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Case Report

A rare case of recurrent urinary tract infection due to *Trichosporon* species in an immune-competent diabetic female patient

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Abstract

Trichosporonosis is a disease caused by *Trichosporon* spp. which are ubiquitous anamorphic yeast that commonly inhabit the soil. In human they are found in the skin, gastrointestinal tract and respiratory tract. Globally, *Trichosporon* spp. infection is rare and remains scantily reported in urinary tract infections and disseminated invasive infection amongst immunocompromised and cancer patients with neutropenia. *Trichosporon* asahii is the most commonly reported species. Virulence factors like proteinases, lipases, and phospholipases may be responsible for disease manifestation. We report a case of recurrent urinary tract infection due to *Trichosporon* spp. in a 62-year-old immunocompetent diabetic female which remained misdiagnosed for a long period of time. The patient was subsequently treated successfully by oral fluconazole drug.

Introduction

Trichosporon spp. are basidiomycetous yeast-like anamorphic organisms mainly found in soil and other environmental sources of tropical and temperate areas. In humans, these are rarely found in gastrointestinal tract, oral cavity, respiratory tract and skin surfaces [1]. The burden of *Trichosporon* spp. infection mainly represent the second or third most common non-Candida yeast infections causing invasive disease in patients with cancer [2]. Their pathogenicity is due to the presence of different virulence factors like production of enzymes (Proteases and phospholipases), presence of glucuronoxylomannan in cell wall (that protects from phagocytic activity) and biofilm formation (that protects from antifungal drugs and host immune responses) [1].

Trichosporon asahii (*T. asahii*) infection is found in different clinical conditions like nongranulocytopenia [3], nosocomial urinary tract infection [4], liver-kidney transplantation [5], acute myeloid leukemia [6], diabetes [7], as well as both immunocompromised [8] and immunocompetent [9], individuals of different age groups and gender. From the past 10-15 years, *Trichosporon asahii* has become an emerging pathogen mainly in cancer patients. However, *Trichosporon* spp. may also cause life-threatening sepsis in non-cancer patients and in disseminated infection it shows variety of clinical features like septic shock, pneumonia, renal failure, chorio-retinitis, and cutaneous lesions [10].

Clinical Case

A 62 year old woman visited hospital with the complaint of burning micturition and pain in the lower abdomen. She had received symptomatic treatment for urinary tract infection with antibacterial drugs several times in the recent past but the problem still persisted. She had the history of diabetes and hypertension. The blood pressure was under control (systolic pressure – 135mmHg and diastolic pressure – 90mmHg). Her blood parameters were as follows: serum urea = 42mg/dl, serum creatinine = 0.9mg/dl, fasting glucose = 162mg/dl and post prandial glucose = 230mg/dl, hemoglobin = 13.4g/dl, total leukocyte count = 9,100/µl. Urine microscopy revealed moderate number of pus cells and few budding yeast cells under high power field (40X objective). The patient's urine

sample was sent for routine culture and sensitivity testing. The sample was inoculated with a standard loop on cysteine lactose electrolyte deficient (CLED) agar media (Hi-media, India) and incubated at 37°C overnight. The next day growth of pure colonies was observed. The colonies were 2-3mm in diameter, creamy, dry with wrinkled margins, raised with depressed centers and having colony forming unit $>10^5$ per milliliter (CFU/mL) (Figure 1). Gram stain was performed (Figure 2) which revealed budding yeasts and hyphae which were septate having arthospores. Repeat urine sample was inoculated on Sabouraud's dextrose agar (Hi-media, India) slants at 28°C and 37°C. Pure growth of yeast-like fungi was observed at both temperatures. Urease test was performed and the fungus was found to hydrolyze urea. Further tests for confirmation of spp. was not possible in the setting. Antifungal therapy with oral fluconazole was given to the patient, and urine sample for culture after treatment was found to be negative for the fungus after one week of treatment.

Discussion

Trichosporon spp. are widely distributed in nature, and are the *emerging etiological agents of disseminated* Trichosporonosis. Seven species including *T. asahii, T. asteroides, T. cutaneum, T. inkin, T. mucoides, T. ovoides* and *T. loubieri* are pathogenic to human[11]. *T asahii* is also well known to be responsible for causing white piedra and onychomycosis in immunocompetent patients [10]. Silvestre, et al. found that Trichosporon spp. colonized in 11.1% (112 out of 1,004) of male healthy volunteers on their normal perigenital skin [12]. Treviño, et al. in their report have isolated *T. asahii* from 32 hospitalized patients in 2 years of study [13]. Mattede, et al. conducted a study on intensive care unit admitted patients in Brazil and found high prevalence rate i.e. 65% and mortality rate i.e. 20% [14] of *T. asahii* infection. Mortality rate has been found high in cancer patients which accounts to 80% among patients receiving amphotericin B therapy for treatment [15]. Early diagnosis and treatment of this kind of infection with appropriate antifungal agents may reduce the mortality rate. In the present case, the patient showed good response to fluconazole therapy. Other studies have also shown similar findings in invasive pulmonary infection [16], invasive Urinary Tract Infection (UTI) and nosocomial UTI caused by *T. asahii* [17]. However, another study also showed that diabetic patients with disseminated Trichosporonosis did not respond to fluconazole therapy and showed remarkable recovery after combination therapy of fluconazole and amphotericin B [18]. Recently, Premamalini, et al. reported that *Trichosporon loubieri on rare occasion* causes disseminated Trichosporonosis with high mortality rate and also causes UTI [19].

Figure 1: Dry, cream white colonies on CLED agar medium after overnight incubation.

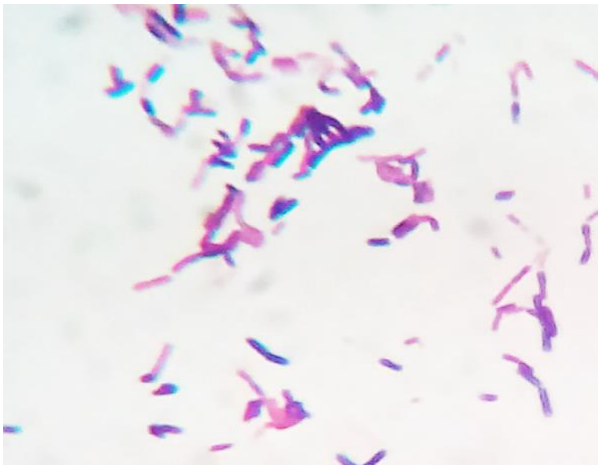

Figure 2: Gram`s stained smear of the isolate as observed under 100X objective.

Although confirmatory tests for species identification could not be done, based on colony morphology, microscopic evidences for the presence septate hyphae and arthospore and urea hydrolysis test the pathogen was most likely *Trichosporon asahii*. To our knowledge, this is the first reported case of *Trichosporon* spp. from Nepal acting as an etiological agent of UTI in immunocompetent patients. The presence of moderate number of pus cells in both urine samples of the symptomatic female suggested active infection. *Trichosporon* spp. was isolated in both urine specimens from the patient and at the same time no bacterial isolate was evidenced. The patient was successfully treated with fluconazole therapy. Based on these evidences *Trichosporon* spp. was established as an etiological agent of UTI.

Conclusion

Most of the pathology laboratories in Nepal lack adequate diagnostic tools. Also, knowledge about fungal infections like with *Trichosporon* spp. is limited due to which they remain undiagnosed. Instead, based on sign and symptom and presence of pyuria patients receive treatment for bacterial infection without laboratory confirmation. Hence, in order to decrease the morbidity and to provide early diagnosis and quality treatment there is an urgent need to increase awareness amongst both laboratorians and clinicians in Nepal about fungaemia and fungal UTI. All laboratory staffs need adequate training on basic fungal culture and identification techniques and the facility for fungal culture should be made available at all microbiology laboratories. All samples for culture from immunocompromised patients should also be sent for fungal culture.

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