



ORIGINAL RESEARCH PAPER

Medical Microbiology

TRICHOSPORON ASAHII: AN EMERGING OPPORTUNISTIC FUNGUS AS A CAUSATIVE AGENT OF SEPSIS WITH ACUTE KIDNEY DISEASE-A CASE REPORT FROM A TERTIARY CARE CENTRE

KEY WORDS:

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INTRODUCTION:

Trichosporon asahii is an opportunistic fungal infection that is rarely seen in children and also an emerging cause of systemic fungal infection in immunocompromised hosts ^[1]. Trichosporon asahii (T. asahii) formerly called T. bigelli is an emerging fungal pathogen seen particularly in immunocompromised patients. Trichosporon species have been reported as a cause of fungemia and fatal life-threatening invasive infections in patients with neutropenia and cancer ^[2]. It has a high mortality rate even with amphotericin B treatment ^[1]. Mortality from the infection can be prevented with early diagnosis and starting proper antifungal therapy at an early stage.

CASE REPORT:

A 40yr old male patient was admitted to tertiary care hospital in casualty with complaints of fever, chest pain. On examination he was in sepsis with acute kidney injury with left lower lobe Pneumonia. Patient was already under treatment for left lower lobe pneumonia and left sided pleural effusion since three months but treatment details could not be traced. He was admitted in medicine intensive care unit (MICU) and started on meropenem(1gm). His blood investigation was sent including blood culture and CBC, LFT, KFT and fever profile (includes dengue, widal, malaria, scrub typhus). In CBC it showed low hemoglobin and neutropenia, LFT & KFT were deranged and fever profile was negative.

His blood was inoculated on Blood agar and macConkey agar. His blood culture was positive on second subculture by conventional method. Gram stain of direct smear showed Gram variable septate hyphae with arthrospores were seen. (Fig 2)

It was simultaneously cultured on 2 Sabouraud's dextrose agar (SDA) slopes; one was incubated at 37° C and other at 25° C in biological oxygen demand incubator (BOD).

Next day on blood agar showed white- creamy colony and on SDA it showed cream to whitish, moist, smooth to wrinkled on obverse side and reverse side showed whitish colony (Fig 1). On MacConkey there was no growth.

Urea hydrolysis was performed by streaking the colony on Christensen's agar to differentiate from Geotrichum spp. Urea was hydrolysed after 24 hrs of incubation.

Slide culture and Lacto-phenol cotton blue (LPCB) mount was made from SDA slope.

LPCB mount showed the presence of blastoconidia, septate hyphae and arthroconidia. (Fig 3)

Based on these findings and slide culture a diagnosis of Trichosporon asahii was made.

Antifungal susceptibility was performed by disc diffusion method for Voriconazole, Itraconazole, Fluconazole using reference of Candida species in CLSI document M44-A. ^[4] (as CLSI document doesn't specify Genus Trichosporon). It was sensitive to Itraconazole and resistant to Fluconazole, Voriconazole. (Fig 4)

After blood culture came positive the resident was informed but the patient had already expired on previous day. He died of sepsis and acute kidney injury followed by respiratory failure even after several attempt of resuscitation.

DISCUSSIONS:

Trichosporon species are the second most frequently isolated yeast species after Candida species from immunocompromised patients. They are found in the normal flora of the skin and respiratory tract and can cause superficial and deep fungal infections. Although uncommon, pathogenic spp of Trichosporon are increasingly reported in patients with malignant, transplantation with HIV infection. The most commonly isolated Trichosporon species is reported to be T. asahii. ^[3] There are few reports of invasive infections of T. asahii in non- neutropenic patients. John R (in 2001) reported a case of disseminated infection of T. asahii in a patient without cancer or neutropenia causing fatal septic shock ^[5]. The likely routes of infection of trichosporonosis are the respiratory tract, the alimentary tract and the urinary tract, which are known to be frequent sites of colonization, leading to haematogenous dissemination ^[6]. The patient in our case was on urinary catheter and this may be portal of entry. Voriconazole is the treatment of choice. If the patient's blood culture was sent to microbiological lab and empirical antifungal treatment could started the patient's life would have saved.

CONCLUSION: This case highlights the early diagnosis of such disseminated Trichosporon infection and early treatment so as to help patient's recover quickly.

LIMITATIONS: Used only conventional method. Molecular and automated methods were not used.



Fig 1: Colony morphology of *Trichosporon asahii* on SDA slope showing whitish-creamy colony.



Fig 2: Gram-stained smear showing gram variable budding yeast cells with septate hyphae and arthroconidia

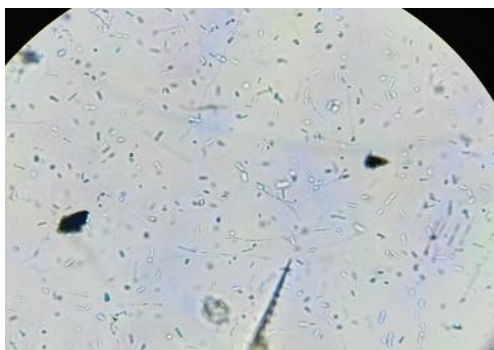


Fig 3: LPCB showing septate hyphae with arthrospores and blastoconidia



Fig 4: Disc diffusion method of antifungal susceptibility testing showing sensitive to itraconazole and resistant to fluconazole and voriconazole

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