Original Research Article

Prevalence of invasive Trichosporonosis by *Trichosporon asahii* and other *Trichosporon species* and their antifungal susceptibility pattern in Chhattisgarh

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Abstract

Introduction: Trichosporonosis is usually known to cause superficial mycoses, but now it is emerged as an opportunistic infectious disease. *Trichosporon species* is fairly uncommon fungus but can cause fatal mycosis in immunocompromised patients. **Objective:** This study is an attempt to know prevalence of invasive trichosporonosis and its antifungal susceptibility. **Materials and Methods:** All patients with a culture that was positive for *Trichosporon* species from February 2012 to February 2015 were included. Routine mycology works up done and suspected *Trichosporon* sp. were confirmed by automated *miniAPI* system. Antifungal susceptibility test was done for Fluconazole (F), Itraconazole (Itr), Voriconazole(V), Flucytosine (5Fc), AmphotericinB (AMB) ~~done~~ by minimum inhibitory concentration (MIC) method by ATB Fungus3 (Biomerieux, France). **Result:** 41 *Trichosporon sp.* was isolated from clinical specimen. *Trichosporon asahii* was the most common isolate (29 out of 41, 70.7%), followed by *T. mucoides* (5 of 41, 12.2%), *T. inkin* (2 of 41, 4.9%) and other *Trichosporon sp.* (5 out of 41,12.2%). Out of 41, 20 cases were proven to cause invasive trichosporonosis. Most invasive infections were associated with indwelling catheter (95%), associated bacterial infection (85%), ICU stay (85% each), prior antibiotic use (75%), cancer (65%), neutropenia, steroid use (55% each) and chemotherapy (50%). Amphotericin B was less susceptible against *Trichosporon* isolates whereas azole had good in vitro activity. Sensitivity of *T.asahii* towards Fluconazole, Itraconazole, Voriconazole, Amphotericin B and Flucytosine was 72.4%, 51.7%, 86.2%, 51.7% and 66.8% respectively. **Conclusion:** *T. asahii* and other unusual *Trichosporon sp.*~~species~~ also cause invasive trichosporonosis. For optimal therapy for trichosporonosis azoles can play a potential role.

Keywords: Trichosporonosis, *Trichosporon* species, *T.asahii*, Antifungal susceptibility.

Introduction

Fungi are known to mankind since a long time and omnipresent in environment. However, reports emanating from mycological works are very few and far in between. Therefore, their clinical relevance often gets neglected. But with changing health scenario, fungi are now considered as an emerging pathogen [1]. The annual incidence of mycoses was increased by over 200% in between 1979 to 2000 [2]. Though *nonalbicans Candida* are the most common fungal pathogens encountered in clinical specimens [2, 3], now-a-days infection due to *Trichosporon sp*. are increasing. It is one of the least understood among emerging opportunistic pathogens causing fatal fungal infection in immunocompromised patients [4]. It is associated with spectrum of clinical diseases from superficial cutaneous mycoses in immunocompetent individual to severe invasive systemic diseases in immunocompromised patients [5].

Trichosporon Behrend is a basidiomycetes yeast, with a unique morphological characters of budding cell and true mycelium that disarticulate to form arthroconidia [6, 7]. Previously, only one species *T. beigelli* was reported as pathological etiological agent for superficial infection such as white piedra, nail infection & tinea

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cruris [8]. Now on the basis of ultrastructure & DNA studies, *T. beigelli* has been divided into number of species. Among them major human pathogens are – *T. asahii, T. astoreoides, T. mucoides, T. cutanei, T. ovoides, T. inkin, T. loubieri* [9].

It is present in environment, mainly in soil, water, air and organic substance. It is also present as normal flora of human skin & gastrointestinal tract [9-11]. Among different *Trichosporon* sp. *T. asahii* is the most important pathogen in imunocompromised and granulocytopenic patients [4, 12].

In routine laboratory set up, usually its diagnosis is likely to be missed, particularly in developing countries. It is due to lack of awareness and lack of acquaintance with the salient diagnostic feature of the etiologic agent.

Barring a few isolated case-reports, there is no information on the prevalence of disseminated invasive *Trichosporon*osis in India.Most of the time *Trichosporon sp.* are reported as *nonalbicans Candida* in routine laboratory study. The aim of this study is to investigate the prevalence of *Trichosporon* sp. in population of Chhattisgarh along with spectrum of clinical features and antifungal susceptibility.

Materials and Methods

This retrospective study was conducted in Department of Microbiology of Chhattisgarh Institute of Medical Sciences, Bilaspur, Chhattisgarh from February 2012 to February 2015. After taking the permission from ethical committee, all patients with a culture positive for *Trichosporon sp*. from clinical specimen included in this study except skin and hair.

Whenever required, a second specimen was requested for confirmation. The strain *Candida albicans* American type culture collection (ATCC) 14053 was used as control.

Methods: The blood culture was done by automated BacT/ALERT (Biomerieux, France) blood culture system. On getting growth signal, gram stain of blood culture broth showed elongated blastoconidia and septate pseudohyphae.

Broth from the positive blood cultures bottles were sub-cultured on blood agar (Hi Media, India) and Sabouraud's Dextrose agar with Chloramphenicol (SDA) (Hi Media, India) and incubated at 37^{o}C. After 24 to 48 hours of incubation, the colonies of yeast like fungi were isolated.

Pus, body fluids, sputum, cerebrospinal fluid (CSF) etc. were inoculated on Blood agar, MacConkey Agar (Hi Media, India) and SDA. Urine specimens were inoculated on Cystein Lactose Electrolyte Deficient (Hi Media, India) and SDA. Culture plates were incubated aerobically at 37^{o}C for 24-48 hours. Gram stain showed gram positive blastoconidia and pseudohyphae. To differentiate from *Candida sp*., germ tube test, urease test and corn meal test were done.

Fungus with negative germ tube test, positive urease test and arthroconidia in Corn Meal test were suspected as *Trichosporon sp*. All suspected *Trichosporon* isolates were identified up to the species level by standard laboratory procedures, including morphological identification & ID 32C strips of *miniAPI* (Biomerieux, France).

Antifungal susceptibility test for Fluconazole (F), Itraconazole (Itr), Voriconazole (V), Flucytosine (5Fc) and AmphotericinB (AMB) was done by minimum inhibitory concentration (MIC) method by ATB Fungus 3 (Biomerieux, France). The macroscopic and microscopic morphology of *Trichosporon sp*. was compatible with the standard description of the species.

Invasive fungal infection was defined according to Invasive Fungal Infection Co-operation Group of European Organisation for Research & Treatment of Cancer and mycoses study group of National Institute of Allergy & Infectious Diseases [13]. According to this group invasive Trichosporonosis was defined as "proven" or "probable" cases.Invasive Trichosporonosis was considered as "proven", when one or more of the following criteria were met: (1) blood culture yielding *Trichosporon sp*. in patients with clinical sign and symptoms (2) positive CSF culture for *Trichosporon sp*. (3) positive tissue biopsy culture for *Trichosporon sp*. with histopathological evidence of fungal growth.

Cases with following criteria were defined as "probable": (1) presence of at least one host factor criterion: neutropenia, cancer, on immunosuppressive therapy, fever refractory to broad spectrum antibiotics (2) one microbiological criterion (3) one major clinical criterion consistent with infection i.e. imaging.

Results

During 3 years study period, 41 isolates of *Trichosporon sp*. were cultured from various clinical specimens from the patients. Out of 41 isolates, 25 isolates were from male patients (61%) and 16 (39%) from female patients, indicating male preponderance (table 1). Maximum 15 isolates (36.6%) were from patients of 61-80 years age group, followed by 11 isolates (26.8%) in 41- 60 years age group, 9 isolates (21.9%) were from patients of 0-20 years age group. From the above distribution, Trichosporonosis appears to be more frequent at extreme of age group. Out of 9 patients of 0-20 years age group, 8 patients were below one year age. Out of 41, 20 patients (48.8%) had proven invasive Trichosporonosis whereas 21 patients (51.2%) had probable invasive Trichosporonosis.

Table No-1: Age distribution of patients included in the study (n= 41).

Age	Male	Female	Total	Proven invasive Trichosporonosis	Probable invasive Trichosporonosis	P value*
0-20 years	6 (14.6%)	3 (7.3%)	9 (21.9%)	8	1	<0.05
21-40 years	4 (9.7%)	2 (4.9%)	6 (14.6%)	3	3	
41-60 years	6 (14.6%)	5 (12.2%)	11 (26.8 %)	4	7	
61-80 years	9 (22%)	6(14.6%)	15 (36.6%)	5	10	
Total	**25 (61%)**	**16 (39%)**	**41(100%)**	**20**	**21**	

* P Value calculated by chi square rule.

From the above table 1, we calculated p value which was <0.05, which showed association between age interval and proven invasive Trichosporonosis.

Table No-2: Specimen wise distribution of *Trichosporon* species (n=41).

Specimen	*T. asahii*	*T. mucoides*	*T.inkin*	Other Trichosporon	Total	Proven invasive Trichosporonosis
Blood	10	1	1	1	13(31.7%)	13 (65%)
CSF	3	0	0	0	3 (7.3%)	3 (15%)
Urine	6	2	1	1	10(24.4%)	0 (0%)
Pus	3	1	0	1	5(12.2%)	1 (5%)
ET Aspirates	4	1	0	1	6(14.6%)	2 (10%)
Other specimen	3	0	0	1	4(9.7%)	0
Total	**29 (70.7%)**	**5 (12.2%)**	**2 (4.9%)**	**5 (12.2%)**	**41(100%)**	**20 (100%)**

In our study, *Trichosporon sp*. was isolated from wide range of clinical specimen. As shown in the table 2, *T.asahii* was isolated from 29 specimen (70.7%), *T.mucoides* from 5(12.2%), *T. inkin* from only 2 specimen (4.9%), other *Trichosporon sp.* 5 (12.2%). In our study *T.asahii* was the most common species of *Trichospron* isolated. Out of 20 proven invasive Trichosporonosis, 13 had blood stream infection, three had central nervous system infection, two had pneumonia and only one had soft tissue infection.

As shown in table 3, out of 20 invasive Trichosporonosis, 40% patients were below 1 year of age whereas 25% patients were above 60 years of age. The most common associated risk factor for proven Trichosporonosis was indwelling catheter (95%), associated bacterial infection (85%), ICU stay (85%), prior antibiotic use (75%), cancer (65%), neutropenia (55%), steroid use (55%) and chemotherapy(50%). Other risk factors include dialysis and diabetes mellitus and road traffic accidents.

Table No-3: Risk factors associated with Trichosporonosis (n =41).

Risk factors	Number of patients (n=41)	Proven invasive Trichosporonosis (n=20)	Probable Trichosporonosis (n=21)
Age < 1 year	8 (19.5 %)	8 (40%)	1(4.8%)
Age > 60 year	15 (36.6%)	5 (25%)	10(47.6%)
Cancer	17 (41.5%)	13(65%)	4(19%)
On chemotherapy	11(26.8%)	10(50%)	1(4.8%)
Prior antibiotic use	35(85.4%)	15 (75%)	20(95.2%)
Intravenous catheter	32(78%)	19(95%)	13(61.9%)
ICU stay	36(87.8%)	17 (85%)	19 (90.4%)
Steroid use	28(68.3%)	11 (55%)	17 (80.9%)
Neutropenia <500 cell/ mm3	15(36.6%)	11 (55%)	4(19%)
Dialysis	7 (17.1%)	2(10%)	4(19%)
Diabetes	13 (31.7%)	5 (25%)	8(38%)
Road traffic accident	7(17.1%)	3(15%)	4(19%)
Associated bacterial infection	28(68.3%)	17((85%)	11(52.4%)

Antifungal susceptibility pattern: As no MIC interpretative criteria for *Trichosporon sp.* was available, the interpretative criteria for *Candida species* were used as reference purpose [14, 15]. They were considered as sensitive, resistant and intermediate (susceptible dose depended in case of Fluconazole) by *miniAPI* as per M27A3 of Clinical and Laboratory Standards Institute (CLSI) guideline [16]. Only 21out of 29 (72.4%) *T.asahii* were sensitive to Fluconazole (F) and 25 out of 29 (86.2%) were sensitive to Voriconazole (V) whereas other *Trichosporon sp*. was 58.3% sensitive to

Table No-4: Antifungal susceptibility of *Trichosporon* isolates (n=41).

Antifungal agents	*T.asahii* (n=29)	*T.mucoides* (n=5)	*T.inkin* (n=2)	Other *Trichosporon*(n=5)	*Trichosporon* other than *T.asahii*(n=12)
Fluconazole					
Sensitive	21(72.4%)	2(40%)	2 (100%)	3(60%)	7 (58.3%)
Resistant	3 (10.3%)	2 (40%)	0	1 (20%)	3(25%)
Intermediate	5 (17.3%)	1(20%)	0	1(20%)	2 (16.7%)
Itraconazole					
Sensitive	15 (51.7%)	3 (60%)	1(50%)	2 (40%)	6(50%)
Resistant	12 (41.4%)	1(20%)	1(50%)	1 (20%)	3(25%)
Intermediate	2 (6.9%)	1(20%)	0	2 (40%)	3(25%)
Voriconazole					
Sensitive	25 (86.2%)	3(60%)	2 (100%)	3 (60%)	8(66.6%)
Resistant	3 (10.3%)	1(20%)	0	1 (20%)	2(16.7%)
Intermediate	1(3.5%)	1(20%)	0	1 (20%)	2(16.7%)
Amphotericin B					
Sensitive	15(51.7%)	2 (40%)	1 (50%)	2 (40%)	5(41.7%)
Resistant	14(48.3%)	2 (40%)	1(50%)	1 (20%)	4(33.3%)
Intermediate	0	1(20%)	0	2(40%)	3(25%)
Flucytosine					
Sensitive	22(66.8%)	4 (80%)	2(100%)	4 (80%)	10(83.3%)
Resistant	5(17.3%)	1 (20%)	0	1 (20%)	2 (16.7%)
Intermediate	2 (6.9%)	0	0	0	0

Fluconazole and 66.6% sensitive to Voriconazole. *T. inkin* showed no resistance toward both Fluconazole and Voriconazole. On the other hand 40% and 20% *T.mucoides* were resistant to Fluconazole and Voriconazole respectively. *T.asahii* showed 41.4% resistant to Itraconazole and 25% of other *Trichosporon sp.* was resistant to it. (Table 4)

In our study, 48.3 % (15 out of 29) isolates of *T.asahii* were resistant to AmphotericinB (AMP), whereas 33.3% (4 out of 12) isolates of other *Trichosporon sp.* were resistant to it. Among them *T.inkin* showed 50% (1 out of 2 isolates) and *T.mucoides* showed 40% resistance. On the other hand, *T.asahii* was 66.8% sensitive to Flucytosine. In comparison other *Trichosporon sp.* were 83.3 % (10 out of 12 isolates) resistant to Flucytosine.

Table-5: Antifungal susceptibility of 41 clinical *Trichosporon* isolates, determined by MIC (mg/lit).

***Trichosporon* sp.**	**Range of MIC**	**MIC_{50}**	**MIC_{90}**
Fluconazole	0.25-64	4	8
Itraconazole	0.03-2	0.12	0.25
Voriconazole	0.015-0.5	0.03	0.12
Amphotericin B	0.5>-16	1	>16
Flucytosine	0.5 -64	4	8

We also made an attempt to determine MIC ranges, MIC_{50} values and MIC_{90} values for the 41 *Trichosporon* isolates against 5 antifungal agents as shown in table 5. Most of the isolates exhibited relatively high Amphotericin B MICs. Azoles had good in vitro activity against the *Trichosporon* isolates, especially Voriconazole which showed 0.12 mg/lit MIC_{90}. For the isolates with higher Fluconazole MICs, Voriconazole also demonstrated good potency (MIC $\leq$ 0.5 mg/lit). Susceptibility profiles were similar among the different *Trichosporon* species and among the different isolates from the various infections.

Discussion

In the present study, *Trichosporon* sp. were isolated from 41 patients. Out of 41, 20 had invasive Trichosporonosis whereas 21 patients had probable Trichosporonosis. All ~~of the~~ isolates were identified as *Trichospron sp*. based on KOH wet mount, colony morphology, gram stain, corn meal test and urease test. Usually *Trichosporon sp*. is confused with *Geotrichium sp*. as both of them possess arthroconidia.

They are differentiated on the basis of urease test and corn meal test. *Trichosporon sp.* are urease test positive and both arthroconidia and blastoconidia seen in corn meal test whereas *Geotrichium sp*. is urease test negative and hockey stick shaped arthroconidia seen without blastoconidia [8]. All the suspected were further tested with API ID 32C. By this we were able to do speciation into *T.ashaii*, *T.mucoides* and *T. inkin*. In our study, *T.asahii* was the most common *Trichosporon sp* isolated (70.7%, 29 out of 41). Similar to our study, *T.asahii* was also the commonest *Trichosporon sp*. isolated from Japan, Turkey, Taiwan and Brazil [17-20]. In one of the largest multicentre retrospective study on invasive *Trichosporon* infections, they found that *T. asahii* accounted for 61% (17of 28) of cases [21].

In our study *Trichosporon sp.* was isolated from wide range of clinical specimen. Blood stream infection was most common (31.7%) form of trichosporonosis. All of them were proven invasive trichosporonosis (13 out of 20 i.e. 65%). 8 out of 20 were reported from NICU. Cases on neonatal sepsis due to *T.asahii* were also reported from India by Vashishtha et al [22]. Fungemia due to *Trichosporon* was most common in cancer patients as reported by Girmenia et al (74.7%) [21].

Unlike other study in which *T. asteroides* was the second most common cause of blood stream infection, in our study *T. mucoides* was second most common causative species causing blood stream infection [20]. In that study, 7 *T. mucoides* had been reported to be associated mostly with invasive infection [20].

In our study, urinary tract infection (UTI) was second most common form of trichosporonosis (24.4%). In India different case report about *T.asahii* causing UTI had been published at different point of time [23, 24]. Like our study other than blood and urine, *Trichosporon sp.* had been isolated from pus, soft tissue, respiratory specimens and CSF [25,26].

Majority of trichosporonosis occur in immune-compromised person, especially in cancer or neutropenic patients [27]. But in our study we came across other risk factors such as extreme of age, indwelling catheter, prior antibiotic uses, prolonged ICU stay, use of steroid and dialysis which was also reported by other author [27, 28]. *Trichosporon sp*. has the ability to produce biofilm, different enzymes like proteases and phospholipases, morphological switching and cell wall antigenic components like glucuronoxylomannan acts as virulence factors. Also associated bacterial and Candida infection increase the invasiveness of *Trichosporon sp*. [27].

Antifungal susceptibility pattern: There is no guideline for treatment of invasive trichosporonosis due to lack of well-designed clinical studies as well as emerging nature of the disease. So in vitro susceptibility testing can provide some useful evidence for guiding the treatment. In our study, we observed the emergence of antifungal resistant strain of *Trichosporon sp,* which is a matter of great concerned in clinical point of view. In the present study, in comparison to AmphotericinB, azoles showed good potency against *T. asahii* isolates, especially Voriconazole.

Among azoles, Itraconazole was least sensitive (51.7%) whereas Fluconazole was (72.4%) sensitive in *T.asahii*. So, our study suggests that azoles are the preferred antifungal agents for invasive Trichosporonosis and that Voriconazole can be the drug of choice. Study by Chagas *et al* on 22 *Trichosporon* isolates also showed poor susceptibility to AmphotericinB but good in vitro susceptibility to azole [20]. In their study,Rodriguez - Tudela *et al* found that MICs of *T. asahii* isolates for Amphotericin B were ≥2 µg/ml[29]. In a study done in Taiwan on 101 strains of *T. asahii*, a low susceptibility to Fluconazole and Amphotericin B was observed and Voriconazole was suggested as drug of choice [19]. Emergence of resistant strain of *Trichosporon sp*. had been reported from different part of world including Turkey, Italy etc [18, 21], but not from India.

The mechanism of antifungal resistant is not known in *Trichosporon sp*. Molecular study can give some light on it. But due to financial constrain we were unable to do it. Increased and indiscriminate use of antifungal drug may lead to selection and isolation of more resistant strain in the future. As this is a tertiary health care center, most of the patients were referred from other hospital and nursing home and were already on different antibiotics and antifungal agents. That may be the reason of emergence of resistant strain.

Conclusion

This present study indicates strongly that *T.asahii* and other *Trichosporon sp.* have the propensity to cause life threatening invasive Trichosporonosis. The emergence of drug resistance in *Trichosporon sp.* must be kept in mind during treatment. So, we recommend to do the species identification of *Trichosporon sp.* for all clinically relevant isolates as well as to determine susceptibility testing of antifungal drugs for better patient care. To the best of our knowledge, this is the first and largest study on *Trichosporon sp.* not only in tribal dominated state of Chhattisgarh but also in this central region of India.

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