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Declarations

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Chronic Urinary Tract Infection Due to *Burkholderia cenocepacia* in an Immunocompetent Adult Without Risk Factors: A Rare Case Report

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ABSTRACT

Background: *Burkholderia cenocepacia*, a multidrug-resistant member of the *Burkholderia cepacia* complex, is an opportunistic pathogen rarely implicated in urinary tract infections (UTIs). Reported cases almost exclusively involve immunocompromised or catheterized patients. Its isolation from the urinary tract of an immunocompetent host without predisposing factors is exceptionally uncommon. **Objective:** To describe a culture-confirmed chronic UTI caused by *B. cenocepacia* in a healthy adult, emphasizing diagnostic accuracy, antimicrobial resistance, and treatment outcome. **Methods:** A 32-year-old male with six-month lower urinary tract symptoms underwent urinalysis, culture, and imaging. Organism identification was confirmed by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) and repeat culture after 14 days. Antimicrobial susceptibility was determined by Kirby–Bauer disc diffusion interpreted per Clinical and Laboratory Standards Institute 2024 criteria. **Results:** Urine culture repeatedly yielded *B. cenocepacia* $>10^5$ CFU/mL, resistant to aminoglycosides, fluoroquinolones, and β -lactams but susceptible to trimethoprim–sulfamethoxazole (TMP–SMX) and meropenem. Initial 14-day TMP–SMX therapy led to transient improvement followed by relapse; a subsequent six-week course achieved complete clinical and microbiological resolution. Follow-up cultures at one and three months were sterile. **Conclusion:** *B. cenocepacia* can cause chronic, community-acquired UTI in immunocompetent adults. Accurate identification using MALDI-TOF MS and prolonged, susceptibility-guided TMP–SMX therapy are essential for eradication and prevention of recurrence.

Keywords*Burkholderia cenocepacia*; urinary tract infection; chronic UTI; multidrug resistance; MALDI-TOF MS; trimethoprim–sulfamethoxazole.

INTRODUCTION

The *Burkholderia cepacia* complex (BCC) comprises over 20 closely related species of aerobic, non-fermenting Gram-negative bacilli that inhabit soil, water, and moist hospital environments (1). Among these, *Burkholderia cenocepacia* is particularly notable for its virulence and intrinsic multidrug resistance, frequently associated with pulmonary infections in cystic fibrosis patients and opportunistic bacteremia in immunocompromised hosts (2,3). Its pathogenicity arises from multiple virulence determinants, including quorum-sensing systems, biofilm formation, and efflux-mediated antimicrobial resistance, rendering treatment challenging even with broad-spectrum agents (4,5).

While *B. cenocepacia* is well recognized in nosocomial and respiratory settings, urinary tract involvement remains exceedingly uncommon and is typically confined to patients with predisposing factors such as catheterization, prior urologic intervention, or immunosuppression (6,7). Fewer than ten cases have been documented in immunocompetent individuals worldwide, emphasizing its rarity and diagnostic difficulty (8).

This report describes a chronic, community-acquired urinary tract infection caused by *B. cenocepacia* in a healthy adult male without known risk factors, highlighting diagnostic challenges, antimicrobial resistance profile, and successful eradication following targeted therapy.

CASE PRESENTATION

A 32-year-old previously healthy male presented to the urology outpatient department with a six-month history of intermittent dysuria, urinary frequency, and suprapubic discomfort. Dysuria was described as a burning sensation scoring 6/10 on a numerical pain scale, typically occurring during micturition and resolving spontaneously thereafter. Urinary frequency averaged 10–12 voids per day, with preserved nocturnal control. There was no history of fever, chills, flank pain, hematuria, urinary incontinence, or urethral discharge, suggesting a localized lower urinary tract process. The patient denied any history of diabetes mellitus, hypertension, chronic kidney disease, or immunodeficiency. He had no prior hospital admissions, urinary catheterization, or urological procedures. There was no history of prolonged antibiotic use, genitourinary trauma, or sexual contact during the preceding year. No family history of urinary or metabolic disorders was reported. On physical examination, the patient was afebrile and hemodynamically stable. The abdomen was soft, non-tender, and without suprapubic distension. Digital rectal examination revealed a normal-sized, non-tender prostate with preserved median sulcus, excluding prostatitis as a contributing factor.

Table 1. Summary of Laboratory, Microbiological, and Treatment Findings

Parameter	Finding	Interpretation / Comment
Age / Sex	32 years / Male	Immunocompetent adult
Symptoms	Dysuria, urinary frequency, suprapubic pain (6 months)	Chronic lower urinary tract infection
Comorbidities / Risk Factors	None	No immunosuppression or catheterization
Urinalysis	Pyuria (15–20 WBC/hpf), no hematuria	Active lower urinary tract inflammation
Renal Function Tests	Within normal limits	No renal involvement
Imaging (Ultrasound KUB)	Normal kidneys and bladder; no post-void residual	Excludes structural cause
Urine Culture (×2)	<i>Burkholderia cenocepacia</i> (>10 ⁵ CFU/mL)	Confirmed on repeat culture
Identification Method	MALDI-TOF MS	Accurate species-level confirmation
Antibiotic Susceptibility (CLSI 2024)	Sensitive: TMP–SMX, Meropenem Resistant: Aminoglycosides, Fluoroquinolones, β-lactams	Multidrug-resistant isolate
Initial Therapy	TMP–SMX 160/800 mg BD × 14 days	Partial response, relapse at 4 weeks
Definitive Therapy	TMP–SMX 160/800 mg BD × 6 weeks	Complete resolution
Follow-up Cultures	Sterile at 1 and 3 months	Microbiological eradication
Outcome	Asymptomatic at 3-month follow-up	Successful long-term clearance

LABORATORY AND IMAGING FINDINGS

Initial urinalysis revealed pyuria with 15–20 white blood cells per high-power field (WBC/hpf) and 0–2 red blood cells per high-power field (RBC/hpf), without casts or crystals. There was no proteinuria or hematuria. Complete blood count and renal function tests were within normal limits (serum creatinine 0.9 mg/dL, urea 25 mg/dL). A midstream urine culture, performed using standard aseptic technique, yielded growth of *Burkholderia cenocepacia* at >10⁵ colony-forming units per millilitre (CFU/mL) after 48 hours of incubation at 37°C on MacConkey agar. Identification was confirmed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) (Bruker Daltonics, Germany), and verified on repeat culture performed 14 days later, which demonstrated the same organism with an identical antibiogram. Antimicrobial susceptibility testing was conducted by the Kirby–Bauer disc diffusion method, interpreted according to the Clinical and Laboratory Standards Institute (CLSI) 2024 guidelines (9). The isolate was resistant to aminoglycosides (gentamicin, amikacin), fluoroquinolones (ciprofloxacin, levofloxacin), and most β-lactams (ceftriaxone, ceftazidime, piperacillin-tazobactam), but retained susceptibility to trimethoprim–sulfamethoxazole (TMP–SMX) and meropenem. Renal ultrasonography demonstrated normal cortical echotexture of both kidneys, no hydronephrosis or perinephric changes, and an empty post-void residual bladder. The prostate was normal in size and echogenicity, with no evidence of prostatitis or abscess.

TREATMENT COURSE

Based on the culture and sensitivity results, the patient was initially prescribed oral trimethoprim–sulfamethoxazole (160/800 mg twice daily) for 14 days. He reported marked symptomatic improvement by day 10, but dysuria and frequency recurred within four weeks after completion of therapy. A repeat urine culture again yielded *B. cenocepacia* with the same sensitivity pattern, confirming persistence rather than reinfection. Given the organism’s biofilm-forming potential and the known propensity of *B. cenocepacia* for chronic infection, a prolonged six-week course of TMP–SMX was commenced. The choice of TMP–SMX was based on its reliable oral bioavailability, intracellular penetration, and proven efficacy as monotherapy for susceptible *B. cepacia* complex infections (10). The patient was advised to maintain high fluid intake (≥2.5 L/day), avoid urinary retention, and adhere strictly to treatment duration.

Follow-up urine cultures were obtained at one month and three months post-treatment. Both were sterile, indicating microbiological clearance. The patient reported complete symptom resolution and no adverse drug effects throughout the treatment period.

Outcome and Follow-up At the three-month follow-up, the patient remained asymptomatic with no recurrence of dysuria, urgency, or frequency. Physical examination and urinalysis were unremarkable. Repeat renal function tests were normal, and surveillance urine culture remained sterile. The overall clinical course confirmed successful eradication of chronic *B. cenocepacia* infection following extended TMP–SMX therapy guided by culture-based susceptibility.

DISCUSSION

Urinary tract infection (UTI) caused by *Burkholderia cenocepacia* is an uncommon clinical entity. The organism, a member of the *Burkholderia cepacia* complex (BCC), is notorious for its environmental persistence, intrinsic multidrug resistance, and pathogenic potential in vulnerable hosts such as patients with cystic fibrosis or chronic granulomatous disease (1,2). However, community-acquired infections in immunocompetent individuals are exceedingly rare, particularly when confined to the urinary tract. The present case demonstrates that *B. cenocepacia* can establish chronic lower urinary tract infection in the absence of classical risk factors such as catheterization, prior instrumentation, or immune compromise.

EPIDEMIOLOGY AND REPORTED CASES

A limited number of *B. cenocepacia* UTI cases have been reported globally, with the majority occurring in hospitalized or immunocompromised patients (3,4). Chiarini *et al.* (5) described BCC infections predominantly in individuals with prolonged hospitalization or invasive devices, whereas Ganesan *et al.* (6) documented urinary isolates in catheterized patients with multiple comorbidities. In contrast, our patient had no prior healthcare exposure, suggesting community acquisition—an observation consistent with sporadic environmental cases reported from contaminated irrigation solutions and antiseptic fluids (7).

PATHOGENESIS AND MECHANISTIC CONSIDERATIONS

The capacity of *B. cenocepacia* to persist within the urinary tract likely stems from its extensive virulence arsenal. The organism forms robust biofilms, facilitating adherence to uroepithelial surfaces and protecting against host immune clearance (8). Quorum-sensing systems (CepIR and CciIR) regulate virulence gene expression, promoting chronic colonization and resistance to oxidative stress (9). Additionally, efflux pumps such

as RND-3 and RND-4 and β -lactamase enzymes contribute to its broad intrinsic resistance (10,11). These mechanisms collectively explain the chronicity and relapse observed after short-course antibiotic therapy.

DIAGNOSTIC AND MICROBIOLOGICAL CHALLENGES

Accurate identification of BCC species remains technically challenging. Automated biochemical systems frequently misclassify *B. cenocepacia* as *Pseudomonas aeruginosa* or *Stenotrophomonas maltophilia*, leading to inappropriate therapy (12). The use of matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS), as employed in this case, provides reliable species-level differentiation and is recommended for atypical Gram-negative isolates showing multidrug resistance profiles inconsistent with Enterobacterales (13). Repeat culture confirmation remains essential to distinguish persistence from reinfection.

THERAPEUTIC IMPLICATIONS

Treatment of *B. cenocepacia* infections is complicated by its intrinsic resistance to aminoglycosides, polymyxins, and most β -lactams (14). Trimethoprim–sulfamethoxazole (TMP–SMX) remains the cornerstone therapy for susceptible strains due to its bactericidal activity through inhibition of folate synthesis, high oral bioavailability, and penetration into intracellular and biofilm environments (15). In our patient, recurrence following a 14-day regimen and successful eradication after a 6-week course underscore the importance of prolonged, culture-guided therapy in biofilm-associated infections. Carbapenems such as meropenem may be considered for severe systemic infections, but monotherapy is often inadequate due to inducible β -lactamases and efflux activity (10,14). Combination therapy, including TMP–SMX with meropenem or ceftazidime, has shown variable success in refractory cases (16). The present case illustrates that oral TMP–SMX monotherapy, when extended adequately, can achieve durable microbiological clearance in localized infection without systemic spread.

CLINICAL AND INFECTION-CONTROL SIGNIFICANCE

Although the infection appeared sporadic, the environmental ubiquity of *B. cenocepacia* raises concern for potential contamination sources within community water systems or disinfectants (7). Its ability to survive in chlorinated water and antiseptic solutions poses challenges for infection control, even outside hospital settings. Clinicians should therefore suspect *B. cenocepacia* in culture-positive UTIs unresponsive to empirical fluoroquinolones or β -lactams, particularly when the patient has no identifiable risk factors.

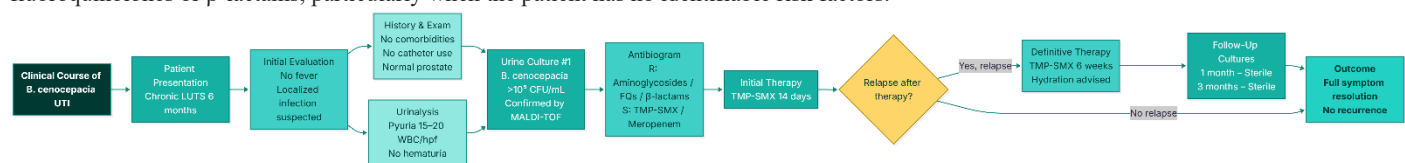


Figure 1 Clinical Course of *B. cenocepacia* UTI

CONCLUSION

This case confirms that *Burkholderia cenocepacia* is capable of producing chronic, community-acquired urinary tract infection even in immunocompetent adults. Persistent symptoms despite standard therapy should prompt culture-based evaluation for atypical, multidrug-resistant pathogens. Reliable species-level identification by MALDI-TOF MS and extended, susceptibility-directed treatment with trimethoprim–sulfamethoxazole can achieve durable microbiological cure while minimizing unnecessary broad-spectrum antibiotic exposure.

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