

TEACHABLE
MOMENT

LESS IS MORE

Asymptomatic Bacteriuria,
What Are You Treating?

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Story From the Front Lines

A man in his 80s with a history of interstitial lung disease, deep venous thrombosis treated with warfarin, and chronic venous stasis presented to the emergency department with swelling of his bilateral lower extremities. He had no other symptoms, and his vital signs were normal. As part of the workup, a urinalysis was obtained, the results of which demonstrated pyuria and positive leukocyte esterase. His urine was sent for culture, and in the meantime he was given a dose of ceftriaxone, 1 g intravenously, for a presumed urinary tract infection (UTI). He was subsequently admitted to the hospital for concern of right-sided heart failure complicating his chronic venous stasis.

Teachable Moment

Bacteriuria is defined in men as 10^5 colony-forming units of the same organism isolated on 1 uncontaminated urine sample and in women as 2 samples with the same parameters. In a catheterized individual, it requires only greater than 10^2 colony-forming units.¹ To be classified as asymptomatic bacteriuria (ABU), these requirements must be met in a patient exhibiting no genitourinary symptoms to suggest infection.²

Screening and antimicrobial treatment for ABU has been supported in certain subsets of patients. In pregnant women, for example, screening and treatment reduces the rate of pyelonephritis in the mother as well as preterm delivery and low birth weight.¹ There is also evidence that the risk of bacteremia is reduced by screening and treating ABU in patients undergoing urologic procedures with mucosal disruption, including transurethral resection of the prostate.¹

However, studies performed in other adult populations, including nonpregnant women, elderly men and women, and institutionalized patients with and without indwelling urinary catheters consistently demonstrate no benefit from antibiotic treatment of ABU and sometimes demonstrate harm.² In a recent systematic review by Dull et al,³ patients with ABU had a slight increased risk of symptomatic urinary infection compared with nonbacteriuric controls, but treatment of the asymptomatic colonization did not reduce their risk of subsequent symptomatic infection. Furthermore, there was no association of ABU with increased kidney dysfunction, hypertension, genitourinary malignant neoplasm, or mortality.³

The increased risk of symptomatic UTI in patients with ABU was likely due to host factors that support bacterial colonization rather than the current strain becoming virulent. Intervening in such cases with antibiotics only increases the risk of progressively resistant infections with no observable clinical benefit.³ A recent prospective randomized clinical trial⁴ that studied sexually active young women indicated that the presence of ABU may even be protective. The study found that colonization by the less virulent strains that tend to cause ABU can decrease the incidence of infection by more virulent strains via bacterial interference.⁴ The systematic review by Dull et al³ found a number needed to harm of 2 to 10, with adverse effects from antibiotics including increased bacterial resistance, superinfections, and *Clostridium difficile*-related complications.

Despite this compelling evidence and the guidelines from organizations including the Infectious Diseases Society of America and US Preventive Services Task Force recommending against screening or treatment of ABU—except in pregnant women and prior to urologic procedures—it remains a significant problem.^{1,5} Recent data show that 26% to 68% of patients with ABU are inappropriately treated with antimicrobial therapy.³ Considering that UTIs are cited as the most common indication for antibiotics in the hospital and the high prevalence of ABU misdiagnosed as UTI, this has a clinically significant impact not only on the individual patient but on widespread antimicrobial resistance and rising health care costs.

In the face of such clear guidance to the contrary, and a general push toward improved antibiotic stewardship, it is worth considering why so many patients with ABU receive antibiotics. It is possible that clinicians fear potential adverse effects of not treating bacteriuria more than the known risks of antibiotics. Knowledge gaps regarding pretest probability of infectious syndromes and laboratory interpretation is likely also a factor considering that UTIs are often diagnosed in the context of vague symptoms accompanying pyuria.

Our patient had no symptoms to suggest UTI and was not in a high-risk category necessitating intervention for his asymptomatic bacteriuria. In receiving ceftriaxone, the patient stood little to no chance of benefit but was exposed to greater risk for antimicrobial resistance, superinfection, and other complications from antibiotics.

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Published Online: January 12, 2015.
doi:10.1001/jamainternmed.2014.7132.

Conflict of Interest Disclosures: None reported.

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