Urinary Tract Infection in Male Patients

Challenges in Management

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KEYWORDS

• Male • Urinary tract infection • Duration • Bacteremia

KEY POINTS

- Treatment of urinary tract infections (UTIs) in male patients has several important distinctions compared to the treatment of UTIs in female patients.
- Distinctions include the more varied microbiology of the causative organisms, the potential need for longer duration therapy, and the relative paucity of clinical trials specifically addressing UTI in males versus females.
- Practical implications include the necessity of obtaining a pretreatment urine culture, careful consideration of therapy duration, and accepting that there are more areas of uncertainty regarding management in UTI in males versus females.

INTRODUCTION

Urinary tract infection (UTI) is among the most common infections in both ambulatory and hospitalized patients.[1] Differences between sexes exist, with UTI being considerably less common in male versus female individuals below the age of 70 years.[1,2] After this age, there is a narrowing of the sex gap, with incidence of UTI among male and female individuals becoming closer, but never reaching parity, over the age of 70 years.[2] Because it is so common, and also it is one of the most commonly misdiagnosed infections, UTI treatment accounts for a large amount of antimicrobial use across all settings: ambulatory care, hospitalized patients, and long-term care facilities.[3] A majority of UTIs are caused by gram-negative organisms, for which there is a relative paucity of oral antimicrobials with predictable activity. This can make choosing empirical therapy challenging for clinicians and further add to the problem of steadily increasing antimicrobial resistance.

The principles of management of UTI in male patients are relatively straightforward: make an accurate diagnosis of UTI, with as much specificity as possible (ie,

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Infect Dis Clin N Am 38 (2024) 311–323
https://doi.org/10.1016/j.idc.2024.03.009
0891-5520/24/Published by Elsevier Inc.
id.theclinics.com

differentiating between cystitis, pyelonephritis, and asymptomatic bacteriuria, etc.); obtain source control (if necessary); identify the causative pathogen; prescribe empirical therapy based on the suspected UTI syndrome and level of illness; and adjust therapy (if needed) for an appropriate duration. Additionally, after the acute UTI episode, consideration should be given as to whether any predisposing factors to UTI are present and whether they can be eliminated or mitigated. Although the principles can be described as relatively straightforward, knowledge gaps can make implementation challenging and sometimes there are limited options for patients and clinicians to turn to. Finally, throughout this article "male" will refer to biological sex versus the patient's gender identity. This is done because specific anatomic structures and differences exist that are believed to confer important differences with regard to the risk of UTI (presence of a longer urethra allowing more distance between the urethral meatus and the anus) and management (including presence of the prostate, with its unique vasculature making it a relatively protected space from certain antimicrobials). Data on optimal management of UTI in transgender men are needed, particularly regarding the effect of gender-affirming surgery.

EPIDEMIOLOGY

The incidence of UTI among men is lower than women, with 50% of women self-reporting a UTI by their early 30s, whereas among 70 year old men only 20% self-report a UTI. At any age, over 10% of female individuals report a UTI episode in the preceding 12 months, whereas among male individuals the percentage exceeds 5% only after the age of 70 years and never reaches 10%.[2,4] Notably, even though UTI rates in male patients are lower than those observed in female patients, with more than 100 million male patients over the age of 18 years in the United States, even these lower rates result in millions of episodes of UTI in male patients. The most accepted reasons for why male individuals have fewer UTI episodes than female individuals are (1) longer distance from anus to the urethral meatus, (2) longer distance from the urethral meatus to the bladder, and (3) less moisture in the periurethral spaces, making them less hospitable for microbial colonization. Conversely, activities which increase the density of enteric bacteria near or in the urethral meatus, including sexual activity, fecal incontinence, and catheter use, can all increase the risk of UTI, although data supporting these as risk factors are of variable quality.[5]

The rise in the incidence of UTI in older males is multifactorial, but functional or anatomic bladder obstruction, along with efforts to mitigate these issues, is believed to be major causes. Functional obstruction can result from neurologic deficits ranging from traumatic spinal cord injury to diabetic neuropathy, ultimately resulting in difficulty voiding and increased postvoid residual urine in the bladder. Anatomic obstruction is typically from prostatic enlargement, which results in similar voiding issues. Efforts to mitigate these issues include behavioral changes such as minimizing fluid intake, which can increase the risk of UTI by decreasing urine volume and flow and the use of urinary catheters (intermittent or indwelling), which can introduce micro-organisms into the urethra, facilitate entry to the bladder, and cause urethral trauma. Efforts to eliminate or reduce the obstruction should reduce the risk of UTI, but depending on the approach used, new risks may be introduced. For instance, surgical removal of prostatic tissue may relieve much of the anatomic obstruction but may also cause neurologic complications impacting continence and control of urination and thus have no net benefit. In general, noninvasive or medical therapies to reduce prostate size should be attempted first, with surgical procedures reserved for those who receive no or insufficient benefit from medications targeting prostatic hypertrophy or malignancy.

MICROBIOLOGY

The causative organisms in UTIs in male individuals are more varied than those seen with women, where *Escherichia coli* is the clear majority.[6] In men, *E coli* still predominates, but often is a plurality versus the majority.[7] The other organisms frequently encountered are also usually from the Enterobacterales family (*Klebsiella*, *Enterobacter*, *Citrobacter*, and others), but other important organisms include *Pseudomonas aeruginosa*, enterococci, staphylococci (both *Staphylococcus aureus* and various coagulase-negative staphylococci), and *Candida* species. The practical upshot of this microbial diversity is that predicting the causative organism is difficult, and this combined with increasing antimicrobial resistance necessitates a urine culture (UC) for optimal management. This will ensure effective antimicrobial therapy, and in certain cases, the pattern of organisms isolated can give clues as to the etiology of recurrence. For instance, a patient with repeated UTIs with isolation of the same organism with similar antimicrobial susceptibility testing may warrant evaluation for a nidus of infection, such as urinary calculi or prostatic involvement. In contrast, a patient with repeated UTIs having a different organism isolated each time is unlikely to have such a nidus and may instead benefit from exploration of risk factors such as sexual activity, catheter use, and so forth. Once a pretreatment UC has been obtained, clinicians should initiate antimicrobial therapy based on the specific UTI syndrome and severity of illness and potentially utilizing the facility antibiogram and any recent history of antimicrobial exposure.

SPECIFIC SYNDROME DEFINITIONS AND NOMENCLATURE

Cystitis

Cystitis refers to infection of the urinary bladder, with symptoms and examination findings reflecting an infection limited to this space. Important features of cystitis include the lack of systemic symptoms and the ability to use drugs that concentrate well in the bladder but lack good systemic distribution, such as nitrofurantoin and oral fosfomycin. Cystitis may also be referred to as lower tract disease or a lower UTI.

Complicated Urinary Tract Infection

The term “complicated UTI” lacks a universally agreed upon definition, which has hampered its clinical utility.[8] Some definitions have considered a UTI to be “complicated” if the infection occurs in the setting of anything making UTI more likely to occur, more difficult to eradicate, or more likely to recur.[9] These include anatomic conditions such as prostate enlargement, urinary calculi, bladder diverticula, polycystic kidneys; medical conditions such as diabetes and chronic kidney disease; and iatrogenic factors such as catheter use, indwelling urethral stents, and percutaneous nephrostomy tubes. Others have utilized a more practical approach to the term “complicated” and considered a complicated UTI to be one where there is evidence that the infection has spread beyond the bladder.[10] This definition focuses on decisions being made at the time of treatment, including whether there is need for an agent with good systemic distribution, and not on the presence of underlying anatomic details that may not be known by the treating clinician. Finally, male sex has sometimes been invoked as sufficient criteria to consider a UTI as “complicated.”[11] Relative to treating UTI in female patients, where microbiology is more predictable and shorter courses of antimicrobials have repeatedly been shown to be effective, it can be argued that treating UTI in male patients is indeed more complex. For the sake of uniformity, here complicated UTI will refer to anything other than cystitis, but because of the lack of an accepted definition, the term will be used sparingly.

Pyelonephritis

Pyelonephritis implies renal infection, with signs and symptoms reflecting this involvement.[12] Note that while nonspecific symptoms such as fever, chills, and malaise are common with pyelonephritis, none of these localize the infection to the kidneys. Symptoms and findings that do suggest renal involvement are a history of flank pain and costovertebral tenderness on examination. Occasionally, imaging studies can suggest renal involvement in a patient without any symptoms or findings to suggest pyelonephritis; the significance of such incidentally discovered renal involvement is uncertain. Pyelonephritis may also be referred to as upper tract disease or upper UTI.

Febrile Urinary Tract Infection

Whereas pyelonephritis is defined by symptoms and signs localizing infection to the kidney, febrile UTI is defined by the presence of a fever, other evidence of a UTI, but no localizing features to support renal involvement. Some evidence supports prostate or seminal vesical involvement in male patients with febrile UTI, although the clinical significance of this is uncertain.[13]

Sepsis and Bacteremia

Patients meeting sepsis criteria from a urinary source are a more severe subset of the UTI syndromes with systemic involvement (pyelonephritis and febrile UTI), many of whom (but not all) will also be found to be bacteremic. Conversely, a small subset of patients with vital signs not meeting sepsis criteria may be found to be bacteremic; indeed, in rare cases, patients may be found to have bacteremia based on blood cultures that were obtained for relatively nonspecific complaints such as lethargy or fatigue in the setting of bacteriuria.

Asymptomatic Bacteriuria

Asymptomatic bacteriuria was previously defined as the repeated isolation of $\geq$ 100,000 colony-forming units (cfu)/mL on 2 consecutive voids in females or the isolation of $\geq$ 100,000 cfu/mL from a single sample in males, presuming that the source patient was asymptomatic.[14] A lower threshold ($\geq$100 cfu/mL) from a catheterized urine specimen was considered to represent asymptomatic bacteriuria in both male and female patients.[14] More recent updates to guidelines on the management of asymptomatic bacteriuria have simplified this to a more simple and clinically useful definition of 1 or more species of bacteria growing in the urine at $\geq$ 100,000 cfu/mL, in the absence of signs or symptoms attributable to UTI, regardless of sex or catheter status.[15]

Bacteriuria with Nonlocalizing Symptoms

Whereas asymptomatic bacteriuria is defined by bacteriuria without symptoms, bacteriuria in the setting of symptoms not plausibly related to the urinary tract frequently comes to medical attention.[16] This includes bacteriuria discovered in the setting of evaluation for fatigue, altered behavior, irritability, and decreased appetite. The presence of a symptom or observable change in behavior distinguishes this from true asymptomatic bacteriuria and can raise concerns among clinicians that the nonlocalizing symptoms are a subtle manifestation of UTI.

DIAGNOSIS AND TREATMENT

Diagnosis and treatment depends on the specific UTI syndrome, severity of illness, and patient-specific factors such as recent antimicrobial exposure of other risk for infection due to a drug-resistant organism. The data supporting treatment for some

syndromes are more robust than others, but for all of them, the general principles of making an accurate diagnosis, considering source control, selecting empirical therapy, and choosing the definitive combination of drug and duration will be relied upon.

Cystitis

Cystitis should be suspected with new-onset dysuria, frequency, and urgency. Cloudy or bloody urine can also occur, although note that urine appearance alone is not sufficient to make a diagnosis of cystitis. Systemic symptoms such as fever, chills, and malaise should be elicited, as should symptoms localizing the infection to the kidney, such as flank, back, or costovertebral angle pain. Any factors that increase the likelihood of increasing the density of bacteria in the periurethral area increase the pretest probability of infection; these would include intermittent catheterization, recent urethral procedures, and insertive anal intercourse. Patients reporting recent sexual activity should also be asked about urethral discharge or other signs of sexually transmitted infection (STI), especially if they have new or multiple partners, since this would increase the suspicion for STI and decrease the likelihood of UTI. Expected physical examination findings of cystitis include lack of fever and no back or costovertebral angle tenderness, with suprapubic tenderness being the only abnormality typically found.

Initial laboratory workup for suspected cystitis includes a urinalysis (UA) and UC. Stewardship efforts to reduce inappropriate treatment of asymptomatic bacteriuria support only proceeding with UC in the presence of an abnormal UA,[17] whereas the practice of automatically ordering a UC with every abnormal UA (regardless of symptoms) will increase the detection of asymptomatic bacteriuria and drive unnecessary antimicrobial use.[18] If STIs are suspected, testing for herpes simplex virus, gonorrhea, chlamydia, syphilis, and human immunodeficiency virus should be obtained, as appropriate. Other laboratory studies are not necessary for most cases of suspected cystitis, unless there is reason to suspect acute renal impairment (pre-existing renal disease, recent increased fluid losses, or dehydration), in which case a chemistry panel to assess the current renal function should be obtained to guide antimicrobial dosing. If cystitis is suspected and the UA shows the evidence of pyuria (either by visualization of white cells on microscopy or detection of leukocyte esterase, a surrogate marker of white cells), empirical treatment for cystitis should be prescribed. If the UA does not show pyuria but suspicion of cystitis is high, clinical judgment is needed. Options include empirical treatment or symptom control with analgesics while awaiting the UC result. A negative UC should prompt exploration for another diagnosis, whereas microbial growth suggests the presence of cystitis despite the lack of pyuria and treatment targeting the isolated organism should be provided.

Empirical therapy for cystitis should target the most likely causative organisms, which typically include *E coli* and other gram-negative bacteria, but gram-positives such as enterococci and staphylococci can be encountered. If a urine-specific antibiogram is available, this can help provide more specific guidance, and review of prior UCs should also be performed. Isolation of an organism within the past 6 months increases the chance of isolating the same organism[19]; some studies have found useful information from even older UC results. Antimicrobial regimens for cystitis are listed in **Table 1**; local susceptibility results, drug tolerability, comorbidities, and drug–drug interaction should be used to select the best empirical option for each patient, with UC results guiding any changes (if necessary) for directed therapy. Therapy duration of 7 days is suggested, based on no observed benefit to longer duration treatment,[7,20] but it is possible that durations used for cystitis in female patients (3–5 days) may be sufficient.

Table 1
Antimicrobial options for the treatment of cystitis in male patients. A duration of 7 days is suggested for all regimens unless otherwise noted

Antimicrobial	Dose (Assuming Normal Renal Function)	Comments
Nitrofurantoin macrocrystals	100 mg po twice daily	Concentrates well in the bladder, but not in other tissues, limiting use to cystitis
Trimethoprim–sulfamethoxazole	160/800 mg po twice daily	Hyperkalemia a concern in patients on ACE inhibitors, spironolactone, and so forth
Fosfomycin	3 g po every 3 d × 3 doses	Frequency of dosing and duration not well established. Similar to nitrofurantoin, for cystitis use only
Ciprofloxacin	500 mg po twice daily	Multiple adverse effects to consider: *Clostridioides difficile*, QT prolongation, dysglycemia, altered mental status, and more
Levofloxacin	500 mg po daily	Identical concerns as with ciprofloxacin
Amoxicillin–clavulanic acid	875/125 mg po twice daily	Activity against enterococci and gram-negative organisms; amoxicillin alone more tolerable for enterococci but with less gram-negative spectrum
Cefpodoxime	400 mg po twice daily	Other oral third-generation cephalosporins (cefdinir, cefixime) can be considered based on availability

Abbreviations: ACE, angiotensin converting enzyme; g. grams; mg, milligrams; po, by mouth.

Pyelonephritis

Pyelonephritis typically presents with fever, flank, back, or costovertebral angle discomfort and often may have associated lower urinary symptoms of dysuria, frequency, and urgency.[12] Other systemic symptoms that can occur include malaise, nausea, vomiting, and loss of appetite. Other infections should be considered, including appendicitis, diverticulitis, cholecystitis, and cholangitis. Examination findings of pyelonephritis include vital signs common to all systemic infections: fever, tachycardia, and hypotension. Costovertebral angle tenderness can often be elicited, usually unilaterally, although bilateral pyelonephritis can be encountered. Findings expected in biliary disease and diverticulitis should be looked for to help make a more definitive diagnosis.

Laboratory evaluation for suspected pyelonephritis is more extensive than that for cystitis. A UA and UC should be obtained, in addition to a complete blood count with differential and a basic metabolic panel. Blood cultures should be drawn if the patient meets sepsis criteria or otherwise appears severely ill, and in such patients, a urine Gram stain may also be very useful in selecting empirical therapy. Other laboratory studies (liver function tests and pancreatic enzymes) may be necessary to exclude other diagnoses. Most patients with pyelonephritis will not need imaging unless it is needed to evaluate another possible diagnosis. Patients presenting with severe sepsis may need imaging to assess the need for emergent source control, such as extraction of an obstructing stone or drainage of an abscess. In that case, contrast-enhanced computerized tomography (CT) scanning is the preferred modality, but ultrasonography can also provide useful information without radiation exposure.

Treatment of pyelonephritis includes both antimicrobial therapy and nonspecific measures such as volume resuscitation, analgesia, antiemetics, and other supportive cares. While vital, the nonantimicrobial measures will not be discussed further here. Empirical antimicrobial therapy for pyelonephritis can range from an oral agent such as ciprofloxacin for a mild case that is managed in the ambulatory setting, to novel antimicrobials such as ceftazidime/avibactam for a patient suspected of having a multidrug-resistant organism causing their UTI. The causative organisms for pyelonephritis are largely similar to those causing cystitis, but important differences are present that make the list of empirical regimens for pyelonephritis quite different (**Table 2**). These differences include a need for a drug with adequate blood and tissue penetration, such that nitrofurantoin and oral fosfomycin should not be used; a spectrum of illness that includes critically ill patients, necessitating empirical therapy with a broader spectrum of activity and the need for intravenous therapy in patients with severe illness or not able to tolerate oral intake. Selection of a regimen requires knowledge of local susceptibility results, severity of illness, ability to tolerate oral therapy, and an assessment of whether the patient has a risk of an organism with significant antimicrobial resistance. Patients who are being treated with oral therapy in the outpatient setting can be given a one-time dose of a parental agent at the onset of treatment, especially if local resistance rates make it impossible to select an oral agent that is reliably active against the expected organisms. Just as with cystitis, UC results should guide any changes for definitive therapy. Increasingly, clinicians will find themselves treating pyelonephritis caused by highly resistant organisms such as extended-spectrum beta-lactamase (ESBL)-producing Enterobacterales, carbapenem-resistant Enterobacterales (CRE), or other organisms. The challenges posed by these organisms are not unique to treating UTI, and guidance for navigating these complex infections and the use of novel agents can be found in recent Infectious Diseases Society of America (IDSA) guidelines.[21] Therapy duration of 7 days is suggested for most patients, based

Table 2
Antimicrobial options for the treatment of pyelonephritis and febrile urinary tract infection in male patients. A duration of 7 days is suggested for all regimens unless otherwise noted

Antimicrobial	Dose (Assuming Normal Renal Function)	Comments
Ciprofloxacin	500 mg po twice daily	Adverse events as per comments in Table 1. Use for empirical therapy should be informed by local susceptibility data and severity of illness
Levofloxacin	500 mg po daily	Identical concerns as with ciprofloxacin
Trimethoprim–sulfamethoxazole	160/800 mg po twice daily	Hyperkalemia concerns. Use for empirical therapy should be informed by local susceptibility data and severity of illness
Amoxicillin–clavulanic acid	875/125 mg po twice daily	Potential use for empirical therapy in ambulatory patients with intolerant of other oral options
Ceftriaxone	1–2 g IV daily	May not be appropriate for severely ill patients if ESBL-producing organisms suspected
Piperacillin-tazobactam	3.375 g IV every 6 h	May be active against ESBL-producing organisms, but not preferred agent if they are identified
Cefepime	2 g IV every 8 h	May be active against ESBL-producing organisms, but not preferred agent if they are identified
Gentamicin	5 mg/kg IV once daily	Nephrotoxicity precludes use for definitive therapy, but can be useful for empirical therapy if highly resistant gram-negative infection suspected
Ertapenem	1 g IV daily	Active against most ESBL-producing organisms; lacks pseudomonal coverage
Meropenem	500 mg IV every 6 h	Active against most ESBL-producing organisms; has pseudomonal coverage
Vancomycin	15 mg/kg IV every 12 h	Pharmacist guidance for dosing encouraged, if available. Reasonable to include as part of empirical therapy if gram-positives suspected or seen on urine Gram stain
Meropenem–vaborbactam, ceftazidime–avibactam, cefiderocol, ceftolozane–tazobactam	See ref[21] for specific dosing guidance for these agents and for other options for treatment of highly resistant gram-negative infections	Relatively novel agents whose use should be reserved for treatment of highly resistant gram-negative infections

Abbreviations: ESBL, extended-spectrum beta-lactamase; g, grams; IV, intravenous; mg, milligrams; kg, kilograms; po, by mouth.

on trials demonstrating similar clinical outcomes in male patients with pyelonephritis and other febrile UTI syndromes compared to longer therapy[22,23] and a trial demonstrating that gram-negative bacteremia can also be treated with 7 days of antimicrobial therapy.[24] In one of the trials comparing 7 versus 14 days of therapy for men with febrile UTI, there was a greater incidence of post-treatment bacteriuria in the shorter group (20.9% vs 6.4%; $P = .001$), but the clinical success was similar, albeit still slightly favoring longer therapy (95.6% vs 100%; $P = .02$).[23] Notably, this trial used a regimen consisting predominantly of ofloxacin, 200 mg twice daily, a relatively unusual and potentially underdosed treatment. Ofloxacin is an equal mixture of the L and R enantiomers of the drug, the L enantiomer (commercially available as levofloxacin) being the active form, with levofloxacin typically dosed at 500 mg daily (see **Table 1**) versus the total of 200 mg daily received as half the ofloxacin dose in this trial. Given the unusual regimen, and a gain of just 5% in clinical success, it is not clear that doubling antimicrobial duration for all men is warranted. A possible exception to shorter therapy being preferred is pyelonephritis caused by *S aureus*, particularly if bacteremia is present; this may require longer duration therapy, with some advocating that the entire course is intravenous in the setting of bacteremia.[25]

Febrile Urinary Tract Infection

Febrile UTI should be suspected in a patient with symptomatic and laboratory evidence of UTI, but without any symptoms or examination findings localizing the infection to the kidney. Laboratory and imaging workup is identical as that described for pyelonephritis, with imaging reserved for cases where the diagnosis is uncertain or with severe illness to evaluate for a potential drainable source. Note that occasionally imaging will reveal evidence of renal involvement, such as abnormal contrast enhancement in the kidney or perinephric fat stranding on CT, or an edematous and enlarged kidney on ultrasound. Whether such imaging evidence of renal involvement in a patient without localizing symptoms is sufficient to diagnose a patient with pyelonephritis is largely academic; management will not be changed. Treatment should again be guided by severity of illness, with oral therapy (and possibly a one-time dose of a parenteral agent) appropriate for patients managed in the outpatient setting, and intravenous therapy for those being admitted to the hospital. Nitrofurantoin and oral fosfomycin should again be avoided, as the presence of fever makes infection beyond the bladder likely. Antimicrobial selection, de-escalation, and duration are all identical to those described for pyelonephritis.

Sepsis and Bacteremia

Sepsis and bacteremia can result from both pyelonephritis and febrile UTI and vital sign abnormalities can be how both syndromes become clinically apparent. Sepsis should be presumed and promptly evaluated with any patient presenting with vital sign derangements meeting sepsis criteria (temperature >38.0°C or <36.0°C, heart rate >90, respiratory rate >20, presence of leukocytosis or leukopenia, and suspicion of infection).[26] Severe sepsis/septic shock criteria such as lactic acidosis or persistent hypotension despite fluid resuscitation should prompt even more urgent evaluation and initiation of treatment. Laboratory evaluation and physical examination should be directed at identifying the source of the infection (UTI, biliary tract, and respiratory), with imaging used to further investigate the area of suspected infection. The threshold to obtain imaging is lower as severity of illness increases, since emergent source control may be necessary to achieve clinical stability. Specific entities that should be looked for because urgent intervention is needed including emphysematous pyelonephritis, urinary obstruction, and perinephric abscess.

If the initial UA is consistent with infection and available history and examination is also suggestive of UTI, a presumed diagnosis of sepsis from a urinary source can be made and treatment should be initiated with antimicrobials targeting the likely organisms based on local susceptibilities and patient risk factors for resistant organisms (including recent travel to areas with increased antimicrobial resistance, recent systemic antimicrobial use, and prior isolation of resistant organisms). The parenteral agents in **Table 2** are all potential options for empirical therapy; a urine Gram stain can help narrow the empirical regimen to a single agent, but if unavailable, a regimen with both gram-positive and negative coverage should be utilized. Piperacillin–tazobactam or cefepime plus vancomycin would be a reasonable empirical regimen for a patient with sepsis of presumed urinary source; if an ESBL-producing organism or CRE is suspected or confirmed, the choice of gram-negative agent will need to be adjusted accordingly. In patients with severe sepsis and a high suspicion for the presence of a resistant organism, adding a second gram-negative agent from a different class can increase the odds of at least one of them having activity. Once the patient's condition improves and the causative organism has been identified, directed therapy can be chosen based on susceptibility testing, current clinical status, and whether oral medications are tolerated. Even with bacteremia, if a patient is clinically stable, can take oral medications, and an effective oral option is available, directed therapy with an oral agent to complete a 7 day course is preferred. Exceptions to the use of oral therapy and shorter duration treatment are *S aureus* bacteremia and potentially patients whose clinical improvement was slower than expected. In this latter group, extending therapy may be beneficial, but supporting evidence for this is lacking. Beyond antimicrobials, expert care for the critically ill patient is vital, including fluid resuscitation, respiratory support, vasopressors, and corticosteroids, if indicated.

Asymptomatic Bacteriuria

Including asymptomatic bacteriuria in the spectrum of UTIs is somewhat counterintuitive. In most cases, asymptomatic bacteriuria should not be treated, since no benefit has been observed in multiple randomized controlled trials, and asymptomatic bacteriuria is a large driver of antimicrobial use in multiple health care settings.[15] The notable exceptions to this are during pregnancy (not applicable to this population) and prior to a urologic procedure where urothelial bleeding is anticipated. Obtaining a UC such that the results of susceptibility testing are available before the procedure allows selection of an active antimicrobial, which should be administered in the 1 to 2 hours before the procedure. One another situation where the benefit of asymptomatic bacteriuria treatment is uncertain is in the first month following renal transplantation; a recent IDSA guideline on the treatment of asymptomatic bacteriuria found insufficient evidence to make a recommendation for or against screening for or treating asymptomatic bacteriuria in this population but did strongly recommend not screening for or treating asymptomatic bacteriuria in patients more than a month from their renal transplant.[15] Regarding testing for asymptomatic bacteriuria, the most useful advice is to only look for it when discovery would warrant treatment. Efforts to eliminate routine preoperative, reflex UCs after abnormal UAs, or other routine UCs can avoid significant amounts of unnecessary antimicrobial use.[17,27,28]

Bacteriuria with Nonlocalizing Symptoms

The identification of bacteriuria in a patient with nonlocalizing symptoms often results in clinical uncertainty. Detecting bacteriuria in such patients can lead to desirable outcomes such as prompt identification and treatment of an occult bacteremia with the same organism that was isolated in the UC, but can also lead to undesirable outcomes

such as attributing falls to a UTI, when in reality there was a treatable non-UTI cause only discovered after significant delay.[16] With generations of physicians having been told a version of "UTI can present in subtle ways," it is not surprising that patients presenting with nonspecific complaints such as fatigue, altered behavior, irritability, decreased appetite, or simply "just not acting themselves" end up having a UC obtained as part of their evaluation. With asymptomatic bacteriuria being common in many patient populations, including those in long-term care facilities, using catheters, with neurologic impairment, and with advanced age,[15] these UCs will often have microbial growth, leaving the clinician with the dilemma of how to manage this result. Clear guidance on how to manage this would be welcome, ideally from a randomized controlled trial with patients randomized to immediate antimicrobials versus supportive care and antimicrobials only initiated with evidence of systemic infection (ie, fever and/or leukocytosis). In the absence of such data, clinical judgment is needed. A suggested approach would be to initiate supportive cares (hydration, addressing possible polypharmacy, evaluation for non-UTI causes of the symptoms) and reserve antimicrobials only for evidence of systemic infection. Alternatively, if antimicrobials are initiated but ultimately a non-UTI cause is found, antimicrobials should be promptly discontinued.

SUMMARY

Appropriate management of UTI in male patients begins with recognition of the specific syndrome and then matches the intensity of the diagnostic evaluation and empirical therapy to the severity of illness. Patients with severe illness may warrant early imaging of the urinary tract to evaluate for obstruction, abscess, or other need for source control. Because the microbiology of UTI in male patients is varied and difficult to predict, a UC is needed for optimizing definitive therapy. Outside of a few specific circumstances such as UTI complicated by *S aureus* bacteremia, therapy duration of 7 days is suggested, with transition to oral therapy as soon as clinical improvement allows.

CLINICS CARE POINTS

- Obtaining a baseline urine culture is essential.
- Defining specific syndrome guides diagnosis and treatment.
- Routine treatment duration should rarely exceed 7 days.
- Severe illness necessitates more aggressive empirical therapy.

DISCLOSURE

Dr D.M. Drekonja has received grant funding from the Veterans Affairs Administration for research on urinary tract infection in male veterans.

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