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Short Communication

Trichosporon asahii urinary tract infection in immunocompetent patients



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ABSTRACT

Trichosporonosis is an emerging infection predominantly caused by *Trichosporon asahii* which is a ubiquitous and exclusively anamorphic yeast. T. asahii urinary tract infection is rare and remains scantily reported.

T. asahii was isolated from urine of two immunocompetent patients who were receiving in-patient treatment for multiple comorbidities. T. asahii was identified phenotypically by a combination of manual and automated systems. Antifungal susceptibility done by E-test revealed multiresistance with preserved susceptibility to voriconazole.

The ubiquity and biofilm formationposes difficulty in establishing pathogenicity and delineating environmental or nosocomial infections. Risk factors such as prolonged multiple antimicrobials, indwelling catheter and comorbidities such as anemia and hypoalbuminemia may be contributory to the establishment of a nosocomial opportunistic *T. asahii* infection. Dedicated efforts targeted at infection control are needed to optimize management and control of Trichosporon infections.

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Introduction

Trichosporonosis is an emerging infection predominantly caused by Trichosporon asahii. Trichosporon (Beigel, 1985) species are ubiquitous, exclusively anamorphic, yeast like fungi belonging to Trichosporonaceae. Trichosporon is implicated in superficial and mucosal infections, however, systemic infections are known in immunocompromised,

cancer, burns, transplant patients as well as patients on steroids, peritoneal dialysis, prolonged mechanical ventilation and those undergoing prosthetic valve surgeries.^{1,2} Alimentary tract, respiratory tract, broken skin and mucosa are possible portals of entry. Persistent and disseminated infections have poor prognosis. Fatal outbreak in neonates and breakthrough trichosporonosis under antifungal therapy has been reported.^{3,4}T. asahii urinary tract infection (UTI) is scantily reported.^{5,6} We report two cases of *T. asahii* UTI in

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Fig. 1 – Trichosporon asahii on CLED agar after overnight incubation.

immunocompetent severely ill patients in a tertiary care set up.

Case 1

T. asahii was isolated from urine on two occasions from a 46 year old male patient with multiple pressure sores over sacrum and both hips. He was offered skin flap and tissue expansion. There was a past history of traumatic quadriplegia and multiple fractures following fall from height. The patient was having persistent fever, hypoalbuminemia, microcytic hypochromic anemia, persistent leukocytosis, pyuria and deteriorated further. During the course of stay in the hospital, the patient had an indwelling catheter and was aggressively managed with meropenem, levofloxacin, Co-Amoxiclav, tobramycin and linezolid in appropriate dosages along with high protein diet, albumin infusions, antacids, multivitamins, enoxaparin and whole blood transfusion.

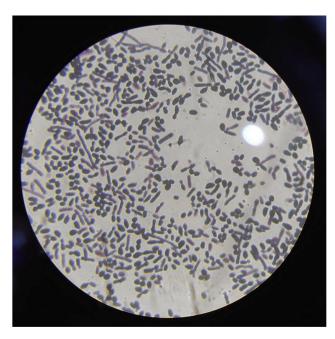


Fig. 2 – Gram stain of Trichosporon asahii showing budding yeasts and barrel shaped arthroconidia.

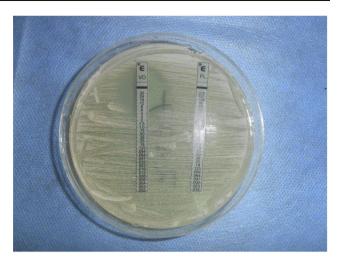


Fig. 3 – Antifungal susceptibility of Trichosporon asahii through E-test showing an MIC of 3 μ g/ml.

The first sample was collected by clamping the catheter while the second after catheter removal. On both occasions, routine microscopy revealed pus cells and yeast cells without budding. Urine sample inoculated with standard loop on cysteine lactose electrolyte deficient agar (HiMedia Laboratories, India) revealed significant growth of dry creamy white colonies after overnight incubation (Fig. 1). Gram stain and lactophenol cotton blue mount revealed septate hyaline hyphae with arthroconidia and few budding yeast cells (Fig. 2). After subculture, the yeast was identified to be T. asahii by morphology on cornmeal agar, Gram stain, hydrolysis of urea, assimilation of carbon/nitrogen compounds and confirmation by VITEK 2 compact automated system (bioMérieux, France).5 Subcultures on Sabouraud's dextrose agar at both 22 °C and 37 °C revealed curdy white growth which turned cottony on subsequent incubation. Antifungal susceptibility was tested by E-test (AB BIODISK, Sweden) for fluconazole, itraconazole, voriconazole, Amphotericin B and anidulafungin (Fig. 3). Minimal inhibitory concentration (MIC) for voriconazole was 3 µg/ml and multiresistance was observed. Prompt treatment with voriconazole and management for comorbidities lead to subsequently sterile urine cultures.

Case 2

T. asahii was isolated twice from urine samples of an 86 year old male patient admitted for generalized tonic clonic seizures, associated loss of consciousness and sphincter incontinence. The patient had comorbid hypothyroidism, hypertension, dyslipidemia, cholelithiasis, glaucoma in left eye and sinus bradycardia with first degree atrioventricular block. Pus cells, occasional yeast cells without budding in urine were seen. Creatinine levels were high. Leukocyte counts and other investigations were normal. The patient was being managed by clindamycin, cefoperazone, phenytoin sodium, levothyroxine, amlodipine, aspirin and antacids. The patient had a condom catheter throughout the course of his stay in the hospital. Pure significant creamy white colonies revealed

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