high-flow priapism, is a persistent erection caused by unregulated cavernous arterial inflow. This generally occurs due to trauma, creating a disruption in the cavernous arterial anatomy, resulting in an arteriolar-sinusoidal fistula. The cavernous environment does not become ischemic secondary to the continuous influx of arterial blood. The corpora are tumescent but

not rigid, and patients typically do not complain of pain with erection, therefore non-ischemic priapism is not an emergency and does not require immediate intervention.

Stuttering priapism, also termed intermittent or recurrent priapism, is characterized by recurrent episodes of ischemic priapism and typically last <4h prior to remission. These episodes may increase in frequency and duration, however, compromising the patient's quality of life and potentially developing into major episodes of ischemic priapism. Both stuttering and ischemic priapism result in the same consequence, namely, ischemic damage to the corporal tissue. Therefore, all episodes of recurrent priapism that progress to prolonged, painful erections should be treated promptly, according to the guidelines set for ischemic priapism.

Diagnosis

Conditions predisposing to ischaemic priapism include sickle cell disease (SCD), assorted haematological dyscrasias, parenteral hyperalimentation, haemodialysis, heparin-induced platelet aggregation, and local primary (penile carcinoma/squamous cell carcinoma, prostatic adenocarcinoma) or metastatic neoplasia (metastases to the penis from prostate, rectosigmoid colon, kidney, urothelial carcinoma of the urinary bladder, chronic myeloid leukaemia). Medications implicated include alpha-adrenergic receptor antagonists (prazosin, terazosin, doxazosin, tamsulosin), anti-anxiety agents (hydroxyzine), anticoagulants (heparin, warfarin), antidepressants and antipsychotics (trazodone, bupropion, fluoxetine, sertraline, lithium, clozapine, risperidone, olanzapine, chlorpromazine, thioridazine), and antihypertensives (hydralazine, guanethidine, propranolol). Second-generation antipsychotics (33.8%), other medications (11.3%), and alpha-adrenergic antagonists (8.8%) account for most reported cases of drug-induced priapism. Alcohol and cocaine may predispose to ischaemic priapism.

Management must begin with a detailed history and physical examination. Diagnosis should focus on identifying any contributory/predisposing conditions, listed above. The duration of priapism, any clinical treatments used, previous priapism episodes, presence of pain, and erectile function status prior to the priapism episode should be noted. In patients with known SCD, it is particularly important to determine the presence of any other systemic symptomatology associated with SCD, such as a sickle crisis. A physical examination involving inspection and palpation of the penis, to assess for the extent of tumescence or rigidity, degree of corporal body involvement, and presence and severity of tenderness, is essential. Abdominal, perineal, and rectal examinations may reveal signs of trauma, pelvic infection, or malignancy. A full neurologic exam may be indicated when a spinal cord injury or lesion is suspected.

Within the UC centers, there is limited other modalities for imaging or laboratory testing to be performed. Patients that present to the UC and have been diagnosed with priapism require referral to the local urological service or emergency department for further work-up and assessment. In secondary care centers, where laboratory and imaging facilities are available, certain tests can be considered. However, these investigations should not delay the referral process but can be used as adjuncts for the referral process.

Laboratory Tests

A cavernous blood gas analysis will provide direct visualization and evaluation of penile blood, serving to provide immediate distinction between the different variants of priapism. In patients with ischemic priapism, the aspirated blood is hypoxic and dark, and typical blood gas values show a partial pressure of oxygen (pO2) of less than 30 mmHg, partial pressure of carbon dioxide (pCO2) of greater than 60 mmHg and a pH of less than 7.25. Conversely, in non-ischemic priapism, the blood is oxygenated and bright red with cavernous blood gas values of a pO2 greater than 90 mmHg, pCO2 less than 40 mmHg, and pH 7.40,

consistent with normal arterial blood at room air. A complete blood count, white blood cell differential, and platelet count which may reveal the presence of acute infections or hematologic abnormalities. Reticulocyte counts and hemoglobin electrophoresis may signify the presence of SCD/trait or other hemoglobinopathies. These tests are recommended in all men unless the etiology of priapism is evident.

Radiological Imaging

Penile imaging may assist in the diagnosis of otherwise equivocal priapism cases and may be used in follow up to verify treatment success. Color duplex ultrasonography (CDU) of the perineum and penis can evaluate intracorporeal arterial blood flow in real time. This serves in conjunction with penile blood gas sampling to further differentiate ischemic from non-ischemic priapism. In ischemic priapism, minimal or absent blood flow is seen in the cavernosal arteries within the corpora cavernosa. Patients with non-ischemic priapism, however, will show characteristic normal to high blood flow velocities in the cavernosal arteries.

Treatment of ischemic priapism

Medical management

The most common complication of priapism is erectile dysfunction, which can occur in as many as 59% of cases. However, recovery of erectile function may be seen in up to 44% of patients who experience priapism for 24–36 h, therefore, “time is erectile tissue,” and timely treatment is crucial. First-line therapy for patients with episodes of acute ischemic priapism is aspiration of blood with irrigation of the corpora cavernosa, in combination with intra-cavernous α-agonist injection therapy (Fig 1,2,3). For most Urgent Care practitioners, this should only be done after discussion with the local urological service and only if there are significant delays in getting the patient definitive treatment by a urologist. The technique of penile blood aspiration involves using a transglanular intracorporal angiocatheter insertion or a proximal penile shaft needle access. For proximal penile shaft access, a 16- or 18-gauge angiocatheter is placed percutaneously into the lateral aspect of the penile shaft entering the corpus cavernosum. With a syringe attached, aspiration and evacuation of blood from the corpora cavernosa is performed with irrigation of normal saline or in combination with intra-cavernous injection of an α-adrenergic sympathomimetic agent.

Other treatment modalities include e hormonal therapies, which include gonadotropin-releasing agonists, androgen receptor antagonists, and 5α-reductase inhibitors, as well as other agents including digoxin, gabapentin, baclofen, terbutaline, and even phosphodiesterase 5 (PDE5) inhibitors.

Figure 1. Representative anatomy of the penile shaft and its innervation and vasculature

Figure 2. Location for installation of local anesthetic. Lidocaine is injected into points 1,2 and 3.

Figure 3. Placement of bilateral angiocatheter for drainage

Key Learning Points:

- Priapism can be a tricky diagnosis – especially if patients present with the uncommon non-ischemic and stuttering varieties.
- Management of ischemic priapism is time sensitive.
- First-line therapy of acute ischemic priapism is aspiration of blood – these should be referred to Urology to be done as soon as possible.
- Other treatment modalities are available, but these should in done only after having discussed the case with the local urologist.

References:

1. Montague D., Jarow J., Broderick G., et al. American Urological Association: guideline on the management of priapism. J Urol 2003(170): 1318–1324.

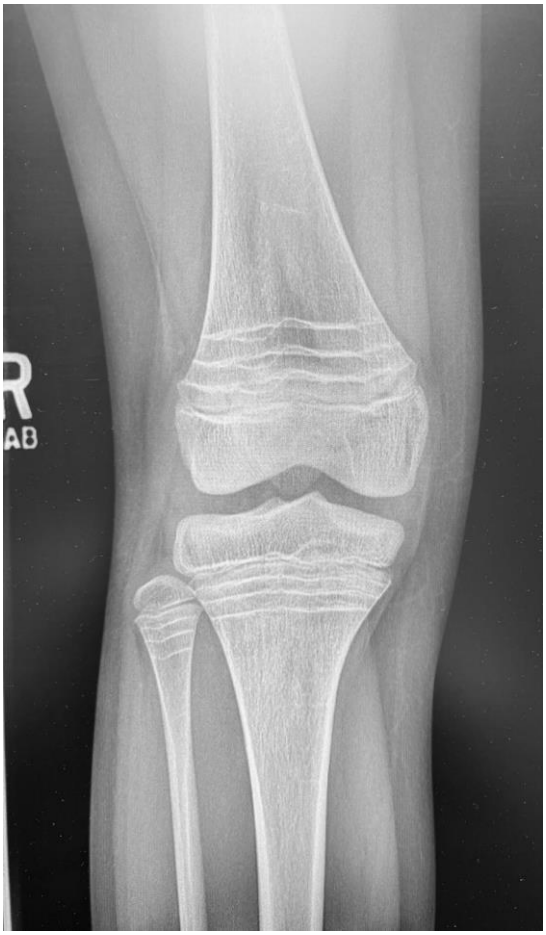
2. Levey H, Segal R, Bivalacqua T Management of priapism: an update for clinicians Ther Adv Urol. 2014 Dec; 6(6): 230–244

3. ALieM Trick of the Trade: Angiocatheter for manual aspiration of priapism https://www.aliem.com/trick-of-trade-angiocatheter-manual-aspiration priapism/

Imaging Challenges—What Are Those Lines?

Tracey Quail Davidoff, MD, FCUCM

An 11-year-old female presents to Urgent Care with complaints of right knee pain. Mom states she has been complaining of a vague pain after exercising and would like an X-ray to make sure there is no pathologic fracture. There was no injury. The radiograph was performed per protocol by the technologist prior to evaluation by the provider. There was no significant past medical history disclosed prior to the X-ray.



These lines are called "Zebra lines" and are seen in patients with osteogenesis imperfecta (OI) who have received bisphosphonate therapy during childhood[12]. Upon further questioning, Mom forgot to mention the child had OI and had received pamidronate in the past.

These lines represent metaphyseal bands, which vary in space according to the age of the patient, the rate of growth, and the location of the metaphysis. Each line corresponds temporally to the administration of a bisphosphonate. The lines are caused by the increased bone mineralization by the bisphosphonate therapy.

OI is a phenotypically diverse group of inherited connective tissue disorders that share similar skeletal abnormalities causing bone fragility and deformity. There is wide variation in the severity of the disease. The initial diagnosis is based on clinical and radiographic findings. Fractures from mild trauma, bowing deformities of long bones, and growth deficiency are the hallmark features. Dependent on age and severity, skeletal features can include macrocephaly, flat midface and triangular facies, dentinogenesis imperfecta, chest wall deformities such as pectus excavatum or carinatum, barrel chest, and scoliosis or kyphosis.[3]

Bisphosphonates are antiresorptive drugs which are widely used to treat children with OI. Treatment aims to increase bone volume by counteracting the high turnover of bone.

Image courtesy of John Manresa, RPA-C

References

[11] Sukumar SP, Balachandran K, Sahoo JP, Kamalanathan S. Zebra lines in osteogenesis imperfecta on bisphosphonate therapy. BMJ Case Rep. 2013 Feb 25;2013:bcr2012008536. doi: 10.1136/bcr-2012-008536. PMID: 23440989; PMCID: PMC3603775.

[2] Al Muderis, M. MD1; Azzopardi, T. FRCS(Ed)1; Cundy, P. FRACS1. Zebra Lines of Pamidronate Therapy in Children. The Journal of Bone & Joint Surgery 89(7):p 1511-1516, July 2007

[3] DOI: 10.2106/JBJS.F.00726A, Marini J. Osteogenesis imperfecta. The Lancet. 387:10028, 16-22 April 2016, 1657-7.

Berberine: A Multisystem Review

Adeeti Gupta MD, FACOG

Abstract:

Berberine is a plant alkaloid with a long history of medicinal use in both Ayurvedic and Chinese medicine. Berberine HCL has been used and has shown purported benefits in glycemic control, metabolic syndrome, polycystic ovarian syndrome, hyperlipidemia and is being used as an adjunctive therapy in some cancers.

Introduction:

Berberine is present in Hydrastis canadensis (goldenseal), Coptis chinensis (Coptis or goldenthread), Berberis aquifolium (Oregon grape), Berberis vulgaris (barberry), and Berberis aristata (tree turmeric). The berberine alkaloid can be found in the roots, rhizomes, and stem bark of these plants.

Discovery and historical journey:

The earliest record of Rhizoma Coptidis as a medicinal herb was in A.D. 200 in *The Herbal Classic of the Divine Plowman* (*Shen Nong Ben Cao Jing*). In about A.D. 500, the anti-diabetes activity of Rhizoma Coptidis was recorded for the first time in a book titled "*Note of Elite Physicians."*

Most berberine used in medical practice is not extracted from this herb because of its high cost. Usually, it is prepared from other herbs such as *Berberis amurense* Rupr. and *Phellodendron amurense* Rupr. Among many chemical forms of berberine, i.e., berberine hydrochloride, berberine sulfate, berberine citrate or phosphate, berberine hydrochloride is the most common form.

A literature search of Berberine, its uses, mechanism of action, new developments, and delivery systems revealed over 10,000 results. I will narrow down the most relevant and easily digestible fragments to inform you of its uses, side effects and the latest research.

Some proven benefits of berberine:

Recently, basic research has proven that berberine can be used to lower the blood glucose level (Liang et al., 2019), improve insulin resistance (Lou et al., 2011), improve hyperlipidemia (Li et al., 2016), and prevent mild cognitive impairment (Kumar et al., 2016). This feature improves the shortcomings of the combination of statins and metformin and shows potential as a new first-line treatment drug.

Here is a brief list of the purported and proven benefits of berberine. In the following section, I will elaborate on the mechanisms that lead to these benefits.

1. Lower blood sugar – improved glycemic control in diabetics.
2. Improve metabolic syndrome parameters (weight, waist circumference, blood pressure and lipid profile).
3. Improve Polycystic Ovarian Syndrome (PCOS).

4. Assist in fighting bacterial and other microbial gut and skin infections due to an antimicrobial property.
5. Improve lipid profile, especially LDL, HDL and Triglycerides.
6. Improvement in NAFLD (nonalcoholic fatty liver disease).
7. Reduce cognitive impairment in diabetics.

Mechanism of action:

1. **Glycemic control:**

Several animal and human studies have shown berberine's unequivocal effects on glucose control. The various mechanistic pathways are:

a. Berberine activates AMP-activated protein kinase (AMPK): AMPK is a key energy-sensing/signaling system in the cells and acts as a fuel gauge by monitoring cellular energy levels.
b. It has an insulin-independent hypoglycemic effect that is related to inhibition of mitochondrial function, stimulation of glycolysis and activation of AMPK pathway. In the newly-diagnosed type 2 diabetic patients, berberine is able to lower blood insulin level *via* enhancing insulin sensitivity. However, berberine may also improve insulin secretion in patients with poor β-cell function by resuscitating exhausted islets.

This study confirmed that administration of berberine (0.5 g three times daily) at the beginning of each meal was able to reduce fasting blood glucose (FBG) and postprandial blood glucose (PBG) in patients with newly-diagnosed type 2 diabetes. Hemoglobin A1c (HbA1c) levels dropped by 2.0%, comparable to the effect of metformin. In poorly-controlled diabetic patients with insulin injection, berberine reduced HbA1c by 0.8%.

In the first *in vitro* study using hepatocytes (HepG2 cell line), berberine was shown to stimulate glucose consumption in an insulin-independent manner, and the activity was similar to that of metformin. Several studies have confirmed the insulin-independent activity of berberine in other cell models, e.g. muscle cells (L6 and C2C12 cell lines) and adipocytes (3T3-L1 cell line). In the presence of insulin, berberine exhibited a synergetic effect on insulin-induced glucose consumption and glucose uptake.

c. The antioxidant and aldose reductase inhibitory activities of berberine may be useful in alleviating diabetic nephropathy.
d. Berberine was shown to protect against endothelial injury, enhance the endothelium-dependent vasodilatation, and downregulate proinflammatory responses through activation of the AMPK signaling cascade.
e. Berberine also acts as an α-glucosidase inhibitor. α-glucosidase is an intestinal enzyme that breaks down carbohydrates into monosaccharides. Inhibition of the enzyme will lead to diminished absorption of dietary carbohydrates.
f. Berberine may have extra beneficial effects on diabetic cardiovascular complications due to its cholesterol-lowering, anti-arrhythmias and nitric oxide (NO) inducing properties.

g. Oxidative stress and aldose reductase activities are closely related to diabetic complications. Several groups have explored the obvious beneficial effect of berberine in this field. In STZ and high-carbohydrate/high-fat diet induced diabetic rats with hyperlipidemia, berberine markedly decreased malondialdehyde level and increased catalase, superoxide dismutase, glutathione peroxidase, and glutathione activities. Berberine also improved cognitive performance, lowered hyperglycemia, oxidative stress, and choline esterase activity in diabetic rats.

2. Cholesterol-lowering effects

Berberine was reported to improve lipid metabolism in both animals and human subjects. Two clinical trials showed that berberine decreased triglycerides by 35% and 22%, serum cholesterol by 29% and 16%, and LDL-C by 25% and 20% in patients with dyslipidemia.

Reduction of cholesterol with berberine is related to the induction of LDL receptor (LDLR) expression in liver, which may be due to extended half-life of LDLR mRNA *via* activation of extracellular signal-regulated kinases (ERK) by berberine.

3. Antimicrobial and antioxidant activities of berberine

The antimicrobial activity of berberine is well-established in the treatment of infection caused by bacteria, viruses, fungi, protozoans and helminthes.

This study showed a significant effect of berberine against Staph Aureus. In this study, berberine showed antimicrobial activity against all tested strains of MRSA. Minimum inhibition concentrations (MICs) of berberine against MRSA ranged from 32 to 128 μg/mL. Ninety percent inhibition of MRSA was obtained with 64 μg/mL or less of berberine.

The authors concluded that BBR reduced the antioxidant capacity of *S. aureus* and the mechanism suggests the inhibition of cell wall synthesis, especially the peptidoglycan synthesis.

4. Improvement in NAFLD

Since liver plays a central role in glucose metabolism, numerous studies focused on effects of berberine, especially in fatty liver disease. In newly diagnosed type 2 diabetics with nonalcoholic fatty liver disease as comorbidity, berberine ameliorated liver steatosis in ultrasonic images, decreased AST and ALT, reduced hemorheology indicators, and improved lipid profile. Similar results were obtained in another study. Berberine lowered FBG effectively in chronic hepatitis B and hepatitis C patients with T2DM or impaired fasting glucose. Liver function was improved greatly in these patients as indicated by the reduction of liver enzymes. This data showed that hepatic steatosis was alleviated by berberine through inhibition of fatty acid synthase (FAS) expression. Berberine decreased fasting blood glucose by direct inhibition of gluconeogenic genes, phosphoenolpyruvate carboxykinase (PEPCK) and glucose-6-phosphatase (G6Pase) in liver.

The antioxidant activity of berberine may directly result from complex I inhibition which is a major place of superoxide production in the electron transport chain.

Mitochondrial inhibition may play a key role in the activities of berberine such as preventing fatty liver, reducing blood glucose and decreasing blood lipids. The details of the regulation remain to be explored.

5. Improvement in metabolic syndrome

The metabolic disorder includes a spectrum of conditions such as nonalcoholic fatty liver disease (NAFLD), type 2 diabetes, impaired glucose tolerance (prediabetes), polycystic ovarian syndrome (PCOS), and hyperlipidemia. Previous studies have demonstrated that metabolic disorders are prone to diabetic encephalopathy and atherosclerosis (Barenbrock et al., 1995), which will generate Alzheimer's disease and coronary heart disease (Razay et al., 2007). NAFLD is closely related to type 2 diabetes and dyslipidemia (Marchesini and Babini, 2006). Characteristic changes in patients with metabolic disorders include a decrease in serum high-density lipoprotein (HDL) or an increase in serum total cholesterol (TC), triglyceride (TG), low-density lipoprotein (LDL), fasting plasma glucose (FPG), and homeostasis model assessment-insulin resistance (HOMA-IR).

In this study, the authors showed that berberine significantly reduced waist circumference and waist/hip ratio significantly in the absence of weight change. Similar results were also reported by other groups. It was indicated that berberine may inhibit visceral fat accumulation. In diabetic rats, adipocyte size and the ratio of white adipose tissue to body weight were decreased, and adipocyte number was increased with berberine treatment.

Although berberine was shown to suppress fat accumulation, the current evidence on mechanisms is controversial.

Berberine may also reduce the risk of developing metabolic syndrome through its beneficial effects on the gut microbiota. In the last decade, many studies have indicated that the composition of gut microbiota is associated with the regulation of the host's health and metabolism. Dysbiosis, defined as an alteration in the quality and/or quantity of the intestinal microbiota, can affect the host's physiology and may be a factor that leads to the onset of various diseases, including obesity and T2DM, as well as cardiovascular diseases, Crohn's disease, and cancer.

6. Cholesterol lowering properties

The results in this study showed that berberine (BBR) supplementation can significantly lower TC, TG, LDL, Fasting blood glucose (FBG), insulin, HbA1c, HOMA-IR, SBP, weight, BMI, and waist circumference (WC) and can elevate HDL. The significant effects of Berberine on HDL and WC were only seen in doses of more than 1 g/day, on FBG and HOMA-IR in the durations of more than 8 weeks, and on HbA1c and weight in both mentioned higher subgroups of dose (>1 g/d) and duration (>8 weeks). Moreover, BBR was significantly effective in alleviating cardiovascular risk factors, mainly in subgroups with impaired metabolic health such as NAFLD, type 2 diabetes, and metabolic syndrome. In addition, BBR was effective for the improvement of LDL, HDL, and FBG only in subgroups with abnormal ranges (HDL ≤ 40, LDL > 100 mg/dl, and FBG > 100 mg/dl). The optimum dose for BBR was 1 g/day for TG, TC, and weight, 1.8 g/day for insulin and HOMA-IR, and 5 g/day for HDL. The most effective duration was 40 weeks for FBG and 50 weeks from the beginning of BBR supplementation for DBP and WC.

BBR is suggested to upregulate the expression of LDL receptors in the human hepatoma cell line (HepG2) and to inhibit both cholesterol and TG synthesis in the liver, dose-dependently. AMPK agonist activity of berberine leads to the inhibition of cholesterol and TG synthesis by inactivating two enzymes, β-Hydroxy β-methylglutaryl-CoA (HMG-CoA) and ACC (acetyl-coenzyme A carboxylase). AMPK activation also increases energy production hence normalizing the imbalance between glucose, lipid, and energy. This

activation can also impose anti-inflammatory effects and can speed up the transport of glucose in the serum by promoting glucose transporter type 4 **(**GLUT4) translocation, although GLUT4 involvement is still unclear.

7. Berberine and cancer treatment

Berberine has exhibited ability to suppress tumor metastasis (Lin et al., 2006; Serafim et al., 2008; Cai et al., 2014). Matrix metalloproteinases (MMPs) degrade the tissue matrix, allowing tumor cells to break through the normal tissue barrier and invade the surrounding normal tissue and distant organs. Berberine inhibits the release of MMP-2 from tumor cells and thus inhibits tumor cell destruction of the tissue matrix.

In vitro studies have demonstrated that the inhibition of FAK, IKK, NF-kB, u-PA, MMP-2, and MMP-9 significantly reduced metastasis.

How is berberine absorbed in the human body?

Berberine exhibits poor absorption, efflux, and extensive metabolism in the human gut. The absolute bioavailability of berberine is far less than 1%. Accordingly, one of the approaches for improving berberine's efficacy is through studying a variety of formulations to improve its bioavailability from the gut.

Which formulations of berberine are the best?

Berberine HCL is the most commonly available preparation. There is explosive research in progress to figure out the best way to increase the bioavailability of berberine.

One groundbreaking area is that of nanoparticles. Nanoparticles are fat-loving particles that help in protecting a drug from the breakdown of gastric enzymes and transport the drug to the bloodstream. Various nanoparticle formulations are being used in cancer treatments.

Nanoparticle formulations that encapsulate berberine for sustained release and improved bioavailability include the use of polymeric natural (e.g., chitosan) and synthetic (PLGA, PLGA-PEG, etc.) agents. Others include a self-micro emulsifying berberine-phospholipid complex of polyethylene glycol 1000 succinate (TPGS 1000) and SiO_2, phytosomes loaded with berberine-phospholipid complex, solid lipid nanoparticles, micelles, liposomes of various nature, etc.

Berberine NPs produced by both APSP and EPN methods have shown promising activities against gram positive and gram negative bacteria, and yeasts, with NPs prepared through the EPN method showing superior results compared to those made with the APSP method and the unprocessed drug.

Side effects and adverse effects of berberine:

Berberine is clinically safe and well-tolerated by the human body. Few adverse reactions are reported, and no negative effect is observed on participants' diet.

In this study, none of the patients suffered from severe gastrointestinal adverse events when berberine was used alone. In combination-therapy (metformin + berberine) the adverse events disappeared in one week after reduction in berberine dosage. The data suggest that berberine at dosage of 30mmg three times daily is well tolerated in combination therapy. Liver and kidney functions were monitored in this

study. No significant changes in plasma ALT, γ-GT and creatinine were observed during the 13 weeks of berberine treatment.

It is recommended that berberine be taken with food and if possible, as a gastroprotective coated formulation.

References:

1. Ye Y, Liu X, Wu N, Han Y, Wang J, Yu Y, Chen Q. Efficacy and Safety of Berberine Alone for Several Metabolic Disorders: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. Front Pharmacol. 2021 Apr 26;12:653887. doi: 10.3389/fphar.2021.653887. PMID: 33981233; PMCID: PMC8107691.
2. Front. Pharmacol., 15 January 2020
   Sec. Ethnopharmacology
   Volume 10 - 2019 | https://doi.org/10.3389/fphar.2019.01461
3. Comincini S, Manai F, Sorrenti M, Perteghella S, D'Amato C, Miele D, Catenacci L, Bonferoni MC. Development of Berberine-Loaded Nanoparticles for Astrocytoma Cells Administration and Photodynamic Therapy Stimulation. *Pharmaceutics*. 2023; 15(4):1078.
4. Wu S, Yang K, Hong Y, Gong Y, Ni J, Yang N, Ding W. A New Perspective on the Antimicrobial Mechanism of Berberine Hydrochloride Against *Staphylococcus aureus* Revealed by Untargeted Metabolomic Studies. Front Microbiol. 2022 Jul 13;13:917414. doi: 10.3389/fmicb.2022.917414. PMID: 35910599; PMCID: PMC9328669.
5. Och A, Och M, Nowak R, Podgórska D, Podgórski R. Berberine, a Herbal Metabolite in the Metabolic Syndrome: The Risk Factors, Course, and Consequences of the Disease. Molecules. 2022 Feb 17;27(4):1351. doi: 10.3390/molecules27041351. PMID: 35209140; PMCID: PMC8874997.
6. Zhao, J., Wang, Z., Karrar, E., Xu, D., & Sun, X. (2022). Inhibition Mechanism of Berberine on α-Amylase and α-Glucosidase in Vitro. *Starch - Stärke*, *74*(3-4), 2100231. https://doi.org/10.1002/star.202100231
7. Xing, L., Zhou, X., Li, A., Li, H., He, C., Qin, W., Zhao, D., Li, P., Zhu, L., & Cao, H. (2021). Atheroprotective Effects and Molecular Mechanism of Berberine. *Frontiers in Molecular Biosciences*, *8*, 762673. https://doi.org/10.3389/fmolb.2021.762673
8. Ma, X., Chen, Z., Wang, L., Wang, G., Wang, Z., Dong, X., Wen, B., & Zhang, Z. (2017). The Pathogenesis of Diabetes Mellitus by Oxidative Stress and Inflammation: Its Inhibition by Berberine. *Frontiers in Pharmacology*, *9*. https://doi.org/10.3389/fphar.2018.00782
9. Koppen, L. M., Whitaker, A., Rosene, A., & Beckett, R. D. (2017). Efficacy of Berberine Alone and in Combination for the Treatment of Hyperlipidemia: A Systematic Review. *Journal of Evidence-based Complementary & Alternative Medicine*, *22*(4), 956-968. https://doi.org/10.1177/2156587216687695
10. Yu HH, Kim KJ, Cha JD, Kim HK, Lee YE, Choi NY, You YO. Antimicrobial activity of berberine alone and in combination with ampicillin or oxacillin against methicillin-resistant Staphylococcus aureus. J Med Food. 2005 Winter;8(4):454-61. doi: 10.1089/jmf.2005.8.454. PMID: 16379555.
11. Koperska, A., Wesołek, A., Moszak, M., & Szulińska, M. (2022). Berberine in Non-Alcoholic Fatty Liver Disease—A Review. *Nutrients*, *14*(17). https://doi.org/10.3390/nu14173459
12. Ye Y, Liu X, Wu N, Han Y, Wang J, Yu Y, Chen Q. Efficacy and Safety of Berberine Alone for Several Metabolic Disorders: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. Front Pharmacol. 2021 Apr 26;12:653887. doi: 10.3389/fphar.2021.653887. PMID: 33981233; PMCID: PMC8107691.
13. Lou T, Zhang Z, Xi Z, Liu K, Li L, Liu B, Huang F. Berberine inhibits inflammatory response and ameliorates insulin resistance in hepatocytes. Inflammation. 2011 Dec;34(6):659-67. doi: 10.1007/s10753-010-9276-2. PMID: 21110076.
14. Petrangolini, G., Corti, F., Ronchi, M., Arnoldi, L., Allegrini, P., & Riva, A. (2020). Development of an Innovative Berberine Food-Grade Formulation with an Ameliorated Absorption: In Vitro Evidence Confirmed by Healthy Human Volunteers Pharmacokinetic Study. Evidence-based Complementary and Alternative Medicine : ECAM, 2021. https://doi.org/10.1155/2021/7563889
15. Javed Iqbal, M., Quispe, C., Javed, Z., Sadia, H., Qadri, Q. R., Raza, S., Salehi, B., Abdulwanis Mohamed, Z., Sani Jaafaru, M., & Abdull Razis, A. F. (2021). Nanotechnology-Based Strategies for Berberine Delivery System in Cancer Treatment: Pulling Strings to Keep Berberine in Power. *Frontiers in Molecular Biosciences*, *7*, 624494. https://doi.org/10.3389/fmolb.2020.624494
16. Yin, J., Xing, H., & Ye, J. (2008). Efficacy of Berberine in Patients with Type 2 Diabetes. *Metabolism: Clinical and experimental*, *57*(5), 712. https://doi.org/10.1016/j.metabol.2008.01.013
17. Zhang, C., Sheng, J., Li, G., Zhao, L., Wang, Y., Yang, W., Yao, X., Sun, L., Zhang, Z., & Cui, R. (2020). Effects of Berberine and Its Derivatives on Cancer: A Systems Pharmacology Review. *Frontiers in Pharmacology*, *10*, 481416. https://doi.org/10.3389/fphar.2019.01461
18. Jun Yin, Jianping Ye, Weiping Jia,
19. Effects and mechanisms of berberine in diabetes treatment, Acta Pharmaceutica Sinica B, Volume 2, Issue 4, 2012.
20. Yin, J., Ye, J., & Jia, W. (2012). Effects and mechanisms of berberine in diabetes treatment. Acta Pharmaceutica Sinica B, 2(4), 327-334. https://doi.org/10.1016/j.apsb.2012.06.003
21. Zamani M, Zarei M, Nikbaf-Shandiz M, Hosseini S, Shiraseb F, Asbaghi O. The effects of berberine supplementation on cardiovascular risk factors in adults: A systematic review and dose-response meta-analysis. Front Nutr. 2022 Oct 14;9:1013055. doi: 10.3389/fnut.2022.1013055. PMID: 36313096; PMCID: PMC9614282.

Simplifying Lead Wire Interchanges

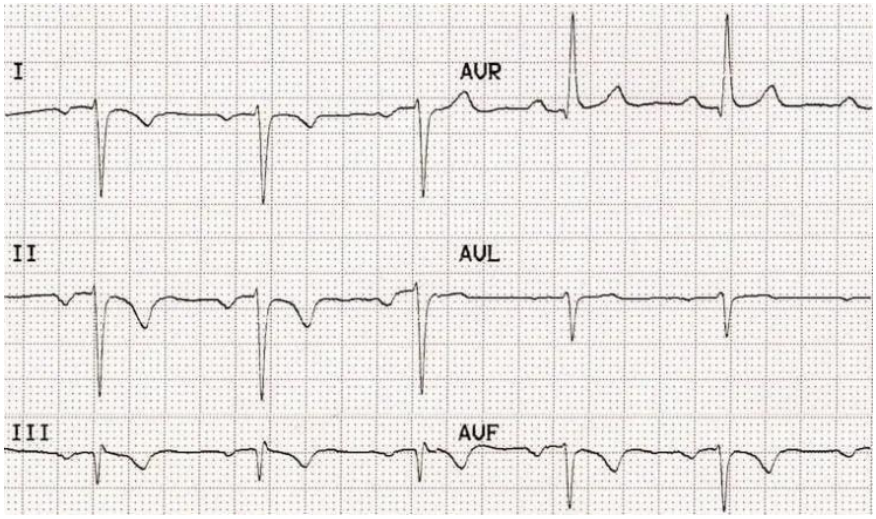
Jerry W. Jones, MD FACEP FAAEM

Section Editor, ECG Corner

Are you pretty good at diagnosing lead wire interchanges? How about this one…

Do you recognize it? A lot of healthcare providers often diagnose a LA/RA lead wire interchange based on the inverted deflections in Lead I but still struggle with the others. I'm going to fix that!

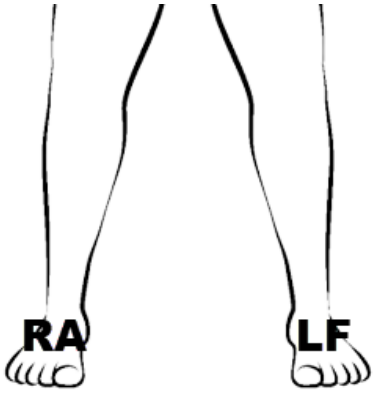
There are six possible lead wire interchanges among all the limb leads, including the neutral wire attached to the right foot. There is no real reason for the neutral wire to be placed on the right foot. One would obtain the same result if it were attached to the patient's forehead.



I will begin with the interchanges involving the neutral wire on the right foot. I want to address those first since they are *very easy to recognize*. Just remember, we aren't going to be concerned with where the neutral wire ends up – we are only going to be concerned with the lead wires that are placed on the right and left feet.

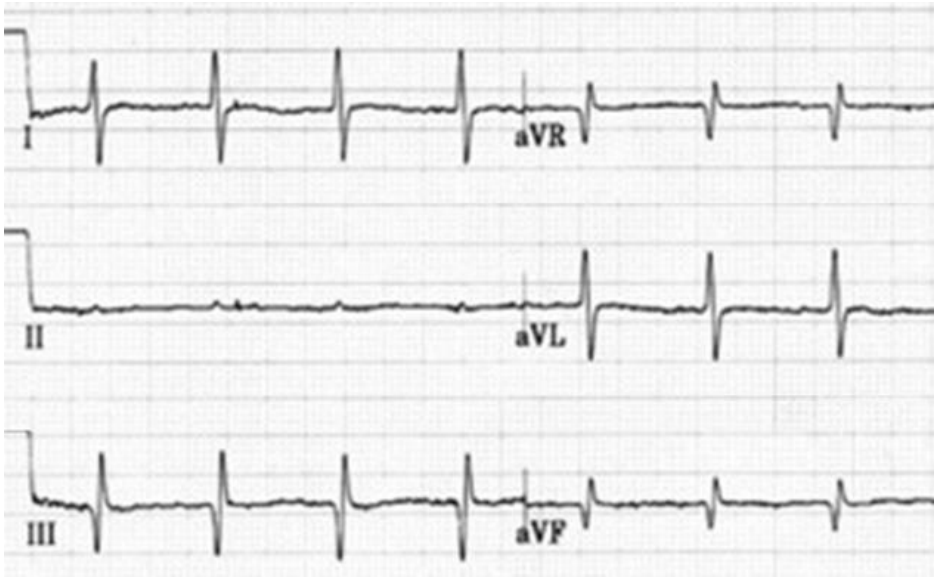
First Interchange: RF (neutral) and RA (aVR) Lead Wire Interchange

The RA wire (Lead aVR) is placed on the right foot electrode and the LF electrode with the wire for Lead aVF is on the left foot, its normal location. Remember: the *right leg* and *left leg* should be considered as the *same extremity*. If you place two electrodes adjacent to each other on the same extremity there would be essentially no difference in electrical charge between them. And that's what happens when you have two *recording* electrodes on the right and left feet – they record a near ZERO DIFFERENCE in electrical charge.



Lead II is the difference between the RA electrode (aVR) and the LF electrode (aVF) – but since the RA wire is now attached to the right foot, Lead II will have **zero voltage**! That means there will be a near-isoelectric line in Lead II.

As you can see, when Lead II finds itself measuring from the right foot (RF) to the left foot (LF), there is **ZERO VOLTAGE**. And this results in a nearly flat line in Lead II.

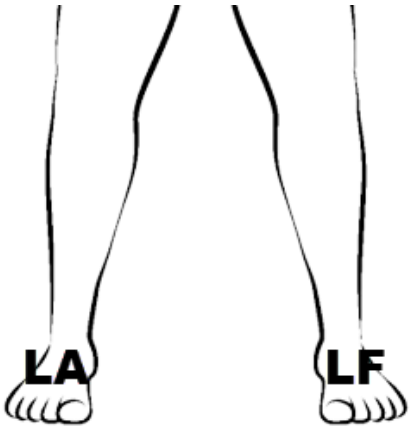


About the only other thing that would do this is a disconnected lead wire.

This is very easy to recognize and now you know the diagnosis, so let's move on.

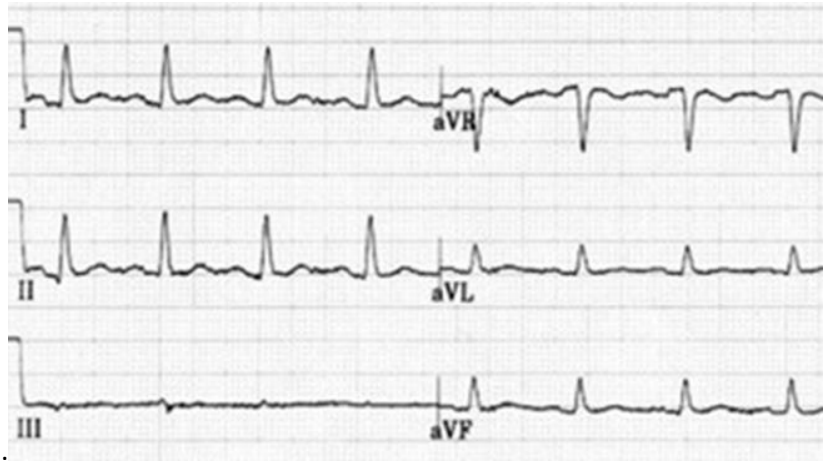
Second Interchange: RF (neutral) and LA (aVL) Lead Wire Interchange

Now we have the left arm (LA) wire attached to the right foot electrode. Keep in mind that the electrodes themselves are not the problem. It's the lead wires being attached to the wrong electrode that is causing this issue.



Now when we record the difference between the LF and LA we again end up with **ZERO VOLTAGE**. And which lead is the difference in voltage between the LF and LA? It's Lead III. And now, Lead III has minimal recorded voltage difference. What will that look like on the ECG?

And there you have it! A near isoelectric line in Lead III.



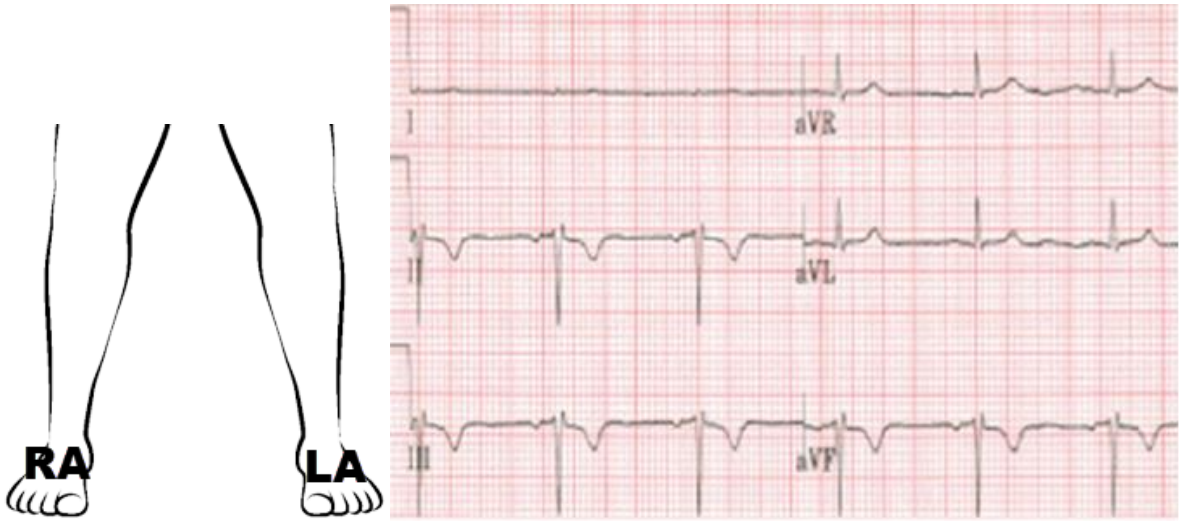
OK… we've seen how Lead II becomes a near-isoelectric line and also how Lead III becomes the same.

We know that Lead II is the difference between the RA and LF electrodes and that Lead III is the difference between the LA and LF electrodes. But how do we end up with a near-isoelectric line in Lead I? Note that both Leads II and III use the LF electrode wire in their calculation; but Lead I does not. Lead I is the difference between the LA electrode and the RA electrode. So how do we get the RA and LA wires on the right foot and left foot (respectively)?

Third Interchange: RA and RF (neutral) and LA and LF <u>Double</u> Lead Wire Interchange

This requires a **DOUBLE** interchange: the RA changes with the RF *and* the LA changes with the LF. Now we have the RA on the right foot and the LA on the left foot. They record a **ZERO VOLTAGE** in Lead I and Lead I develops a near-isoelectric baseline. This is very rare, but it does happen.

But be careful… *advanced emphysema* can also produce a Lead I that looks a lot like this due to the shifting of the mean QRS axis into the horizontal plane (but it's usually accompanied by very tall P waves in the inferior leads).



Now let's look at the interchanges in which we use the JONES method…

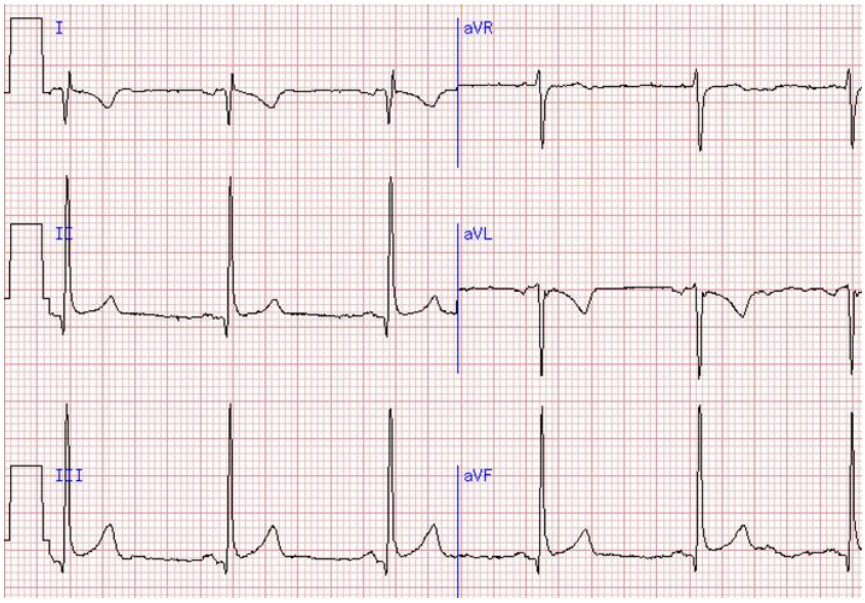
There will be leads switching places with other leads but again, nothing for you to memorize or be concerned about. Just remember one fact: standard leads (I, II, III) can only switch places with other standard leads. Augmented leads (aVR, aVL, aVF) can only switch places with other augmented leads. Lead aVL cannot switch places with Lead II, for instance. This fact benefits YOU because when a lead changes places with another lead, there will be only TWO places for you to look – and you will know those places immediately. Now back to the JONES method.

We've reviewed the three lead wire interchanges involving the RF (neutral) wire. Now there are just three lead wire interchanges remaining.

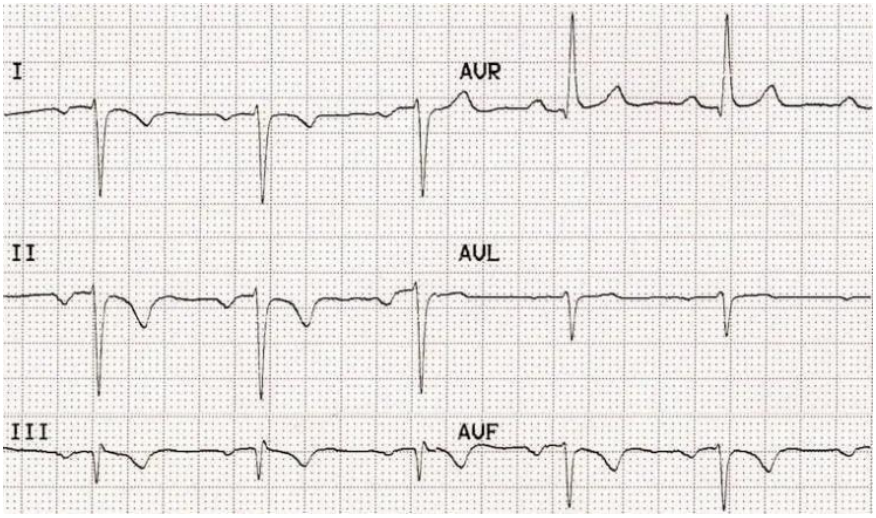
The Jones method begins the moment you pick up the ECG and begin deciding which rhythm is present. There are two leads you will scrutinize very carefully: **Leads I and II**. If the P wave in Lead I is upright, that means atrial depolarization is traveling from right to left – and that's normal. You will also look closely at Lead II for two reasons. An upright P wave means that the P vector is traveling toward the positive pole of Lead II located at +60°. That is very suggestive of an impulse coming from the vicinity of the SA node. But there is one more characteristic in Lead II that you want to see: ***during sinus rhythm Lead II will almost always have the largest P wave of all the limb leads***. And that is where the JONES method begins. If, while determining the rhythm, you notice that Lead II does *not* have the largest P wave but lead I has the largest P wave, then you have diagnosed a *lead wire interchange*. A quick look at Lead aVR to ascertain that Lead aVR is in its correct location will then tell you that this is a **LA/LF lead wire interchange** – probably the most difficult lead wire interchange to diagnose. There are several papers that can provide you with the trigonometric calculations that explain why this is so… but you will do just as well by skipping them (if you still want to look further, I have listed them in the references for this article).

The remaining two lead wire interchanges are diagnosed simply by looking at Lead aVR. Why Lead aVR? Because both of the remaining lead wire interchanges involve Lead aVR. Consequently, Lead aVR will always be "out of place" with the two remaining lead wire interchanges.

If you find Lead aVR (RA) in the space for Lead aVL (LA), then Lead aVR has switched places with Lead aVL and there is a LA/RA lead wire interchange (**aVL = LA** and **aVR = RA**). This is a LA/RA lead wire interchange on the left.



If Lead aVR (RA) is located in the space for Lead aVF (LF), then Lead aVR has switched with Lead aVF and there is a RA/LF lead wire interchange (**aVR = RA** and **aVF = LF**). Remember that ECG snippet at the beginning of this article? Look at it again (left)…



You may have thought this was a LA/RA lead wire switch based on the negative deflections in Lead I… but you would have been wrong. Had you used the JONES method, you would not have made that mistake. Instead, you would have found Lead aVR in the space for Lead aVF indicating a RA/LF lead wire interchange. With the JONES method, there is no need to scrutinize the other leads for inverted deflections.

In fact, ALL the standard leads (I, II and III) look like Lead aVR – but they cannot be Lead aVR. Why? **Because augmented leads (aVR, aVL, aVF) cannot switch with any of the standard leads (I, II, III). Standard leads can only switch with other standard leads and augmented leads can only switch with other augmented leads**.

So, in summary…

1. You learned how to recognize the three lead wire interchanges involving the neutral wire on the right foot. It has nothing to do with the JONES method *which only involves the recording electrodes*, but these interchanges are *very obvious* and *very simple to recognize*.

2. You used the JONES method with the remaining three lead wire interchanges using only Leads II and aVR. One interchange involved the LA and LF and is diagnosed by recognizing that the largest P wave is in Lead I – not in Lead II where it *should* be located. The remaining two lead wire interchanges involved Lead aVR, so all you had to do was locate Lead aVR knowing that there were only two possible places it could be (aVL or aVF).

If Lead aVR was found in the space for aVL, then aVR and aVL switched places which is the same as the RA and LA switching places. On the other hand, if Lead aVR was found in the space for aVF, then aVR and aVF switched places which is the same as RA and LF foot switching places.

There's nothing to memorize.

References

1. Heden B, et al. Detection of frequently overlooked electrocardiographic lead reversals using artificial neural networks. Am J Cardiol 1996;78: 600.
2. Hoffman I. A flatline electrocardiogram in lead II is a marker for right arm/right leg electrode switch. J Electrocardiol 2007;40:226.
3. Edenbrandt L, Rittner R. Recognition of lead reversals in pediatric electrocardiograms. Am J Cardiol 1998;82:1290.
4. Ho KKL, Ho SK. Use of the sinus P wave in diagnosing electrocardiographic limb lead misplacement not involving the right leg (ground) lead. J Electrocardiol 2001;34:161.
5. Rudiger A, et al. Electrocardiographic artifacts due to electrode misplacement and their frequency in different clinical settings. Am J Emerg Med 2007;25:174.
6. Rowlands DJ. Inadvertent interchange of electrocardiogram limb lead connections. Analysis of predicted consequences. J Electrocardiol 2008;41:84.
7. Rowlands DJ. Inadvertent interchange of electrocardiogram limb lead connections. Analysis of predicted consequences. Part II: double interconnection errors. J Electrocardiol 2008;41:84.
8. Batchvarov, V. et al. Incorrect electrode cable connection during electrocardiographic recording. Europace (2007) 9, 1081 – 1090.

New: Oral & Maxillofacial Section--Management of Oral Wounds

H. Brian Sun, DMD, MS

Dental injuries are one of the most common drivers of emergency and Urgent Care visits. Approximately 4.1% of all emergency visits in Australia are attributable to dental traumatic injuries[1] and U.S. pediatric providers treat at least one oral trauma every 2 to 3 days.[2] A vast majority of the services rendered were limited to simple palliative measures even when their treatment did not require specialized dental training.[3]

Lacerations and soft tissue injuries are the most common sequelae of maxillofacial trauma.[4] Fortunately, punctures and lacerations measuring less than 1 cm typically do not require formal closure especially when they occur on proliferative surfaces like oral epithelia. These wounds should be gently debrided of gross debris and irrigated thoroughly. Patients should also be instructed to practice good oral hygiene and to remain on a soft food diet without small, firm components such as seeds or grains for approximately two weeks.

Suture closure is the preferred method of addressing lacerations 1 cm or longer in length. As always, the injured site should be debrided and irrigated prior to closure using size 3-0 or 4-0 resorbable suture. Any visible underlying muscle tears should also be reapproximated. Mucosal closures can be completed with relative ease because they do not scar easily and tolerate poor or even mis-approximations well. A course of antibiotics that cover common oral flora - such as one week of amoxicillin (500mg q8h) or clindamycin (300mg q6h), or a 5-day course of azithromycin (500mg 1st day, 250mg q24h subsequently) - may be appropriate for patients with complex lacerations, increased risks of pathogenic contamination (especially from animal bites), and immunocompromised states.[5]

Chromic gut sutures (CGS) provide adequate strength, handling, and half-life when compared to non-coated traditional or plain gut sutures. Braided polyglycolic-acid (PGA) sutures (i.e. Vicryl®, Polysorb®) are similarly easy to handle but their weeks-long life spans can lead to mucosal irritation and food entrapment. Long lasting but unbraided Poliglecaprone-25 sutures (i.e. Monocryl®, Biosyn®) minimize tissue inflammation but their glossy texture and coil memory risk fraying the already-injured tissues. Many oral surgeons utilize CGS for mucosal closures and PGA sutures for sub-surface muscles where the risk of food entrapment is minimal and tensile demands are prolonged.

Table 1: Common Resorbable Sutures in Oral Surgery

	Time to Lose 50% Strength	Resorption Time	Suture Texture
Plain Gut	24 hours	3 to 5 days	Rough, brittle
Chromic Gut	5 days	7 to 10 days	Rough, coated
Polyglycolic Acid	21 days	21 to 28 days	Flexible, braided
Poliglecaprone 25	14 days	90 to 120 days	Flexible, glossy

Abrasions, burns, sores, ulcers, large punctures, and even partially or completely closed lacerations may benefit from the placement of oral dressings. Resorbable, porous matrix dressings like the collagen tape and plug (ZimVie® Dental, Westminster, COPalm Beach Gardens, Florida) are particularly versatile in a wide variety of traumatic injuries. The collagen tape is a rectangular, thin, absorbent collagen barrier that may be cut to size and placed over the injured mucosa where it adheres to and protects the internal tissues. While the tape's collagen surface aids in hemostasis, its greatest benefit may be the relief it

provides against pain and the insults of the oral environment. There are anecdotal indications that the collagen matrix may also serve as a porous scaffold for the migrating new epithelial cells – somewhat like the collagen of the underlying connective tissues – though additional studies are needed.[6]

Figure 1: Credit ZimVie® (Formerly, Zimmer Biomet Dental)

Deep and bleeding osseous openings from missing teeth (from dental extractions or avulsions) are difficult to treat via closure only. The collagen plug is a tooth root-shaped collagen matrix that can be inserted into a hemorrhagic dental socket. This aids in obtaining hemostasis, preventing impaction of oral debris, and preserving the contour of the nearby soft tissues. The plug expands as it absorbs fluid to prevent dislodgement and to aid in further compression hemostasis. It can also be cut to better match the anatomic variations of the various tooth roots. A figure-of-eight suture may be placed over the socket as an additional measure of protection.

Figure 2: CollaplugRegenerOss® Bone Graft Plug

Credit: ZimVie® (Formerly, Zimmer Biomet Dental)

A dentally-mindful Urgent Care provider may use a similar collagen plug from the same manufacturer that is also impregnated with bone-graft materials (RegenerOss® Bone Graft Plug). The bone and gingiva surrounding missing teeth atrophy rapidly within the first six to twelve months, leading to an esthetic defect of "shrunken gums" that also complicate the any future dental implant surgeries of the area.[7] Bone-graft impregnated plugs contain not only collagen to for hemostasis and protection but also calcified osteoconductive materials which help delay undesirable periodontal bone atrophy.

Regardless of the material and technique used to manage oral wounds, the provider must perform a detailed examination to rule out any other significant underlying injuries such as concussion, fracture, or infection. If neurological or osseous complications are suspected, specialist referrals should be considered to oral-maxillofacial surgery, otolaryngology or plastic surgery. Sutures and dressings should not be placed in obviously infected areas. Most importantly, the contents of any literature should not replace the judgments of the evaluating clinician.

H. Brian Sun is a Clinical Instructor at the University of the Pacific, a Clinical Assistant Professor at the Western University of Health Sciences, and a private practice oral-maxillofacial surgeon at Peninsula Oral Surgery San Jose. He can be reached via his website at http://www.maxfacedoc.net.

References

1. Dang K, Day P, Calache H, Tham R, Parashos P. Reporting dental trauma and its inclusion in an injury surveillance system in Victoria, Australia. *Australian Dental Journal*. 2015;60(1):88-95. doi:10.1111/adj.12273

2. Nelson LP, Shusterman S. Emergency management of oral trauma in children. *Current opinion in pediatrics*. 1997;9(3):242-245. doi:10.1097/00008480-199706000-00010

3. Matsunaga M, Chen JJ, Donnelly P, Fok CCT, Partika NS. Emergency Room Visits with a Non-Traumatic Dental-Related Diagnosis in Hawaii, 2016–2020. *International Journal of Environmental Research and Public Health*. 2022;19(5):0-7. doi:10.3390/ijerph19053073

4. Khan TU, Rahat S, Khan ZA, Shahid L, Banouri SS, Muhammad N. Etiology and pattern of maxillofacial trauma. *PLoS ONE*. 2022;17(9 September):1-10. doi:10.1371/journal.pone.0275515

5. Katsetos SL, Nagurka R, Caffrey J, Keller SE, Murano T. Antibiotic prophylaxis for oral lacerations: our emergency department's experience. *International Journal of Emergency Medicine*. 2016;9(1). doi:10.1186/s12245-016-0122-7

6. Seok H, Jo YY, Kweon HY, Kim SG, Kim MK, Chae WS. Comparison of Bio-degradation for Ridge Preservation Using Silk Fibroin-based Grafts and a Collagen Plug. *Tissue Engineering and Regenerative Medicine*. 2017;14(3):221-231. doi:10.1007/s13770-017-0055-0

7. Hämmerle CHF, Araújo MG, Simion M, 2011 OB of the OCG. Evidence-based knowledge on the biology and treatment of extraction sockets. *Clinical Oral Implants Research*. 2012;23(s5):80-82. doi:https://doi.org/10.1111/j.1600-0501.2011.02370.x

Coding Corner: URI Recheck

Brad Laymon PA-C, CPC, CEMC
Section Editor, Coding Corner

SUBJECTIVE

44 y/o established patient here for a recheck of a URI. He was seen at our clinic 3 days ago. He has been ill for a total of 6 days. I reviewed the note on 12/29/22: COVID and Influenza POCT were both negative. CXR was negative for infiltrate. He was prescribed Tessalon Perles 200 mg TID PRN for cough. He states he is not feeling better. His cough has improved with Tessalon but he still has a low grade fever up to 100.1. Appetite is decreased and taste and smell "are gone". His wife tested positive for COVID 4 days ago. He is taking OTC ibuprofen for fever and achiness.

Past Medical History

Hypercholesterolemia

Current Medications

He takes Lipitor 10 mg daily and aspirin 81 mg daily.

Drug Allergies

NKDA

OBJECTIVE

BP 138/88

HR 108R

RR 16

Temp 99.2

SPO2 96%

HT 6' 1"

WT 205lbs

A&OX3, NAD, speaks in complete sentences.

HEENT: Normocephalic, PERRLA, EOMI, TMs- clear, no erythema, Mouth: no erythema or exudates

Neck: No adenopathy or JVD

Lungs: CTAB, no rhonchi, rales, or wheezing

Heart: Tachy rate, no murmur

Skin: warm and dry, no edema or cyanosis

Recent results
POC COVID is positive
POC Influenza test is negative

ASSESSMENT

COVID-19 infection

PLAN
Orders Placed in the Encounter

POC COVID
POC Influenza

Facility-Administered Encounter Medications

None

Medications Prescribed During the Encounter

None

He will continue Tessalon 200 mg TID PRN for the cough. He will also continue ibuprofen for body aches and fever. Increase fluids and rest. He will quarantine for 4 more days. I sent a message to his PCP for further evaluation to include a video visit in 2-3 days to ensure he is improving. He will go to the ED for worsening of symptoms or if new symptoms arise (CP, SOB, high fever). Patient understands and agrees.

We will break this case down by referring to the 3 Elements of Medical Decision Making:

Number and Complexity of Problems Addressed

Patient presents for follow up of a URI with stable vital signs. He does test positive for COVID, but this would be a low, level 3 acute uncomplicated illness or injury.

Amount and/or Complexity of Data to be Reviewed and Analyzed

The provider ordered a POC Influenza and COVID test during this office visit. Reviewing the office visit of 12/29/22 does NOT count towards the data section since it was not an **external** note. The CXR does not count towards the data column for the same reason. The complexity of data would be low, level 3 (2 POCT).

Risk of Complications and/or Morbidity or Mortality of Patient Management

Although no new prescription was prescribed, the provider informed the patient to continue Tessalon for the cough so this would count towards prescription medication/management. Risk would be a moderate, level 4.

Two of the three elements of MDM must be met to choose a level of service. Problems addressed and data both met the low, level 3 criteria and risk meets the moderate, level 4 criteria so this is a 99213.

A Best Practice from the College of Urgent Care Medicine

Diagnosing and Treating Uncomplicated Urinary Tract Infection and Pyelonephritis in Urgent Care

Date Reviewed	09/01/2023
Subject	Diagnosing and treating acute uncomplicated acute cystitis and pyelonephritis
Patient Population	Females 18 years of age and older
Rationale	Patients with urinary tract infection (UTI) commonly present to Urgent Care (UC) and there is the potential for over-diagnosis and inappropriate treatment. Understanding the elements of accurate diagnosis and appropriate treatment will lead to the best patient outcomes.
Introduction	UTIs may be categorized as uncomplicated or complicated. Uncomplicated UTIs involve nonpregnant females without fever or other systemic symptoms (chills, significant fatigue or malaise), recent urological procedures, indwelling devices, underlying urologic abnormalities, or immunosuppression, and are much more common than complicated UTIs, which comprise all other patients and scenarios. This document discusses the diagnosis and treatment of acute uncomplicated cystitis and pyelonephritis only. The differential diagnosis of uncomplicated acute cystitis includes pyelonephritis, sexually transmitted infections, vaginitis, ureteral and bladder calculi, and noninfectious cystitis and urethritis. History, physical examination, pelvic examination and associated testing, urinalysis, and urine culture may be needed to distinguish between these. The most recent evidence-based treatment guidelines for uncomplicated cystitis were published in 2011 by the Infectious Diseases Society of America (IDSA)[1].
Evidence based guideline with strength of evidence	The IDSA treatment guidelines published in 2011 are the most recent evidence-based guidelines and are being updated. At the time of publication, the strength of antibiotic treatment recommendations was Grade A and B, according to the *IDSA Handbook on Clinical Practice Guideline Development*, referred to in the guideline itself, which indicates good (Level A) or moderate (Level B) strength. The quality of evidence was related predominately to randomized controlled trials (Quality level I) but also respected authority opinion (Quality level III) in some cases. The time since publication may create some uncertainty in terms of current relevance, but while awaiting an authoritative update, there are no alternatives available. The goals of the guideline were to provide a reasonable empiric approach to the selection of antibiotics to maximize the chance of cure while reducing the harms associated with inappropriate antibiotic prescription.

Discussion	**History and Physical Examination**[2] The accurate diagnosis of UTI has been shown to correlate with the absence of symptoms of vaginal irritation or discharge and the presence of specific urinary symptoms (dysuria, urinary frequency, urinary urgency, suprapubic pain and hematuria). These are sufficiently predictive that tele-diagnosis based on history alone is reasonable in certain situations. For example, the presence of dysuria and frequency in a patient without vaginal discharge yields a very high likelihood ratio (LR) of 24.6 for decision-making. In comparison, when vaginal discharge or irritation is present in a patient with either dysuria or frequency (but not both), the LR is 0.7. The presence of flank pain and fever also increases the probability of UTI, but supports a more complicated infection possibly being present. Low abdominal discomfort can indicate UTI but also broadens the differential diagnosis, and so the absence of significant abdominal tenderness should be sought. "Self-diagnosis" of UTI by a patient based on current symptoms being similar to prior UTI is also an accurate indicator for diagnosis, though an UTI very closely following another should increase suspicion of resistance to prior antimicrobial therapy or a more complicated infection or alternative diagnosis being present. **Urinalysis and Urine Culture**[2,3] Urinalysis may be used to increase or decrease the probability of a UTI after historical and exam features establish a pretest probability. The presence of leukocyte esterase or nitrites on urine dipstick or white blood cells on microscopic exam increases the likelihood of UTI and a normal urinalysis decreases the likelihood. The diagnostic value added by urinalysis is highest when results are concordant with the history and much lower when they are not. For example, when the pretest likelihood of UTI is high based on history and exam, a normal urinalysis **cannot** rule out UTI. And even such a "positive" urinalysis is less likely to support the diagnosis of UTI when urinary symptoms are **not** present. Moreover, when evaluating patients with nonspecific symptoms such as fever or altered mental status but without urinary tract symptoms, diagnoses other than UTI should be considered, regardless of urinalysis results. Though often considered the gold standard for UTI diagnosis, the sensitivity and specificity of urine culture for diagnosing UTI is not perfect and many factors (specimen collection and transport methods, lab handling) can affect results. Patients with UTI can have negative or inconclusive (single organism below a defined threshold, e.g., 100k colony-forming unit/cc or the presence of multiple organisms) results and those without UTI may have positive cultures (asymptomatic bacteriuria). Concordance with history, exam and urinalysis adds the most diagnostic value. Urine culture and sensitivity results can guide treatment and are strongly recommended for patients with complicated UTI, patients with risk factors for complication and patients with high risk for antibiotic resistance (history of multidrug resistance urine culture and recent within the past three months

	hospitalization and use of fluoroquinolones, trimethoprim/sulfamethoxazole and broad-spectrum beta-lactam). They do inform knowledge of local antimicrobial resistance but are not typically needed for uncomplicated UTI. The evaluation of patients with uncomplicated but recurrent or repeated UTI symptoms may benefit from urine culture. **Antibiotic treatment for UTI**[1] In any case where culture and sensitivity results are available, choice of therapy can be based on those results and the antibiotic preference order explained below. For uncomplicated UTI, unless contraindicated, empiric treatment (in the absence of urine culture and sensitivity results) should be limited to one of the following: **First-line** ● Nitrofurantoin monohydrate/macrocrystals 100 mg PO BID x 5 days ● Trimethoprim-sulfamethoxazole 160/800 mg (one double strength tablet) PO BID x 3 days (*avoid if typical uropathogen resistance prevalence is known to be > 20%, or if used for UTI within the previous 3 months*) ● Fosfomycin trometamol 3 g PO single dose ● Pivmecillinam 400 gm PO BID x 5 days (not available in the US) **Second-line** ● Beta-lactam agents such as amoxicillin-clavulanate, cefdinir, cefaclor, and cefpodoxime in 3-7 day courses can be considered if none of the above can be used. Cephalexin may be considered as well, but may be less effective, according to the guidelines. ● Fluroquinolones (ofloxacin, ciprofloxacin, levofloxacin) may be effective in 3 day regimens but due to higher side effects are considered alternatives if none of the above can be used. ● Amoxicillin or ampicillin should **not** be used due to higher resistance levels. For pyelonephritis treatable as an outpatient (stable, nonpregnant, nontoxic patient, who is able to take PO medications), urine culture and sensitivity should be performed prior to treatment. Empiric treatment options while awaiting culture and sensitivity results include: *If typical uropathogen resistance prevalence is known to be < 10% to the agent listed* ● Ciprofloxacin 500 mg PO BID x 7 days with or without ciprofloxacin 400 mg IV at time of initial visit ● Levofloxacin 750 mg PO daily or Ciprofloxacin ER 1000 mg daily x 7 days *If typical uropathogen resistance prevalence could be > 10% to the agent listed*

	● Ciprofloxacin 500 mg PO BID, Levofloxacin 750 mg PO daily or Ciprofloxacin ER 1000 mg daily **WITH** ceftriaxone 1 g (IV or IM) or a consolidated 24-hour dose of an aminoglycoside at time of initial visit *Other choices* ● Trimethoprim-sulfamethoxazole 160/800 mg (one double strength tablet) PO BID for 14 days **WITH** ceftriaxone 1 g (IV or IM) or a consolidated 24-hour dose of an aminoglycoside at time of initial visit ● If none of the above can be used, Beta-lactam agents such as amoxicillin-clavulanate, cefdinir, cefaclor, and cefpodoxime x 10-14 days can be considered **WITH** ceftriaxone 1 g (IV or IM) or a consolidated 24-hour dose of an aminoglycoside at time of initial visit ● Though effective for acute cystitis, renal penetration of nitrofurantoin, fosfomycin and pivmecillinam is poor, and these are **not** options for the treatment of pyelonephritis.
Summary	● The diagnosis of UTI starts with a patient's symptoms. Complaints of urinary frequency, discomfort, and symptoms similar to prior UTI are highly sensitive and specific. The presence of vaginal symptoms (e.g., discharge or irritation) favors a vaginal etiology rather than UTI. ● Urinalysis is not necessary when the history strongly supports the diagnosis. It is more helpful when there is indeterminate probability after history and exam. A positive urinalysis may not indicate UTI requiring treatment when there are no specific urinary symptoms or exam findings. ● Use of antibiotics in accordance with the IDSA guidelines[1] as above provides a reasonable empiric approach which maximizes the chance of cure while reducing the harms associated with inappropriate antibiotic prescription. ● The guidelines emphasize specific narrow spectrum antibiotics and shorter time courses, **so avoid prescribing broader-spectrum antibiotics or longer time courses than recommended.** ● Clinicians should be aware that the antibiotic choices for cystitis differ from pyelonephritis due to renal penetration of medication. ● Knowledge of local antibiotic resistance is important in modifying any planned empiric regimen. ● The main value of urine culture is in determining whether empiric antibiotic treatment needs to be changed for patients with complicated or recurrent UTI, and culture should be obtained in these situations. Urine culture is **not** needed for cases of nonrecurrent, uncomplicated cystitis. ● Diagnostically, a negative culture does not rule out a UTI and a positive culture does not diagnose a UTI. Urine culture adds the most value to UTI diagnosis when it matches the pretest probability based on history, exam and urinalysis.
References	1. Gupta et al. International Clinical Practice Guidelines for the Treatment of Acute Uncomplicated Cystitis and Pyelonephritis in Women: A 2010 Update by the Infectious Diseases Society of America and the European Society for Microbiology

	and Infectious Diseases. *Clin Infect Dis*. 2011;52(5):e103-20. DOI: 10.1093/cid/ciq257 2. Bent S, et al. Does this woman have an acute uncomplicated urinary tract infection? *JAMA*. 2002;287:2701-10. DOI: 10.1001/jama.287.20.2701 3. Werneburg GT, et al. Diagnostic stewardship for urinary tract infection: A snapshot of the expert guidance. *Cleve Clin J Med.* 2022;89(10):581-7. DOI: 10.3949/ccjm.89a.22008
Reviewers	Joseph Toscano, MD and Cesar Mora Jaramillo, MD FAAFP FCUCM

Exploring Non-Opioid Pain Treatments in the Urgent Care

Maureen McCaffrey, PA-C, Medical Editor, Hippo Education

Treating acute pain is a challenge we face during every Urgent Care shift. We're all aware of the devastation that the opioid crisis has had on millions of people worldwide. And this epidemic - marked by addiction, overdose, and unintended consequences - has highlighted the need for alternative treatments to manage pain.

As clinicians, we regularly encounter patients wanting or expecting opioids to treat their pain. For years, opioids were the go-to option. We've since learned that even short-term opioid prescriptions can lead to tolerance, dependence, and addiction, making them a potentially risky choice for pain management.[1] However, not all providers are comfortable or possess the knowledge to discuss safer and more effective alternatives.

By the time patients decide to come to Urgent Care, they've often tried over-the-counter medications such as acetaminophen and ibuprofen to manage their pain. We know these medications are highly effective when dosed appropriately. However, patients rarely now how to dose these medications correctly, which is possibly why many of them report them to be ineffective.

Statistics have shown that in head-to-head trials with combined acetaminophen and ibuprofen versus that combination in addition to opioids, the same pain reduction score was achieved.[2] This is valuable information that we can share with our patients to help them understand that we're all trying to accomplish the same goal and not trying to deny them treatment for their pain.

Combining these medications with adjunct options for breakthrough pain allows us to effectively treat pain without the risks and harmful side effects of prescribing opioids alone. Having an open discussion with our patients regarding reasonable expectations of pain relief, the importance of dosing schedules, and varied treatment options increases the odds of improved pain and decreases the chance of bounce backs.

What else can we prescribe for pain?

Many patients can't take acetaminophen or ibuprofen due to comorbidities, contraindications, or allergies, so what other options exist? Sometimes it's as easy as getting back to basics. Physical modalities such as heat or cold therapy can decrease pain by promoting circulation and altering nerve signals. Compression and elevation can significantly reduce swelling, which decreases pain. Knowing which modality to use in which phase of injury can be essential to help guide an effective treatment plan.

Do topicals work?

Yes, topical pain medications can be an effective addition to a treatment plan. They're low risk and have few side effects associated with them.[3] These products deliver medication to a localized area of pain and may be used alone or as an adjunct to prescribed oral medications. The many delivery methods include gels, sprays, creams, ointments, and patches. The main classes are as follows:

- **Counterirritants:** These products contain ingredients like menthol, camphor, or capsaicin, which create a cooling or warming sensation. This overwhelms the nerves on the skin and distracts the brain from the underlying pain.
- **NSAIDS**: This medication works locally to decrease inflammation at the site, which can be helpful in the management of sprains and strains as well as chronic conditions like arthritis.
- **Lidocaine patches:** Lidocaine anesthetizes the area where the patch is applied. These patches can effectively manage localized pain, particularly nerve pain such as post-herpetic neuralgia.

What about nerve blocks?

Low-risk and highly effective nerve blocks can treat dental pain and headaches. This option gives clinicians a fantastic tool to quickly and easily alleviate a patient's acute symptoms. By combining short and long-acting anesthetics, you can effectively provide hours of pain relief or complete resolution of their pain. As Urgent Care clinicians, these skills are easy to learn, safe, and can be performed in the clinic with minimal tools.[4,5]

Is there a place for opioids?

As we strive to meet best practice standards, we must realize that opioids may still have a role in treating pain. The pendulum has swung so far away from prescribing these medications that it is essential to remember our responsibility to treat our patients appropriately.

Many patients can't take acetaminophen or ibuprofen, or perhaps their injury is severe enough that there may be some expected breakthrough pain even with adjunct medication. By having alternative medications or modalities available and educating our patients, we can decrease the amount of opioids prescribed and simultaneously improve patient care. Our patients are best served when we provide them with the safest and most efficacious treatments. As the landscape of pain management continues to evolve, we owe it to our patients to stay current on viable options to treating acute and chronic pain.

1. Butler MM, Ancona RM, Beauchamp GA, et al. Emergency Department Prescription Opioids as an Initial Exposure Preceding Addiction. *Ann Emerg Med*. 2016;68(2):202-208. doi:10.1016/j.annemergmed.2015.11.033 PMID: 26875061
2. Chang AK, Bijur PE, Esses D, Barnaby DP, Baer J. Effect of a Single Dose of Oral Opioid and Nonopioid Analgesics on Acute Extremity Pain in the Emergency Department: A Randomized Clinical Trial. JAMA. 2017;318(17):1661–1667. doi:10.1001/jama.2017.16190 PMID: 29114833
3. Leppert W, Malec-Milewska M, Zajaczkowska R, Wordliczek J. Transdermal and Topical Drug Administration in the Treatment of Pain. *Molecules*. 2018;23(3):681. Published 2018 Mar 17. doi:10.3390/molecules23030681. PMID: 29562618
4. Miller S, Lagrata S, Matharu M. Multiple cranial nerve blocks for the transitional treatment of chronic headaches. Cephalalgia. 2019;39(12):1488-1499. doi:10.1177/0333102419848121. PMID: 31084198
5. Shauly O, Gould DJ, Sahai-Srivastava S, Patel KM. Greater Occipital Nerve Block for the Treatment of Chronic Migraine Headaches: A Systematic Review and Meta-Analysis. *Plast Reconstr Surg*. 2019;144(4):943-952. doi:10.1097/PRS.0000000000006059. PMID: 31568309

Tick-Borne Illness: A Diagnostic Approach for the Urgent Care Clinician

KidBits: Tick-borne Illnesses in Children

Due to the nonspecific nature of tick-related illness, combined with decreased body awareness, tick bites and their associated symptoms can often be less conspicuous in children, and easier to overlook or attribute to an alternative diagnosis. This is important to remember, especially with rickettsial disease, because while children represent 6% of all reported cases of RMSF, they account for 22% of fatalities.[98] Children's style of play (outdoors, with pets, etc.) may place them at increased risk for tick exposure. Because of this, familiarity with the pathophysiology of tick-borne disease is essential to making an accurate diagnosis and treatment plan. Preferred pediatric treatment regimens for Lyme disease include doxycycline, amoxicillin, or cefuroxime. Doxycycline is especially indicated if neurological symptoms are present. The preferred agent for treatment of RMSF, ehrlichiosis, and anaplasmosis in pediatric patients is also doxycycline. As with pregnancy, the adverse effects of doxycycline in children (in particular, teeth staining) have been largely disproved in recent studies.[99,100]

5 Things That Will Change Your Practice

1. Many patients with tick-borne illness never report finding a tick.
2. Do not rely on serological tests to make a clinical decision or to initiate antibiotic treatment.
3. Doxycycline is the first-line antibiotic treatment for Lyme disease, RMSF, ehrlichiosis, and anaplasmosis in patients of all ages. Recent research shows no evidence of tooth staining when doxycycline is used in short courses in pediatric patients.
4. Prophylactic treatment is not needed for tick bites, except for prevention of Lyme disease transmission when the recommended criteria are met.
5. The presence of an erythema migrans rash in a patient at risk for Lyme disease is diagnostic and treatment should be initiated without testing.

Risk Management Pitfalls for Tick-Borne Illness in Urgent Care

1. **"My practice is not located in a Lyme disease-endemic area, so there's no way this patient has Lyme disease."** Although 95% of Lyme disease cases are reported in 14 states in the Northeast and upper Midwest regions of the US, the geographic footprint of Lyme disease is expanding. In states where Lyme disease is not endemic, positive cases are usually attributed to patient travel (e.g. a backpacker in the Adirondack Mountains who returns home to San Diego after vacation). Clinicians should always obtain a complete travel history.
2. **"My patient told me she didn't have any tick bites, so I didn't consider tick-borne illness."** A significant number of patients do not recall a tick bite. Patients also may have the misperception that tick bites only occur in the woods and therefore overlook potential exposures in backyards, developed areas, etc.
3. **"I didn't start doxycycline in my 8-year-old patient because I was concerned about the effects on his teeth and bones."** Doxycycline is the first-line treatment for all suspected rickettsial infections in the

pediatric population. Recent studies have shown no adverse effects when the antibiotic is used in short courses. Children are at higher risk for serious disease and death from tick-borne illnesses and prompt treatment is critical to minimize this risk.

4. **"My patient was diagnosed with babesiosis, but I didn't realize he also had Lyme disease."** Coinfections should always be considered, as tick species often can carry and transmit multiple diseases. *Ixodes* ticks are vectors for anaplasmosis, Lyme disease, babesiosis, Powassan disease, and *B miyamotoi* disease. Rash is infrequently seen with babesiosis, so if rash is also present, coinfection should strongly be considered.
5. **"I clinically diagnosed my patient with influenza. RMSF wasn't in my differential diagnosis."** The initial symptoms of spotted fever rickettsiosis are nonspecific and cannot be easily differentiated from viral illnesses such as influenza and COVID-19. Clinicians must maintain a high index of suspicion, especially if a patient presents with "flu-like" symptoms during the spring and summer months.
6. **"The tick serology test was negative, so I ruled out tick-borne illness."** Serology testing measures antibodies produced by the body in response to an illness, a process that takes a few days. If testing was performed too early in the disease course, antibodies would not be detectable. For this reason, a 4-fold increase in antibody titers is needed two to four weeks apart for laboratory confirmation of disease. In RMSF or ehrlichiosis, the disease can be fatal before laboratory confirmation is finalized.

Time- and Cost-Effective Strategies

- Among patients who present to Urgent Care with concern for tick-borne illness, Lyme disease is a frequent concern. Patients may request diagnostic testing for reassurance of the absence of disease. It is important to keep in mind that in most cases of Lyme disease, test results should not and ultimately do not change management. False-positive results can lead to undue stress and unnecessary treatment. Avoiding over-ordering of tests reduces costs, but also improves quality of management and prevents false alarm or false reassurance.
- Rather than ordering tests for all possible tick-borne diseases, the prevalence of local tick-borne illnesses should always be considered, and testing for specific tick-borne illnesses should be selected based on geographic considerations and patient risk.
- Though patients often bring ticks into the clinic, the tick should not be sent for testing but should only be used for visual identification of the tick species.
- The persistent duration of antibodies makes follow-up testing unnecessary to confirm resolution of illness. Rather, clinicians and patients can be assured if appropriate treatment was initiated in a timely manner.

References

98. Dalton MJ, Clarke MJ, Holman RC, et al. National surveillance for Rocky Mountain spotted fever, 1981-1992: epidemiologic summary and evaluation of risk factors for fatal outcome. *Am J Trop Med Hyg*. 1995;52(5):405-413. **(Review of epidemiological data)**

99. Volovitz B, Shkap R, Amir J, et al. Absence of tooth staining with doxycycline treatment in young children. Clin Pediatr (Phila). 2007;46(2):121-126. **(Retrospective study; 61 patients)**

100. Casey Barton Behravesh, Schutze GE. Doxycycline can be used in young children without staining teeth. *AAP News.* 2015;26(5):16. **(Expert opinion)**

Excerpted from: Chao C, Decker KW. Tick-borne illness: a diagnostic approach for the Urgent Care clinician. *Evidence-Based Urgent Care*. 2023 June;2(6):1-30. Reprinted with permission of EB Medicine. Learn more about *Evidence-Based Urgent Care* and get a free sample issue at https://www.ebmedicine.net/urgent-care-info

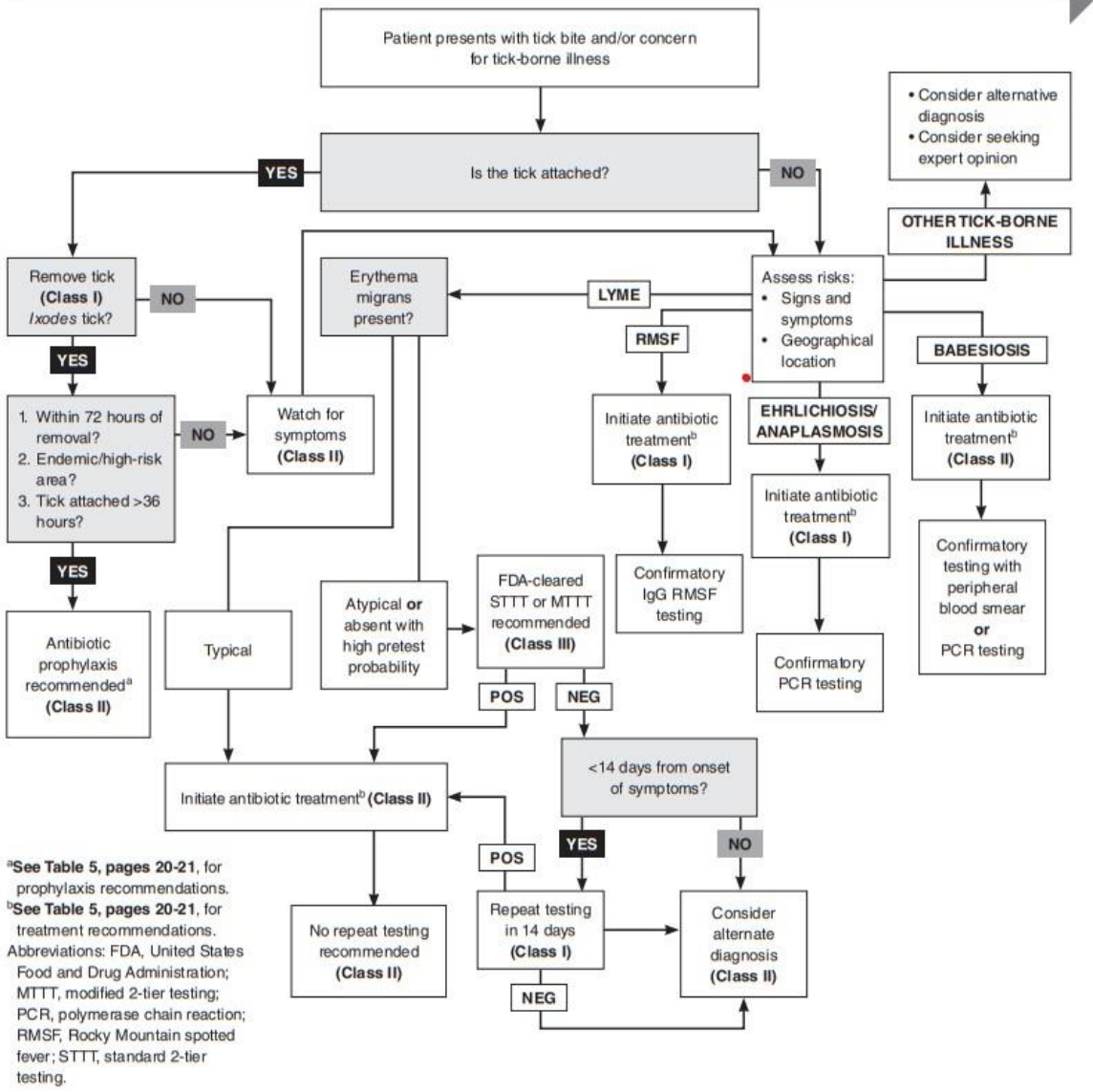
Urgent Care Evaluation and Management of the Red Eye

5 Things That Will Change Your Practice

1. Developing a routine set of questions and pattern of examination, with increased proficiency through consistent use, can assist in establishing a diagnosis in patients presenting with a red eye.
2. Visual acuity should be obtained on any patient who presents with an eye complaint.
3. Antibiotic stewardship should be considered for topical antibiotic use; even bacterial conjunctivitis is often self-limited.
4. Acute angle-closure glaucoma requires emergent ophthalmology evaluation and is a sight-threatening condition. Classic symptoms in addition to a red eye include acute onset of unilateral vision change classically with halos around lights, a larger dilated pupil in the affected eye, and brow/ eye pain.
5. Recent eye procedures warrant consultation and follow-up with the ophthalmologist or surgeon due to the risk of endophthalmitis.

Excerpted from: Shakelford CE. Urgent care evaluation and management of the red eye. *Evidence-Based Urgent Care*. 2023 August;2(8):1-21. Reprinted with permission of EB Medicine. Learn more about *Evidence-Based Urgent Care* and get a free sample issue at https://www.ebmedicine.net/urgent-care-info

Clinical Pathway for Urgent Care Management of Tick-Borne Illness



[a]**See Table 5, pages 20-21**, for prophylaxis recommendations.
[b]**See Table 5, pages 20-21**, for treatment recommendations.
Abbreviations: FDA, United States Food and Drug Administration; MTTT, modified 2-tier testing; PCR, polymerase chain reaction; RMSF, Rocky Mountain spotted fever; STTT, standard 2-tier testing.

Class of Evidence Definitions

Recommendations in the clinical pathways section of *Evidence-Based Urgent Care* receive a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

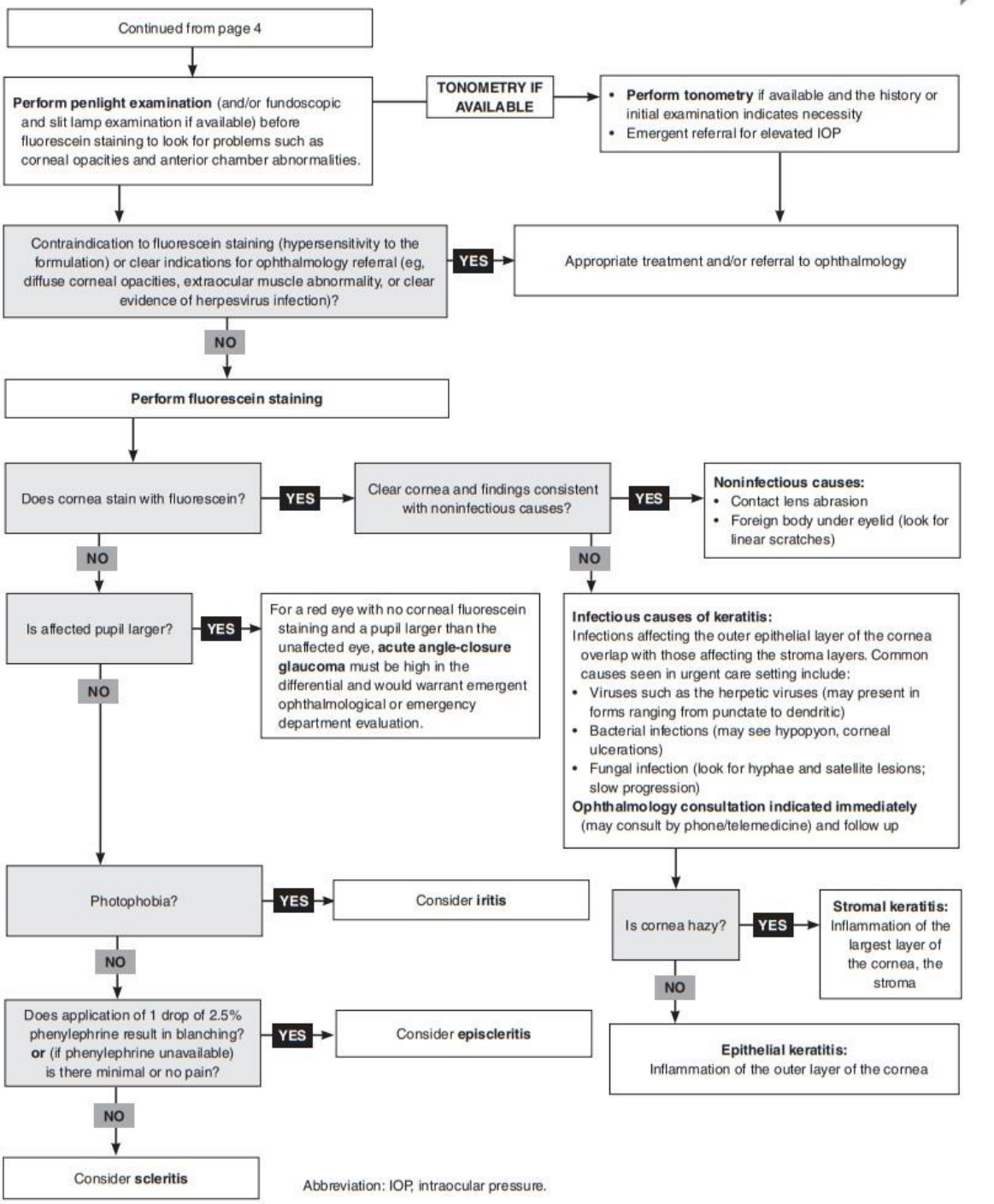
Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

© 2023 EB MEDICINE. ALL RIGHTS RESERVED.

Clinical Pathway for Diagnostic Evaluation of the Red Eye



Abbreviation: IOP, intraocular pressure.

© 2023 EB MEDICINE. ALL RIGHTS RESERVED.

Urgent Care Q&A

Can allergic conjunctivitis cause ulcerations?

Most seasonal allergic conjunctivitis will respond optimally to a combination of antihistamine and mast cell stabilizer drops. Patients with severe allergic conjunctivitis may develop a corneal ulceration in the superior aspect of the cornea due to hypertrophic papillae.

Source: EBM

Does penicillin allergy sensitivity decrease over time?

Many patients who report penicillin allergies do not have true IgE-mediated reactions. When evaluated, fewer than 1% of the population are truly allergic to penicillins. Approximately 80% of patients with IgE-mediated penicillin allergy lose their sensitivity after 10 years. Correctly identifying those who are not truly penicillin-allergic can decrease unnecessary use of broad-spectrum antibiotics.

Source: CDC

What is the best method to obtain urine samples in infants?

If a clinician decides that a febrile infant with no apparent source for the fever requires antimicrobial therapy to be administered because of ill appearance or another pressing reason, the clinician should ensure that a urine specimen is obtained for both culture and urinalysis before an antimicrobial is administered; the specimen needs to be obtained through catheterization or suprapubic aspiration (SPA), because the diagnosis of UTI cannot be established reliably through culture of urine collected in a bag.

A clean-catch urinary sample is acceptable in toilet-trained children or if obtained via spontaneous voiding methods. American Academy of Pediatrics (AAP) guidelines recommend obtaining a catheterized specimen in children <6 months of age. Provocative methods have been described for obtaining a clean catch urine sample in infants. A urinary bag specimen is only helpful if negative. Culture is frequently difficult to interpret with a positive bagged specimen because most positives are false positives. Infants/children not able to spontaneously void (or with a bagged specimen with a positive UA) should have a catheterized (or suprapubic tap) specimen obtained.

Source: AAP

Email your **clinical questions** to the Editors:
Tracey Davidoff, MD, FCUCM tdavidoff@coucm.org
or Cesar Mora Jaramillo, MD, FAAFP, FCUCM cmjaramillo@coucm.org

Disclaimer: This material is for educational purposes only. Medical practice and knowledge are constantly evolving and changing.
This information is peer-reviewed but should not be your only source. Providers of care should use discretion when applying knowledge to any individual patient.

Urgent Updates

West Nile Infections Rising in the US
West Nile Virus is the leading cause of mosquito-borne disease in the continental U.S. And as of August 8, 126 human cases had been identified across 22 states, according to the Centers for Disease Control and Prevention (CDC). **Full Access:** Medscape

Bird Flu Researchers Turn to Finland's Mink Farms, Tracking A Virus with Pandemic Potential
H5N1 virus does not infect people easily. But the fear is that uncontrolled spread in animals like mink gives the virus plenty of chances to evolve in ways that could enable it to spill over into people. Already in Finland, a paper from government researchers indicated the virus has spread from mammal to mammal at the farms — and in some cases has picked up mutations indicating an adaptation toward replicating in mammalian cells. **Full Access:** STAT

FDA Approves First Oral Treatment for Postpartum Depression
The U.S. Food and Drug Administration approved Zurzuvae (zuranolone), the first oral medication indicated to treat postpartum depression (PPD) in adults. PPD is a major depressive episode that typically occurs after childbirth but can also begin during the later stages of pregnancy. The efficacy of Zurzuvae for the treatment of PPD in adults was demonstrated in two randomized, double-blind, placebo-controlled, multicenter studies. **Full Access:** FDA

Identifying Children Likely to Benefit from Antibiotics for Acute Sinusitis - A Randomized Clinical Trial
Randomized clinical trial including 515 children aged 2 to 11 years diagnosed with acute sinusitis based on clinical criteria. The trial was conducted at primary care offices affiliated with 6 U.S. institutions and was designed to evaluate whether symptom burden differed in subgroups defined by nasopharyngeal *Streptococcus pneumoniae, Haemophilus influenzae*, or *Moraxella catarrhalis* on bacterial culture and by the presence of colored nasal discharge. In children with acute sinusitis, antibiotic treatment had minimal benefit for those without nasopharyngeal bacterial pathogens on presentation, and its effects did not depend on the color of nasal discharge. Testing for specific bacteria on presentation may represent a strategy to reduce antibiotic use in this condition. **Full Access:** JAMA

Prescribing Medications for Alcohol Use Disorder: A Qualitative Study of Primary Care Physician Decision Making
Over 29 million Americans have alcohol use disorder (AUD). Though there are effective medications for AUD (MAUD), they are underutilized. Physicians endorsed that it is challenging to prescribe MAUD due to several reasons, including: (1) somewhat negative personal beliefs about medication effectiveness and likelihood of patient adherence; (2) competing demands in primary care that make MAUD a lower priority; and, (3) few positive subjective norms around prescribing. There is a challenging implementation context for MAUD. **Full Access:** Annals of Family Medicine

The Efficacy and Safety of Metoclopramide in Relieving Acute Migraine Attacks Compared With Other Anti-Migraine Drugs: A Systematic Review and Network Meta-Analysis Of Randomized Controlled Trials
A significant decrease in headache scores was seen with the administration of intravenous (IV) metoclopramide, compared with placebo and sumatriptan, according to the findings of a study published in the journal *BMC Neurology*. Sixteen studies were included with a total of 1934 patients: 826 received metoclopramide, 302 received placebo, and 806 received other active drugs. Metoclopramide was

effective in reducing headache outcomes even for 24 h. Regarding side effects, metoclopramide showed a lower incidence of mild side effects than pethidine and chlorpromazine and showed a higher incidence of mild side effects than placebo, dexamethasone, and ketorolac. **Full Access:** BMC Neurology

The Burden of Antimicrobial Resistance in The Americas in 2019: A Cross-Country Systematic Analysis
Antimicrobial resistance (AMR) is an urgent global health challenge and a critical threat to modern health care. Researchers estimated deaths and disability-adjusted life-years (DALYs) attributable to and associated with AMR for 23 bacterial pathogens and 88 pathogen–drug combinations for countries in the WHO Region of the Americas in 2019. They estimated 569,000 deaths (95% UI 406,000–771,000) associated with bacterial AMR and 141,000 deaths (99,900–196,000) attributable to bacterial AMR among the 35 countries. Lower respiratory and thorax infections, as a syndrome, were responsible for the largest fatal burden of AMR in the region. **Full Access:** The Lancet

Management of Acetaminophen Poisoning in the U.S. and Canada - A Consensus Statement
This qualitative study used an expert-derived consensus according to a modified Delphi process to provide explicit clinical guidance on the assessment, management, and treatment of acetaminophen poisoning. The panel developed guidelines for emergency department management of single or repeated ingestion of acetaminophen. In addition, the panel addressed extended-release formulation, high-risk ingestion, co-ingestion of anticholinergics or opioids, age younger than 6 years, pregnancy, weight greater than 100 kg, and intravenous acetaminophen use. **Full Access:** JAMA

Thromboprophylaxis for Lower Limb Immobilization
Venous thromboembolism (VTE) occurs in approximately 1-2% of patients who have lower limb immobilization after injury. The National Institute for Health and Care Excellence (NICE) recommends VTE risk assessment to determine prescribing but is not prescriptive on method and advocates only parenteral prophylaxis. This was a cross-sectional survey sent to EDs in the UK to evaluate present practice by clinicians and was conducted between February and March 2022.
The authors received responses from m 116 EDs (England 89, Scotland 15, Wales 6 and Northern Ireland 6) accounting for 69.5% of type 1 (EM specialist led with 24-hour resuscitation capabilities) UK departments. ≥95% respondents reported considering thromboprophylaxis in ambulatory patients managed in a lower limb rigid cast of any sort, while half would do so for a walking boot and 20% when using removable knee splints.

Comments – Although this was an ED based survey, some of the considerations regarding use of VTE thromboprophylaxis would be useful for UC practitioners, when treating patients who require lower limb immobilization. Use of locally agreed guidelines would help in the matter and where possible potential UC/secondary care interphase could help with patient journeys. **Full Access:** BMJ

The OPAL Trial – Are Opioids Really Necessary to Treat Lower Back and Neck Pain?
Lower back and neck pain are common presentations to UC. Most of the present clinical guidelines suggest the prescription of opioids for these conditions only as a last resort measure and limiting the use of opioids due to the risks of adverse events including dependency. This study set out to investigate the efficacy and safety of a judicious short course of an opioid analgesic for the management of acute non-specific low back and neck pain. Participants were randomly assigned (1:1) to the opioid or placebo group. The opioid group were treated with 5 mg oxycodone and 2.5 mg naloxone as a modified release tablet, twice a day and titrated up to a maximum of 10 mg, twice a day, based on individual participant progress, tolerability, and sedation score. The placebo group received identical-looking tablets made of colloidal silicon dioxide, microcrystalline cellulose, sodium starch glycolate, and sodium stearyl fumarate, coated in brilliant blue.

347 participants were randomly assigned to a treatment or control groups, (174 opioid, 173 placebo). No significant difference was found in pain scores at 6 weeks between the opioid group and the placebo group. Pain severity was not significantly different between groups at week 12. No significant difference was found in physical functioning measured by the generic scale or condition-specific scale for patients with neck pain. No significant difference was found between groups for quality of life on the physical function subscale, but a small yet significant difference favoring placebo for the mental health subscale was found at 6 weeks and 12 weeks.

Comments – The findings of this study further highlight the lack of evidence for the prescribing of opioids in acute back pain and correlates with other previous studies on the matter. As UC practitioners, we need to be able to convey this message to our patients as part of managing expectations during our consultations. **Full Access:** The Lancet

New: Urgent Updates in Pediatrics

Ivan Koay MD, MBChB, MRCS, FRNZCUC
Urgent Care Physician and Medical Lead Kings College Hospital Urgent Treatment Centre, London
Abstracts Section Editor, Journal of Urgent Care Medicine
Convenor Ireland and UK Faculty of the Royal New Zealand College of Urgent Care
Independent Assessor European Reference Network, Andalusian Agency for Healthcare Quality

Anxiety and Depression Treatment in Pediatric Population

There has been a rise in anxiety and depression in the pediatric population in the U.S. and globally. Primary and Urgent Care are frequently the first point of contact for patients with these conditions. Engagement in care is crucial as exclusive management by subspecialist isn't feasible due to the high prevalence of these conditions, a shortage of trained subspecialty clinicians, long wait lists, and insurance challenges. This was a medical record review study to determine whether PCPs followed clinical practice guidelines when providing care to children and adolescents who had been prescribed an SSRI medication for anxiety and/or depression.

The authors randomly selected 110 cases for review and found that in 82% of cases, PCPs documented reasons for starting an SSRI, most commonly clinical change (57%). PCPs documented psychiatry or developmental behavioral pediatrics subspecialist involvement in 30% of cases and referred 34% of patients for nonspecific psychotherapy. PCPs documented the use of a screening tool (e.g., SCARED, GAD-7, PHQ-9) in 26% of patients. **Full Access:** AAP

Is Immunotherapy the Answer to Peanut Allergy?

Peanut allergy affects approximately 2% of the U.S., Canadian and worldwide population and is a common cause for pediatric anaphylaxis. There are presently no approved therapies for children under 4 years. This was a phase 3, double-blind, randomized, placebo-controlled trial at 51 sites in eight countries across the United States, Canada, Australia, and Europe. The trial was conducted between July 2017, and April 2022, and 362 patients were randomized. An epi-cutaneous peanut patch system was used as a therapeutic device and the placebo group received a similar device without the peanut ingredient.

The authors found treatment with a peanut patch was superior to placebo in desensitizing children 1 to 3 years of age with peanut allergy. Anaphylaxis was reported in 7.8% of patients in the intervention group and 3.4% of those in the placebo group. 3.3% of patients discontinued the study in the intervention group and there were no discontinuations in the placebo group.

Comments – The study's participants were highly selected to those without severe allergies or anaphylaxis reactions prior, therefore generalization is limited. This study, however, is the first to use a non-oral approach to food desensitization and may have some future application of peanut and other allergy treatments. **Full Access:** AAP

Cause for Applause Q3 2023—The College's Newest Fellows

We would like to welcome the following new fellows of the College of Urgent Care Medicine! These fellows represent the best of us who work every day to provide the highest quality of medicine and advance the specialty of Urgent Care Medicine. The following individuals applied and earned the distinction of Fellow in the College of Urgent Care Medicine since our last announcement in April 2023.

Brian Cruz, MD, MBA, FACEP, FCUCM
Dale Dupaquier, MD, FCUCM
John George, PA-C, FCUCM
Lynda Gerberg, MD, FCUCM
Michael Green, MD, FCUCM
Claudia Guerra, DNP, FCUCM
Jeanne Kenkare, DO, FCUCM
Michael Kim, DO, FCUCM
Graig Straus, DNP, APRN, FCUCM
Lyndsie Watkins, PA-C, FCUCM
Mark Zeitzer, MD, FACEP, FAAEM, FCUCM

Do you want to be recognized? Requirements to become a fellow include: Actively practicing as a physician, PA, and NP with a solid foundation in Urgent Care and being an active member of CUCM for at least one year. Further requirements can be found here.

Those who achieve fellowship status will be entitled to use the initials FCUCM for as long as they remain members of the College.

CONTINUING MEDICAL EDUCATION (CME)

Target Audience

This CME activity is intended for medical professionals who practice medicine in the on-demand space including Urgent Care, retail medicine and other similar venues. These providers may include physicians, nurse practitioners, and physician assistants.

Designation Statement

The Urgent Care Association (UCA) designates this enduring material activity for a maximum of 3 AMA PRA Category 1 Credit(s)™. Physicians should claim credits only commensurate with the extent of their participation in the activity. Credits may be claimed for one year from the date of release of this issue.

CME Objectives

1. Provide updates on the diagnosis and treatment of clinical conditions commonly managed by on-demand providers
2. Alert on-demand providers to potential unusual cases that may present to them
3. Utilize tips and tricks to improve patient care in the on-demand space

Accreditation Statement

This activity has been planned and implemented in accordance with the accreditation requirement and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the Urgent Care Association and the College of Urgent Care Medicine. UCA is accredited by the ACCME to provide continuing medical education for physicians.

CME Credit Instructions

Once you have read the article, please log into your UCA profile. Once you are logged in go to Learn-> CME->Request CME. Complete the survey with the requested information for Urgent Caring. Your certificate will then be emailed to you within 3-5 business days. Please email learning@urgentcareassociation.org with questions.

CUCM CME Planning Committee

Chris Chao, MD
Reports no financial interest relevant to this publication
Tracey Davidoff, MD, FCUCM
Reports no financial interest relevant to this publication
Ryan Grabeic, FNP
Reports no financial interest relevant to this publication
Patrick O'Malley, MD
Reports no financial interest relevant to this publication
Cesar Mora Jaramillo, MD, FCUCM
Reports no financial interest relevant to this publication
Brad Layman, PA-C, CPC, CEMC
Reports no financial interest relevant to this publication
Max Lebow, MD, MPH, FACEP, FACOEM
Reports no financial interest relevant to this publication

Sean McNeeley, MD, FCUCM
Reports no financial interest relevant to this publication
Gita Pensa, MD
Reports no financial interest relevant to this publication
Victoria Pittman, PA-C
Reports no financial interest relevant to this publication
Michael Weinstock, MD
Reports no financial interest relevant to this publication

Disclaimer

Medical practice and knowledge are constantly evolving and changing. This information is peer-reviewed but should not be your only source. Clinicians should use discretion when applying knowledge to any individual patient.

URGENT CARING STAFF MEMBERS

Editors-in-Chief
Tracey Q. Davidoff, MD, FCUCM
Cesar Mora Jaramillo, MD, FAAFP, FCUCM

Occupational Medicine Section
Max Lebow, MD, MPH

Advancing the Specialty/Antibiotic Stewardship Section
Joseph Toscano, MD, FCUCM

Wound Management Section
Patrick O'Malley, MD

ECG Section
Jerry Jones, MD, FACEP, FAAEM

Oral and Maxillofacial Section
Brian Sun DMD, MS

Coding Section
Brad Laymon PA-C, CPC, CEMC

Administrator
Laurel Stoimenoff, PT, CHC

Publisher
Urgent Care Association

CUCM Board of Directors: Executive Committee

Chris Chao, MD, President
Chrysa Charno, PA-C, FCUCM, Vice-President
Cesar Mora Jaramillo, MD, FAAFP, FCUCM, Treasurer
Erin Topf- Loo, PA-C, Secretary
Jasmeet Bhogal, MD, FCUCM, Immediate Past President

Disclaimer: This material in this publication is for educational purposes only. Medical practice and knowledge are constantly evolving and changing. This information is peer-reviewed but should not be your only source. Clinicians should use discretion when applying knowledge to any individual patient.

Exploring Non-Opioid Pain Treatments in the Urgent Care

Was brought to you by:

HIPPO EDUCATION

From Hippo Education: For more on this topic, listen to Dr. Ross Cohen and Jackie McDevitt-Capetolla PA-C discuss "Non-Opioid Pain Medication Tips and Tricks" in a special Urgent Care RAP podcast segment recorded *live* at the UCA conference!

And if you're looking for a way to fulfill the new DEA educational requirements, please check out Hippo Education's OUD Decoded: A DEA Compliant Course. This course is available for free until the end of September.

Tick-Borne Illness: Diagnostic Approach for the Urgent Care Clinician and Evaluation and Management of the Red Eye

Was brought to you by:

EB MEDICINE
EMPOWERING PHYSICIANS WITH EVIDENCE-BASED CONTENT

From EB Medicine: Learn more about *Evidence-Based Urgent Care* and get a free sample issue at https://www.ebmedicine.net/urgent-care-info

https://clinicalpathways.ebmedicine.net/