JK SCIENCE pISSN: 0972-1177

CASE REPORT

Colonizer or Culprit? Repeated Isolation of *Trichosporon asahii* in an Immunocompromised Patient with UTI – Diagnostic Challenges in Distinguishing Colonization from Infection: A Case Report

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Abstract

Trichosporon asahii, an emerging opportunistic pathogen in immunocompromised, catheterized patients, rarely causes urinary tract infections (UTI), accounting for only 0.2% of fungal UTIs. We report a catheter-associated-UTI in a 77-year-old male with diabetes and stage IV chronic-kidney-disease who developed fever and pyuria on day eight of hospitalization. Urine culture isolated *T. asahii* resistant to fluconazole, amphotericin B, and echinocandins but susceptible to voriconazole, with clinical improvement, highlighting diagnostic challenges in distinguishing colonization from true infection.

Key Words

Trichosporon asahii UTI, Fungal UTI, Nosocomial UTI

Introduction

Trichosporon asahii, a yeast-like fungus found in the environment and human microbiota, usually causes superficial infections like white Piedra, but can act as an opportunistic pathogen in immunocompromised individuals. Though increasingly linked to systemic infections, urinary tract infections are extremely rare, accounting for only about 0.2% of fungal UTIs.[1]

While fungal UTIs are usually caused by *Candida* species in hospitalized, catheterized, or immunosuppressed patients, non-*Candida* fungi like *Trichosporon* can also cause significant morbidity.[2] *Trichosporon asahii*, the most pathogenic of six human-infecting species, causes UTIs with >75% mortality in immunocompromised patients, especially those with diabetes, prior antimicrobial use, chronic catheterization, or immunosuppression.

Diagnosis of *T. asahii* is challenging due to frequent misidentification as *Candida* and its intrinsic resistance to echinocandins with variable azole susceptibility, making timely identification and targeted therapy critical to avoid poor outcomes.[3]

Distinguishing colonization from true infection is crucial, requiring careful evaluation of clinical context and risk factors. Here, we present a rare case of *T. asahii* UTI in an immunocompromised patient, highlighting diagnostic challenges, treatment complexities, and the need for greater clinical awareness of this emerging fungal

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Manuscript Received: 14.10.2025; **Revision Accepted**: 25.12.2025
Published Online First: 10th April, 2026
Open Access at: https://journal.jkscience.org

Cite this article as: Bhaumik N, Balaji L, Subbalakshmi E. Colonizer or Culprit? Repeated Isolation of *Trichosporon asahii* in an Immunocompromised Patient with UTI – Diagnostic Challenges in Distinguishing Colonization from Infection: A Case Report. JK Science 2026; 8(2):118-20.

pathogen.

Case Report

A 77-year-old male with bilateral proximal ureteric calculi causing obstructive nephropathy, bilateral hydroureteronephrosis, stage V chronic kidney disease, and type 2 diabetes mellitus presented with severe abdominal pain, hypotension, and altered sensorium. On admission, his Glasgow Coma Scale was 7/15, and he was in shock, requiring intensive care unit admission.

The patient was intubated, catheterized, and initiated on empirical intravenous meropenem (1 g twice daily) for suspected urosepsis in the setting of obstructive uropathy. Empirical fluconazole (400 mg once daily) was added due to high risk for fungal infection associated with advanced CKD, diabetes, critical illness, and indwelling urinary catheterization. Bicarbonate infusion was started for severe metabolic acidosis.

Computed tomography of the abdomen demonstrated bilateral proximal ureteric calculi with gross hydronephrosis and inflammatory changes. Laboratory investigations revealed leukocytosis (12,400 cells/mm^3), severe anemia (hemoglobin 6.3 g/dL), renal dysfunction (creatinine 6.0 mg/dL; urea 191 mg/dL), hyponatremia (124 mEq/L), hyperkalemia (6.7 mEq/L), and severe metabolic acidosis (bicarbonate 9.7 mEq/L). Hemodialysis and blood transfusions were initiated.

Initial blood and urine cultures were sterile, and the patient showed clinical improvement during the first week of hospitalization. On day eight, he developed new-onset fever (100°F) with chills despite ongoing broad-spectrum antibacterial therapy. Blood and endotracheal aspirate cultures remained sterile, and the absence of an alternative infective focus raised suspicion of a fungal urinary tract infection.

Urinalysis revealed significant pyuria (>25 white blood cells/high-power field), leukocyte esterase positivity, and yeast cells. Gram staining showed numerous pus cells with budding yeast cells exhibiting pseudohyphae and barrel-shaped arthroconidia (Figure 1). Urine culture on cystine lactose electrolyte-deficient agar yielded small, dry, wrinkled colonies (Figure 2). The isolate was urease positive and identified as *Trichosporon asahii* with a colony count of 10^6 CFU/mL using the VITEK® 2 Compact system.

To rule out colonization, the urinary catheter was removed, and a repeat urine sample was collected, which again yielded *T. asahii* at $\geq 10^6$ CFU/mL, confirming true infection. Antifungal susceptibility testing showed

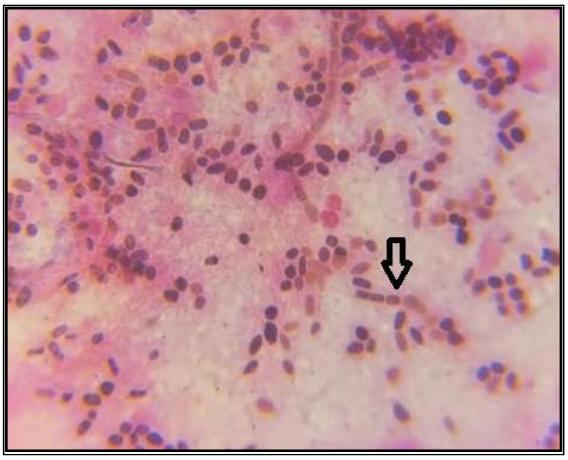

Figure 1

Figure 2

resistance to fluconazole, amphotericin B, and echinocandins, with susceptibility to voriconazole. Intravenous voriconazole (400 mg/day) was initiated and continued for 10 days.

The patient demonstrated steady clinical improvement over the next 10 days, with resolution of fever and normalization of inflammatory markers. Follow-up urine cultures were sterile, and he was transferred to the urology ward for further management.

Discussion

Trichosporon asahii is an emerging opportunistic yeast increasingly associated with healthcare-associated infections, particularly in patients with advanced CKD,

diabetes, prolonged ICU stays, or indwelling catheters.[4]While the organism is frequently isolated from urine, it rarely causes true urinary tract infection, representing nearly 0.2% of fungal UTIs.[1] The differential diagnosis included *Candida* UTIs and fungal colonization. *Candida* species were considered likely due to diabetes and catheterization; however, the gram staining demonstrated budding yeast cells with pseudohyphae and characteristic barrel-shaped arthroconidia, favoring *Trichosporon* species over *Candida*species. Colonization was considered but deemed unlikely due to the presence of fever, significant pyuria, repeated isolation of the same organism at high colony counts (10^6 CFU/mL), and clinical response to targeted antifungal therapy, favouring true infection.[5]

Standardized diagnostic criteria for trichosporonuria are lacking; therefore, diagnosis relies on correlating microbiological findings with host risk factors and clinical features. The resistance profile observed in this case is consistent with emerging trends, as *T. asahii* commonly exhibits resistance to amphotericin B and echinocandins. Voriconazole remains the preferred agent and was effective in achieving clinical and microbiological cure.[6]

Conclusion

Isolation of *Trichosporon asahii* from urine represents a diagnostic dilemma. Repeated high-count isolation with supportive clinical and laboratory evidence confirmed true infection in this case. Early recognition and prompt initiation of targeted antifungal therapy are crucial to prevent progression to invasive disease.

Consent Statement

Written informed consent was obtained from the patient's legally authorized representative for publication of this case report. Patient confidentiality has been maintained.

Financial Support and Sponsorship : Nil
Conflict of Interest: Nil

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