

Urinary Tract Infection Due to *Trichosporon asahii* in a Diabetic Patient

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Abstract

Trichosporon asahii is a basidiomycetous yeast which causes white piedra and onychomycosis in immunocompetent host and disseminated infections in immunodeficient hosts. Urinary tract infection by this fungus though rare, have been reported earlier. We report a case of urinary tract infection of a 46 year old male diabetic patient. Yeast like organisms was isolated from urine samples on three consecutive days, in pure forms. The patient responded well to antifungal treatment. To the best of knowledge, this is the first report of urinary tract infection caused by *Trichosporon asahii* from Mumbai.

Introduction

Trichosporon is an anamorphic yeast, with distinct morphological characters of budding cells and true mycelium that disarticulate to form arthroconidia.¹ It is present in external environment and is usually isolated from soil. It may also be present in water, air or organic substrate.²

In the past, a sole species *Trichosporon beigelli* (*Trichosporon cutaneum*) was correlated to human pathology. It was recognized as the cause of superficial fungal infections as white piedra, tinea cruris and onychomycosis. Genus *Trichosporon* has recently gone taxonomic re-evaluation.³ The genus is divided into six species of which *Trichosporon asahii* causes the most infections. Disseminated infection is uncommon but increasingly reported in immunocompromised hosts. It may produce disseminated infections in immunocompromised hosts.⁴ Urinary tract infection by the fungus is reported from a seven month

old child, one month after posterior urethral valve surgery.⁵ We are reporting here, urinary tract infection by this fungus in a diabetic patient.

Case Report

A 46 year old male patient presented with burning micturition since one month, mild fever with chills. As per his previous medical records, he was a known diabetic. On examination the patient was febrile (100°F). His Hb was 12 g/dl, TLC 9200/ml with 80% neutrophils and 20% lymphocytes. His fasting and postprandial blood sugar was 180 g/dl and 240 mg/dl respectively. His midstream urine sample was collected in a sterile container. Microscopic examination revealed presence of pus cells and budding yeast cells. The sample was inoculated with a standard loop which delivers a drop of 0.001 ml of urine on Blood agar and MacConkey agar and incubated overnight. Tiny, creamy white, dry wrinkled colonies were observed on Blood agar, the next day. More than 100 colonies were observed on Blood agar which corresponds to 10⁵ colony forming units (CFU/ml). This is a significant count in urine. The Gram stained smear of colony from blood agar revealed septate hyphae with arthrospores and budding yeast cells (Fig. 1). The colony was subcultured on Sabouraud's Dextrose agar (SDA), which was incubated at 37°C. Numerous pasty creamy colonies appeared on SDA after 24 hours of incubation. Two more early morning samples were analyzed which showed similar findings.

The yeast was identified with cornmeal agar

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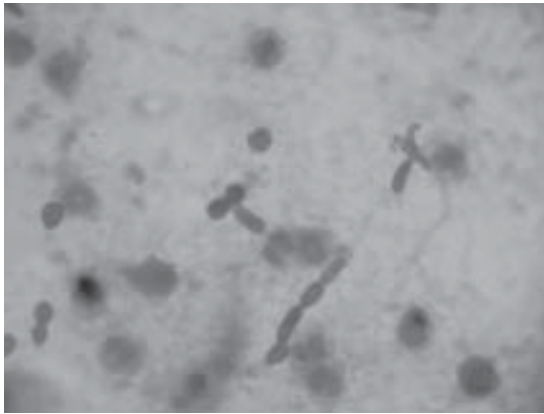


Fig. 1 : Gram stained smear from colony on Blood agar showing septate hyphae with arthroconidia (X 1000) and few spores.

morphology, urea hydrolysis of carbohydrate assimilation tests. Depending upon the morphology, cultural and biochemical properties, the isolate was identified as *Trichosporon asahii*. The patient was started on oral fluconazole 100 mg/day for 7 days to which the patient responded.

Discussion

The increase in profoundly immunocompromised patients has been accompanied by an increase not only in frequency of opportunistic fungal infections but also in the variety of species involved.¹⁻⁵

Trichosporon species is one of the emerging mycosis in neutropenic patients, usually in haematological malignancies. It may cause disseminated infection in immunocompromised patients. Clinically trichosporonosis appears with fever, pulmonary infiltrates, azotaemia, renal dysfunction, skin lesions.² The yeast can be isolated from the sputum, urine, skin and blood.¹⁻⁶

Isolation of the same yeast in three consecutive urine samples in significant counts and the finding that no bacteria was isolated, establishes *Trichosporon asahii* as an aetiological agent of urinary tract infection. The fact that there was clearance

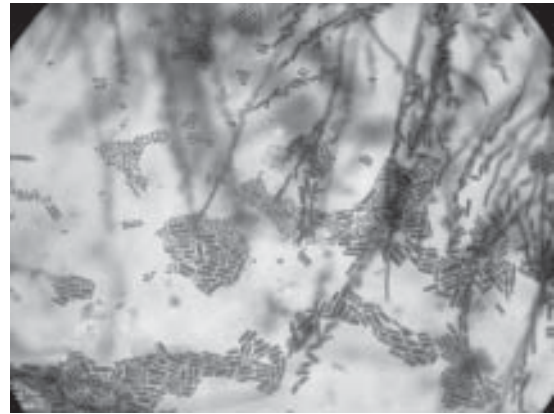


Fig. 2 : Cornmeal agar (LPCB) after 48 hours of incubation (X 400) demonstrating rectangular arthroconidia.

of the organism from the urinary tract with recovery of the patient following antifungal treatment strongly associates the yeast as causative agent of UTI. Though reported rarely *Trichosporon asahii* is a known pathogen to cause UTI. It has recently been reported to cause UTI in seven month old child following urethral valve surgery from India.⁵

Factors that enhance the mucosal colonization and subsequent invasion of the fungus include broad spectrum antibiotic treatment of break in mucosal barrier.⁷ Other yeasts as *Candida species* is well known in diabetic patients.⁴ Similarly it serves as the predisposing factor in this patient.

Trichosporon species are occasionally a part of normal flora of human skin. In fact this yeast has been documented on intact perigenital skin in 12-4% of population in one study.² Probably this is the source of infection in patients to cause UTI. Nosocomial UTI due to *Trichosporon asahii* has been reported from Chile.⁸

Trichosporonosis is usually an insidious disease but it can present as an opportunistic infection in susceptible hosts. Its diagnosis is likely to be missed particularly in developing

countries because of a general lack of awareness and lack of diagnostic features of the aetiological agent.

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CAN LOW-DOSE RADIATION INCREASE RISK OF CARDIOVASCULAR DISEASE?

Risk of heart disease increases after high-dose therapeutic radiation (typically > 30-40 Gy), such as that received for treatment of Hodgkin's lymphoma and breast cancer.

Assessment of cardiovascular disease risk from radiation doses much lower than 0.5 Gy is challenging. Data for survivors of the atomic bomb have substantial uncertainty about the cardiovascular disease risk at doses in this range.

The absence of an established biological model of cardiovascular effects at low doses hampers the acceptance of a causal association, but there are plausible mechanisms.

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