Asymptomatic Versus Symptomatic Urinary Tract Infections In Long-Term-Care-Facility Residents

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*You are in your office seeing patients when a nurse calls from the nursing home to report the urinalysis results of Mrs. X. Mrs. X, 75 years-old, with mild Alzheimer's dementia, was admitted to the facility for rehabilitation and low back pain control after two falls. Her medical history also includes hypertension, hyperlipidemia, **chronic obstructive pulmonary disease (COPD)**, osteoporosis, and two new vertebral compression fractures. Yesterday the nursing staff noticed foul smelling urine and contacted your covering physician for an order to obtain a clean catch urine. Currently, Mr.s X is afebrile, without any specific complaints. She takes oxycodone and acetaminophen for pain control, donepezil for her Alzheimer's, a bisphosphonate for her osteoporosis, and an albuterol inhaler for her COPD. She has no medication allergies. The urinalysis reveals 20-30 WBC/ml, 5-10 RBC /ml, and numerous bacteria. A urine culture has been sent but results are unavailable. The nurse asks whether you would like to start antibiotics.*

Urinary tract infections (UTIs) are the most common bacterial infection in older populations, both in the community and in the **nursing home (NH)**.[1] UTIs are the most common reason for antimicrobial prescriptions in NHs, and are responsible for the initiation of 20-60% of systemic antimicrobial courses in long term care residents. [2] Fifty percent of female and 40% of male residents have been reported to have a UTI at one time or another. Nursing home residents are at particular risk for UTIs. Risk factors for infection in this population include immobility, which leads to incomplete bladder emptying; poor hygiene, which favors bacterial growth; incontinence; and age-associated physiological changes, such as a decline in the effectiveness of immune function, loss of estrogen effect on genitourinary mucosa, and changes in colonizing flora. [3-5]

Despite the high prevalence of UTIs in the NH, most patients are clinically asymptomatic. Numerous organizations (e.g., the American Medical Director's Association, The Infectious Disease Society of America) have advocated against the treatment of asymptomatic bacteriuria for a number of reasons. First, the presence of asymptomatic bacteriuria in the older patient - including the diabetic - does not predict future UTI or mortality. [2,6,7] Furthermore, treatment of asymptomatic bacteriuria does not prevent recolonization or reduce the risk of developing symptomatic UTI. On the other hand, unnecessary treatment of asymptomatic UTIs has been correlated with increased resistance in colonizing bacteria.

Are there criteria that I can use to determine if my patient has asymptomatic or symptomatic bacteriuria?

The Department of Health and Human Services and the Centers for Medicare and Medicaid Services issue yearly guidelines for NHs that state that only residents meeting the McGeer Criteria should be treated for UTI. [8,9] Urine culture results are not required by these criteria to make a decision on empirical coverage. The McGeer criteria for NH residents without an indwelling catheter state that 3 of the following criteria must be met to identify a UTI: (1) a temperature of 38 C (100.4 F) or higher; (2) new or increased burning sensation on urination, frequency of urination, or urgency of urination; (3) new flank or suprapubic pain or tenderness; (4) change in character of urine; and (5) worsening of mental or functional status. Other guidelines for UTIs proposed by Loeb et al. can also be useful as minimum criteria necessary for empirical antibiotic therapy.[10] For nursing home residents without an indwelling catheter, the Loeb criteria recommend empirical coverage in the setting of: acute dysuria alone **or** fever (a temperature of greater than 37.9 C (100 F) or an increase of 1.5 C (2.4 F) above baseline temperature) plus at least 1 of the following symptoms: new or worsening urgency or frequency of urination, suprapubic pain, gross hematuria, costovertebral angle tenderness, or urinary incontinence. [11,12]

You decide not to start antimicrobial therapy for Mrs. X, since her clinical presentation does not meet either the McGeer or Loeb criteria. Three months later, Mrs. X develops dysuria and a fever to 101. Examination reveals tachycardia and suprapubic tenderness. Her urinalysis reveals 30 WBC/ml, 3-5 RBC /ml, 1+ squamous epithelial cells. Given her overt symptoms, you decide to start empiric antibiotic,s pending results from a urine culture. Your patient has no known drug allergies. Which antibiotic is the best option?

EMPIRICAL TREATMENT FOR SYMPTOMATIC UTIs

The treatment of a lower tract UTI can usually be managed in the NH. Choices for empiric regimens should always be made with the knowledge of the individual nursing home's antibiogram and resistance pattern, and resistance patterns of the usual colonizing flora. In most nursing homes, the medical director leads the infection control team and assembles an antibiogram for distribution to other clinicians practicing there. If one is not available, there are several general recommendations.

In the setting of an acute uncomplicated bacterial cystitis in an otherwise healthy adult nonpregnant woman, current recommendations suggest that a 3-day course of a recommended antibiotic is as effective as the same antimicrobial given for a longer duration. [13,14] **Trimethoprim-Sulfamethoxazole (TMP-SMZ)** for 3 days is generally considered first line therapy in non-sulfa allergic patients. Other antibiotics that have been shown to be as equally effective as TMP-SMZ for empiric therapy include: Trimethoprim alone, and renally excreted members of the fluoroquinolone family (e.g., ofloxacin, ciprofloxacin, levofloxacin). Although empirical therapy with a quinolone or other broad-spectrum antimicrobial may be appropriate in selected clinical presentations, the universal application of empirical therapy with a given agent should be discouraged, given the developing resistance to these drugs and the overall expense related to prescribing these medications compared with less costly alternatives. [6,7,14,15]

Other drugs to consider empirically include Nitrofurantoin and fosfomycin, both of which may become more useful in the future as resistance to TMP-SMZ and trimethoprim alone increases. Nevertheless, nitrofurantoin should not be prescribed for patients with a creatinine clearance < 60 ml/min. [14,15] Oral amoxicillin-clavulanate may be used as an alternate agent, and is especially useful when a patient has a polymicrobial infection with both susceptible gram-negative rods and *Enterococcus* spp. Finally, several oral second-and third-generation cephalosporins, such as cefuroxime axetil, cefixime, ceftibuten, and cefpodoxime, may be used as alternate therapies in the management of UTI when patients cannot tolerate first-line therapies or have organisms with resistance to first-line agents. [6,7]

There are many antibiotics that should be avoided for empiric therapy. For example, when given for 3 days, B-lactam penicillins and cephalosporins as a group are less effective than the previously described drugs. Amoxicillin and ampicillin should not be used empirically because many community-acquired and nursing home-acquired strains of *E coli* produce B-lactamase, which renders these agents inactive. Additionally, numerous studies over the years have shown that B-lactam antibiotics, such as penicillins and cephalosporins are not as efficacious in curing cystitis or eradicating uropathogens from their perineal reservoirs. [6,7]

When an oral agent cannot be used, IM ceftriaxone, cefotaxime, or another injectable cephalosporin is appropriate. The duration of treatment for older women with uncomplicated cystitis can be 3 days. Seven to 10 days of treatment should be prescribed for: [6,7]

- Women with more than 1 week of symptoms prior to diagnosis
- Women with structural or functional abnormalities of the urinary tract
- Infection caused by *S saprophyticus*
- Men

Double strength TMP-SMZ is started on Mrs. X. At 48 hours, she feels much better and final urine culture report reveals E coli that is sensitive to TMP-SMZ, ciprofloxacin, levofloxacin, *amoxicillin/clavulanic acid, nitrofurantoin, and ceftriaxone. You opt not to change her previous antibiotics. After the three-day course of TMZ-SMZ, MRs X's family raises a concern as to whether she should repeat urine tests to confirm success of treatment.*

Routine follow-up, including urine culture, is generally unnecessary after treatment for cystitis, unless symptoms do not abate. [16]

A review of Mrs. X's medical records reveals that this is her 3ʳᵈ UTI in 6 months. The nurse asks you if you would like to consider prophylactic antibiotics to prevent future infections.

Prophylaxis should not be initiated until the eradication of active infection is confirmed by a negative urine culture at least one to two weeks after treatment is discontinued. Continuous prophylaxis, typically with medication taken once daily at bedtime, is an option for women who have had two or more symptomatic infections during one 6-month period or three or more such infections over a 12- month period. [16] Acceptable choices for prophylaxis include single strength TMP-SMZ ½ tablet a night or three times weekly; Trimethoprim 100 mg nightly; or Nitrofurantoin macrocrystals 50-100 mg nightly. [16]

The TMP-SMZ prophylaxis is initiated after a negative urine culture at two weeks confirms sterile urine.

Summary of recommendations:

1. Only patients meeting the McGeer or Loeb criteria should be treated for UTI based on currently accepted guidelines.
2. Screening for and treatment of asymptomatic bacteriuria is not recommended for diabetic women, elderly, institutionalized patients, and catheterized patients while the catheter remains *in situ*
3. Most patients with consistent symptoms and a positive dipstick test can be treated without the need to obtain a urine culture unless any of the factors associated with an upper tract or complicated infection is present
4. Cultures are warranted to identify usual or resistant organisms in women whose symptoms either do not abate or recur within two to four weeks after the completion of treatment
5. Prophylaxis might be warranted for two or more symptomatic infections during one 6-month period or three or more such infections over a 12-month period

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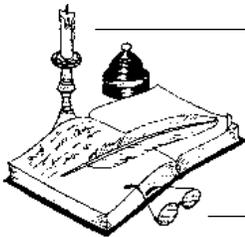*Porpon Rotjanapan, MD, is an Infectious Disease Fellow at the University of Iowa Hospitals and Clinics.*

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Physician's Lexicon

The Wanderings of the Vagus Nerve

The vagus nerve, sometimes called the pneumogastric nerve, is the tenth of twelve paired nerves emanating from the primate brain stem and are collectively called the cranial nerves. It is the longest and most complex of the cranial nerves. It emerges from the medulla oblongata, between the olivary nucleus and the inferior cerebellar peduncle; it then exits the infratentorial space through the jugular foramen, courses caudally through the carotid sheath and finally distributes its roots to structures in the neck, thorax and, via the diaphragm, the abdomen. About 80% of its fibers are sensory but it does innervate numerous muscles including those of the larynx and also carries parasympathetic fibers.

The phrase, vagus nerve, is derived from the Latin, *nervus vagus,* meaning wandering nerve. A number of other English words are also descended from the Latin, *vagus*, all reflecting the sense of wandering or impermanence. Thus, one encounters the word, vagabond, (a person leading a wandering, nomadic life and sometimes thought to be shiftless, irresponsible and without a permanent home); the word, vagrant, (a person with neither home not visible means of support, a wanderer); the word, vague, (something not clearly perceived or understood, something imprecise); and extravagant (spending too much; wandering beyond the bounds of fiscal reason and prudence). And the words reverie and rave are distantly related.

Still further English words trace back to the Latin, *vagus*. A vade mecum (literally, in Latin, "go with me") defines working or instructional manuals in various occupations and avocations (in more modern vernacular, "how-to" books). Even the slang, vamoose (directly from the Spanish, *vamos,* meaning let us go; previously from the Latin, *vadere*, meaning to go, and ultimately from the Latin, *vagus*.)

The word, wander, however, is purely Germanic (through various north Teutonic permutations including Old English) with not a trace of Latin or Greek. The word, vandal, derives from it; and through a reverse linguistic migration then turns up in Latin as *Vandalus*, the name that the Romans bestowed upon the pagan Germanic tribes that ravaged Spain and Gaul particularly during the Fifth Century.

The vagus nerve has been a sturdy mainstay within textbooks on neuroanatomy for centuries. No medical student could possibly consider promotion to the clinical years without knowing the distribution and sensory/motor responsibilities of this important cranial nerve. And now, with the increasing employment of the **vagus nerve stimulator** (**VNS**) as a therapeutic adjunct, the nerve has assumed even greater practical importance.

– STANLEY M. ARONSON, MD