INVASIVE TRICHOSPORONOSIS DUE TO TRICHOSPORON ASAHII IN A NON-IMMUNOCOMPROMISED HOST: A RARE CASE REPORT

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Abstract

A case of invasive Trichosporonosis due to Trichosporon asahii in an otherwise healthy young adult male presenting as meningoencephalitis and pneumonia is reported here. T. asahii was isolated from cerebrospinal fluid and sputum of the patient and morphologic forms of organism was also demonstrated on direct Gram stain of sputum. The isolate was identified as T. asahii. The patient partially responded to fluconazole therapy. Our case suggests that T. asahii can no longer be linked with Trichosporonosis in immunocompromised patient alone and any case of meningitis needs thorough mycological workup for its correct etiological identification and appropriate management.

Key words: Trichosporonosis, T. asahii, nonimmunocompromised host

T. asahii (previously known as T. beigelii) is one of the emerging life-threatening opportunistic pathogens especially in granulocytopenic and immunocompromised hosts. Besides, it has been implicated as a cause of hypersensitivity pneumonitis in Japan. However, there is not much information on the prevalence of disseminated trichosporonosis in India and especially none at all regarding its prevalence in nonimmuno compromised host. In this communication, we report a case of central nervous system and pulmonary trichosporonosis due to T. asahii in a nonimmunocompromised patient.

Case Report

An 18-year-old apparently healthy male was admitted to the Medicine department of JLN Hospital, Ajmer in the month of April, 2005. He was unconscious at the time of admission. He had a history of insidious onset of Fever-101°F, chills and rigor associated with headache, nausea and vomiting (since 45 days) and altered sensorium (since three days). The clinical picture was suggestive of chronic meningitis. He had received empirical treatment for malaria and typhoid at a nearby primary health center for three days from where he was referred to our Hospital.

After clinical examination he was started on empiric treatment for malaria (Inj.-quinine) again along with routine conventional antibacterial therapy, viz - Inj. ceftriaxone, amikacin and metrogyl and mannitol and dexamethasone after collection of CSF, urine and blood samples for investigations.

X-ray chest and CT scan of brain was also done. CT scan revealed hypodense area in the left paraventricular region in frontal lobe suggestive of an infarct and also signs of impending hydrocephalus and meningitis. X-ray chest showed obliteration of left CP angle suggesting pleural effusion. There was dullness on percussion of the left side of the chest. Pleural fluid and sputum were also sent for Gram stain and culture as per our advice.

Routine hematological investigations were within normal range, except for ESR (30 mm at one hour). Malarial parasite QBC for malaria and widal test were negative. Peripheral blood smear showed normal picture and fasting blood sugar was 151 mg.

The CSF pressure was slightly raised and on gross examination was clear. Biochemical analysis of CSF revealed raised protein (400 mg%) diminished glucose (18 mg%) and 9.6 mg% chloride. The cytological analysis showed a slightly increased WBC count of 40/mm³ (L-50%, N-50%). Thus the picture was suggestive of both aseptic and tubercular meningitis.

The centrifuged deposit of CSF was cultured onto MacConkey agar, blood agar and chocolate agar for recovery of aerobic pathogens. Wet mount preparation of the deposit showed few mononuclear cells and Gram stain showed few inflammatory cells (both lymphocytes and neutrophils) but no microorganism was seen. After 24h incubation, all the three media showed growth of numerous colonies that were of 3-4 mm in size, wrinkled and folded with irregular margins. The colonies on MacConkey’s agar were non-lactose fermenting and oxidase positive; while that on blood agar were white, more profusely wrinkled and non-hemolytic.

Gram stain of the colonies from all the three media showed budding yeasts, hyphae and arthroconidia. A provisional diagnosis of yeast like fungi was intimated to the treating clinician for prompt institution of anti-fungal therapy. Gram...
stain of sputum also showed similar morphologic forms and the sputum was cultured onto Sabouraud dextrose agar (SDA). Subcultures of the colony from blood agar were also made onto two sets of SDA with and without cycloheximide and incubated at 25°C and 37°C respectively which also yielded similar colonies within 24h. After 72h incubation, the colonies on SDA were bigger, 1cm in size, more profusely wrinkled and heaped up in the center. There were two to three radial cracks in the medium and the margin was irregular and wavy. Growth however was delayed on SDA containing cycloheximide (0.05 mg/L) for up to 72h. The isolate was identified as *Trichosporon* spp. based on cornmeal agar morphology, urease activity, growth on SDA at 37°C and 42°C but not at 45°C and resistance to cycloheximide (0.01 mg/L). The isolate was also sent to PGIMER, Chandigarh where it was confirmed to be *T. asahii*.

The identification of the isolate as *Trichosporon* was intimate to the treating physician within four days of arrival of the sample in our laboratory and accordingly the patient was started on fluconazole. The patient partially responded as he became semiconscious and followed verbal command within 48h of institution of antifungal therapy but was still febrile. The left-sided chest dullness persisted even on the seventeenth day of admission after which the patient left against medical advice and was lost to follow-up.

**Discussion**

Invasive infection by rare and new opportunistic fungal pathogens have recently emerged as a significant problem in the treatment of immunocompromised hosts. Since the first report on disseminated trichosporonosis in a leukemic patient in 1970, sporadic cases have been reported over the years. Mathews and Prabhakar in 1995 reported a case of localized invasive trichosporonosis of CNS due to *T. beigelii* from India. Recently Chakrabarti and co-workers described a case of generalized lymphadenopathy caused by *T. asahii* in a patient with Jobs syndrome. While these instances were in immunocompromized patients, our patient was immunocompetent (physically well-nourished, no neutropenia, HIV negative, normal blood picture, no detectable malignancy, not on chemotherapy or immunosuppression). Diabetes mellitus is recognized as a risk factor for developing invasive trichosporonosis and is the only risk factor, which could have contributed to trichosporonosis in this case (indicated by high blood sugar) though the patient gave no past history of this illness. Wolf and colleagues reported *T. asahii* infection in six nongranulocytopenic patients in ICUs in 2001. Our case of invasive trichosporonosis had manifestations of CNS disease and pneumonia not responding to conventional antibacterial antibiotic therapy. The etiological role of *T. asahii* was unequivocally established by direct demonstration in sputum and isolation in culture from this material as well as from CSF.

SDA is not routinely used for culture of CSF for pyogenic organisms in many laboratories and this in no way is going to affect the sensitivity of isolation of *Trichosporon* spp. as is evident from this case. *T. asahii* was seen to grow luxuriantly on MacConkey and blood agar. The macroscopic and microscopic morphology, biochemical and physiological characteristics of *T. asahii* was compatible with the standard description of the species.

The likely route of infection in the present case is through inhalation and the mode of spread to CNS is probably through disruption of blood brain barrier. Matsunaga and colleagues have reported the role of TA-19 protein antigen in *T. asahii* as a virulence factor for developing summer type hypersensitivity pneumonitis. This could have contributed to the pathogenesis of invasive disease.

Azoles are shown to be more effective in the treatment of Trichosporonosis than amphotericin B. In the present case, the patient seemed to partially respond to fluconazole therapy. However, combination therapy with both fluconazole and amphotericin B would have yielded better response as was reported by Wolf et al in immunocompetent patients. But the ultimate outcome of the treatment could not be assessed as the patient away against on medical advice.

With the establishment of the pathogenic role of *T. asahii* even in nonimmuno compromised host the incidence of trichosporonosis is expected to escalate. This demands intensive mycological investigations for early identification of the biological agent and timely institution of appropriate antifungal therapy along with elimination of predisposing factors for favorable patient outcome.

**Acknowledgement**

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**References**

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**Announcement**

**Dr. J. C. Patel Birth Centenary Celebration Committee**

The year 2008 is the Birth Centenary Year of Dr. J. C. Patel. Some of his students/admirers felt that it would be a good idea to celebrate this Centenary Year by organizing CMEs, Orations/Lectures, Conferences, etc. during the year. He was associated with many professional bodies, which meet regularly every year; during these annual meetings/conferences, a lecture/symposium, etc. can be organized as a part of Centenary celebrations. We would like to form a Dr. J. C. Patel Birth Centenary Celebrations Committee. All his past students/admirers are invited to join the committee (without any financial commitment). Kindly communicate your name, designation, postal address, telephone number and E-mail ID to Dr. B. C. Mehta at Flat 504, Prachi Society, Juhu-Versova Link Road, Andheri (W0, Mumbai 400 053 (drmehta.bc@gmail.com).