CASE REPORT

A rare case of urinary tract infection due to *Trichosporon asahii*

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Abstract

Trichosporon species are fungi that commonly inhabit the soil. They colonise the skin, gastrointestinal tract and respiratory tract of humans. They are a commonly reported cause of disseminated invasive yeast infection in immunocompromised patients. Invasive urinary tract infection caused by Trichosporon asahii is rare. We report a case of urinary tract infection due to Trichosporon asahii in an 80-year-old male patient with idiopathic dilated cardiomyopathy. Yeast-like organisms were isolated from urine samples on three consecutive days as pure cultures. It was identified as Trichosporon asahii using API ID 32C. T. asahii was found to be sensitive to amphotericin B, fluconazole and voriconazole, but resistant to itraconazole by E-test method. The patient responded well to fluconazole treatment. To the best of our knowledge, this is the first report of invasive urinary tract infection caused by Trichosporon asahii in a patient with heart failure.

Keywords: Urinary tract infection, Trichosporon asahii, Fluconazole.

Introduction

Trichosporon species commonly inhabit the soil and may also be present in air, water, organic substrate and in other external environments.[1,2] They colonise mucosal surfaces, skin, respiratory and gastrointestinal tract of humans and have also been detected in faeces, sputum, blood and central venous catheters.[3,4] All pathogenic members of the Trichosporon genus were regarded as single species (Trichosporon beigelii). According to biochemical and morphologic differences, T. beigelii has been divided into distinct species, at least eight of which are considered potential human pathogens: T. cutaneum, T. mucoides, T. inkin, T. domesticum, T. ovoides, T. montevideense, T. asteroides and T. asahii.[4]

T. asteroides, T. ovoides and T. cutaneum are responsible for white piedra or other superficial infections.[5] T. inkin has been reported in superficial as well as disseminated infections. Trichosporon species can cause a disseminated invasive infection known as trichosporonosis. Trichosporonosis is an uncommon but frequently fatal mycosis in immunocompromised patients.[3] Trichosporon genus is an indicator of summer-type hypersensitivity pneumonitis (SHP) in Japan.[6,7]

T. asahii is an emerging fungal pathogen seen particularly in immunocompromised patients. Other reported predisposing factors include extensive burns, organ transplantation, prosthetic valve surgery, human immunodeficiency virus (HIV) infections, corticosteroid therapy, peritoneal dialysis and intravenous catheter.[3,4] T. asahii is an aetiological agent of urinary tract infection in patients. It is possible that the organism colonised the catheter from the human flora during catheterisation and subsequently developed invasive trichosporonosis.[5]

We report a rare case of successfully managed T. asahii infection with intravenously administered fluconazole in a patient with invasive urinary tract infection who had no immunocompromising disorder.

Case Report

An 80-year-old male patient was admitted to the Emergency Department in Istanbul University Cardiology

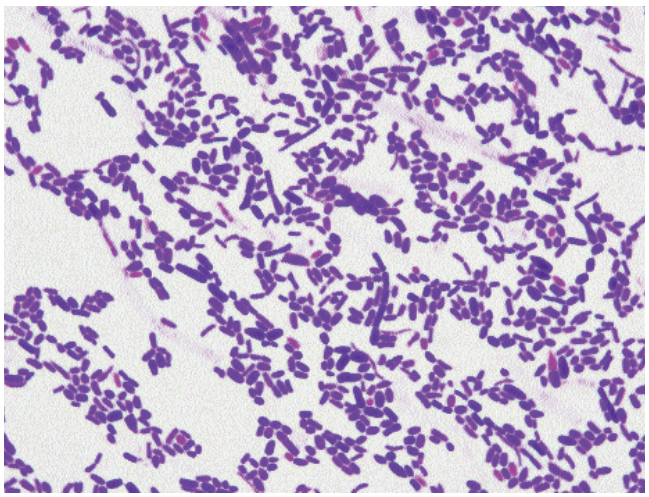

Figure-1 Gram-stained smear from colony on blood agar demonstrating septate hyphae with arthrospores and yeast cells.

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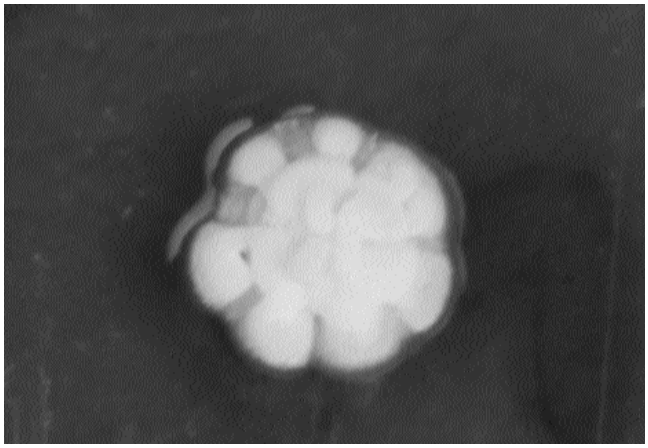

Figure-2: Colony morphology of Trichosporon asahii strain on Sabouraud dextrose agar.

Institute. He was diagnosed as having idiopathic dilated cardiomyopathy. Cardiac Resynchronisation Therapy Defibrillator (CRT-D) implantation was applied in another cardiac centre, but his rhythm was still of atrial fibrillation. He was not on an effective anticoagulant treatment. Because he had passed through a cerebrovascular event, he had left hemiplegia and urine and faeces incontinence. He had impaired swallow reflex and was being nourished via nasogastric tube. He was transferred to a medicine ward for further investigation and examination. After seven days of stay in the medicine ward, dysuria started and his urine sample was sent for routine examination, aerobic culture and sensitivity testing to microbiology laboratory and ciprofloxacin 500 mg twice daily was started per oral. The urine sample was inoculated on blood and Endo agar plates and incubated overnight at 37°C. We did not use CLED media which is standard recommended media due to non- availability. The Gram stain of urine revealed numerous leukocytes and Gram negative rods. Inducible beta-lactamases (IBLs) producing Enterobacteriaceae species were grown (>100.000 CFU/ml). The strain was sensitive to amikacin and ertapenem and was resistant to ciprofloxacin, ofloxacin, amoxicillin/clavulanate, ampicillin/sulbactam, piperacillin/tazobactam, cefazolin, cefuroxime and aztreonam. Ciprofloxacin was stopped because of resistance to quinolones. Ertapenem 1 g 24h intravenous (IV) was started. After the third day of antibiotic treatment, urine sample was sent for repeat culture to microbiology laboratory. The urine sample was also inoculated on blood and Endo agar plates and incubated overnight at 37°C. Tiny, creamy-white, dry, wrinkled colonies were observed on blood and Endo agar as pure culture the next day (>10.000 CFU/ml). Microscopic examination in lactophenol cotton blue and the Gram stain of colony from blood and Endo agar revealed septate hyaline hyphae with arthrospores and budding yeast cells (Figure-1). Fluconazole 200 mg once daily IV was added on the fifth day of antimicrobial therapy. The colony was subcultured on Sabouraud dextrose agar (SDA), which was incubated at 28°C and 37°C. At both temperatures numerous colonies of yeast-like fungus were obtained in pure cultures within 24 hours (Figure-2). Two more consecutive urine samples of the patient were obtained and analysed, which showed similar findings (>1.000 CFU/ml). Yeast-like fungus was grown in both agar plates. The yeast was identified with cornmeal tween 80 agar morphology, urea hydrolysis and by API ID 32°C (Biomerieux, France) and it was identified as Trichosporon asahii. In vitro studies of susceptibility to amphotericin B, fluconazole and voriconazole were performed by use of the E-test (AB Biodisk, Solno, Sweden) method, according to CLSI (Clinical Laboratory Standards Institute) guidelines. Minimum inhibitory concentrations (MIC) were as follows: for amphotericin B 0.25µg/ml; fluconazole 2µg/ml; voriconazole 0.023 µg/ml and itraconazole 3µg/ml. T. asahii was sensitive to amphotericin B, fluconazole and voriconazole, but resistant to itraconazole by E-test method. After seven days the patient responded to treatment. Urine sample was sent for repeat fungal culture to microbiology laboratory and was negative. Ertapenem and fluconazole were stopped.

Discussion

Invasive infections by rare and new opportunistic fungal pathogens have recently emerged as a significant problem in the treatment of immunocompromised hosts. They can be isolated from skin, sputum, urine, blood and IV catheter. T. asahii is an emerging aetiologic agent of disseminated trichosporonosis.[4,5] In three consecutive urine samples isolation of the same yeast in significant counts was isolated, and established T. asahii as an aetiological agent of urinary tract infection.

Trichosporon species are characterised by their ability to form hyphae, pseudohyphae, arthroconidia and blastoconidia. They grow on ordinary Sabouraud-dextrose agar as yeast with cream-colored cerebriform colonies. Arthroconidia is the major microscopic feature that differentiates Trichosporon from Candida species.[4] Though reported rarely, T. asahii is a known pathogen causing urinary tract infection, and has recently been reported sporadically in the literature.[8-10] Trichosporon species are occasionally a part of the normal flora of human skin and in one study this yeast has been documented on intact perigenital skin in 12.4% of the

population.[1] Therefore, it is probable that the fungus colonised the catheter from the skin flora during catheterisation and subsequently progressed towards invasive trichosporonosis.[10] Appropriate antifungal treatment is crucial for these patients. Some studies report that fluconazole and itraconazole are more active in vitro and perhaps are more clinically effective than amphotericin.[5,11,12] In our case, T. asahii was sensitive to fluconazole, but was resistant to itraconazole.

The optimal treatment regimen for trichosporonosis has yet to be identified despite current advances in antifungal therapy of invasive fungal infections. Voriconazole, alone or in combination, is probably the drug of choice for this infection especially in a granulocytopenic patient. In patients who cannot be treated with voriconazole because of its side effects, amphotericin B, flucytosine, fluconazole and itraconazole might be alternative antifungals, although multidrug resistance to these agents resulting in treatment failure has been reported.[3,4] In our patient, we immediately initiated fluconazole and the patient responded well.

Conclusion

Early diagnosis is crucial for successful treatment. Diagnosis is likely to be missed due to lack of acquaintance with the marked diagnostic features of the aetiological agent. Clinicians need to have an increased awareness of fungaemia due to Trichosporon species. Early detection of fungus and accurate identification of this unusual pathogen are necessary to provide specific and appropriate treatment.

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