Case Report

A rare case of fatal sepsis caused by *Trichosporon asahii* in an immunocompetent chronic obstructive pulmonary disease patient

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ABSTRACT

Trichosporon asahii is a yeast-like organism that is emerging as an important cause of invasive fungal infection in immunocompromised patients, especially in patients with cancer and neutropenia. Invasive infections due to *T. asahii* in immunocompetent patients are rarely reported. We describe a 62-year-old chronic obstructive pulmonary disease patient who contracted severe sepsis from *T. asahii* without any prior history of immunosuppression. He was successfully treated with voriconazole and was discharged from the hospital.

Key words: Chronic obstructive pulmonary disease, Immunocompromised, Sepsis, *Trichosporon asahii*

Trichosporon species are yeast-like anamorphic organisms that are classified under *Basidiomycetes* yeasts. It is an opportunistic fungus that mainly causes fatal infections in immunocompromised patients and has high mortality rates despite adequate treatment [1]. Systemic infections are more likely in people with cancer, burns, solid organ transplants, patients on steroids, peritoneal dialysis, prolonged mechanical ventilation, and those having prosthetic valve surgeries [2,3]. Although many cases have been reported in immunocompromised patients and hosts, only a few cases have been documented in the literature [4-6].

We describe an unusual instance of *Trichosporon asahii*-caused fungemia in a chronic obstructive pulmonary disease (COPD) patient who had neither malignancy nor neutropenia.

CASE REPORT

A 62-year-old man who had been diagnosed with COPD for 10 years and worked as a farmer was sent to our intensive care unit (ICU) after complaining of a persistent cough and shortness of breath for 10 days. He was initially admitted to another hospital, where he received 5 days of empirical treatment with ceftriaxone 1 g bid, azithromycin 500 mg once daily, and non-invasive ventilation (NIV) for a lower respiratory tract infection. He was referred to our hospital for the requirement of tertiary care and invasive mechanical ventilation.

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On assessment, the patient had tachypnea (40/min), tachycardia (123 beats/min), and a saturation of 68% at room air. He had a silent chest on auscultation and other systemic examinations were normal.

Initial arterial blood gas analysis revealed Type 2 respiratory failure that was acute on chronic (pH-7.30, pCO2-60.2, pO2-60, and HCO3-29). An X-ray of the chest showed hyperinflated lung fields (Fig. 1). His initial laboratory tests showed an increased C-reactive protein (250.7), and procalcitonin (1.19). His COVID reverse transcription-polymerase chain reaction was negative, and the total WBC count was 13100/mm^3 with neutrophilia (88%). An acute COPD exacerbation with a likely infectious cause was diagnosed.

He was originally treated with bronchodilators, empirical piperacillin-tazobactam 4.5 g qid, and NIV. After the failure of NIV, he was intubated and managed with a lung-protective ventilation strategy. He developed shock during his time in the ICU; other potential causes of shock were eliminated, and sepsis with septic shock was determined. Due to the patient's deteriorating clinical condition and recent history of hospital admission, the antibiotic was empirically escalated to Meropenem 1 g tid. His blood culture revealed the growth of *T. asahii* (Fig. 2). Due to the lack of growth media in our laboratory, antifungal susceptibility testing was not performed. In accordance with the recommendation of the ESCMID/ECMM guidelines, he was started on voriconazole 400 mg bid on day 1 and was afterward given 200 mg bid. His sputum culture revealed the growth of *Klebsiella pneumoniae*, for which Meropenem was continued according to culture and

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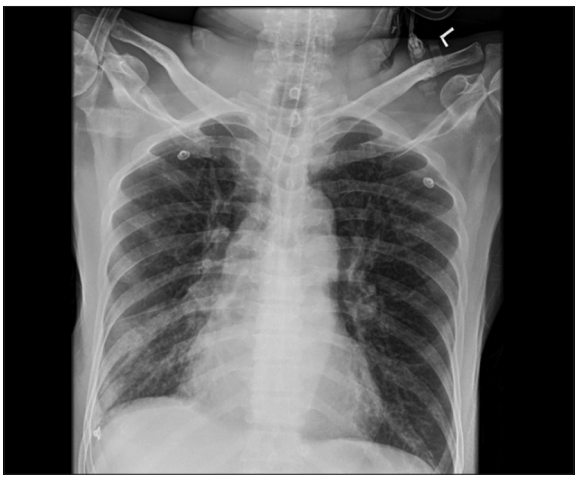

Figure 1: Chest X-ray showing hyperinflated lung fields with endotracheal tube *in situ*

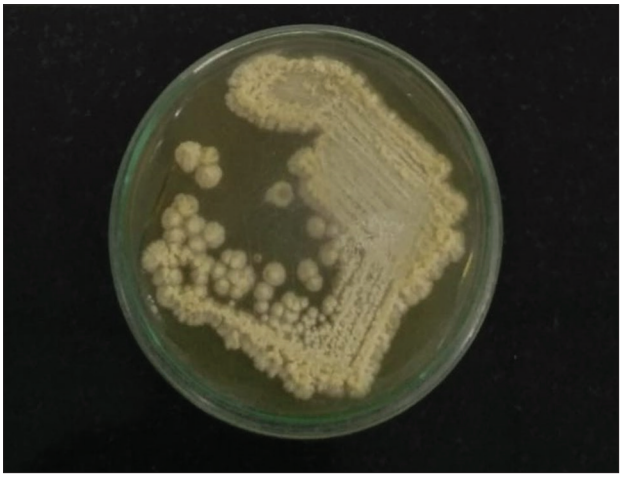

Figure 2: Cream-colored wrinkled colonies of *Trichosporon asahii* on sabouraud dextrose agar after 1 week of incubation

sensitivity. His clinical condition improved as his respiratory failure and shock subsided. After 4 days in the ICU, he was extubated. Post-extubation, he was managed with intermittent NIV for a day. On the 7th day after being admitted, he was transferred out of the ICU.

DISCUSSION

Trichosporon species are anamorphic organisms that belong to *Basidiomycetes* yeasts. They are ubiquitous in nature and are part of the typical flora of the human skin, respiratory, and gastrointestinal tracts [7]. Six *Trichosporon* species, namely, *Trichosporon. asahii, Trichosporon asteroides*, *Trichosporon cutaneum*, *Trichosporon inkin*, *Trichosporon mucoides*, and *Trichosporon ovoides*, are responsible for invasive and superficial infections of the skin and mucosa. *T. asahii* is the most common etiological agent in invasive *Trichosporon* infections [8]. *T. asahii* can cause a wide range of clinical presentations ranging from cutaneous infections to fatal disseminated infections in immunocompromised individuals. Urinary tract infections, fungemia, and disseminated infections are the three most frequent kinds of invasive infections [1]. The patients who are immunocompromised have an 80% mortality rate from invasive trichosporonosis [9].

Risk factors for invasive trichosporonosis are cancer (hematological malignancy), burns, transplant patients as well as patients on steroids, peritoneal dialysis, prolonged mechanical ventilation, and those undergoing prosthetic valve surgeries [2,3]. Hematologic disorders, diabetes mellitus, and pulmonary disorders were shown to be the most prevalent pre-existing conditions in individuals with *T. asahii* infections in a study by Li *et al*. [1]. The ICU admission, usage of invasive medical equipment, and broad-spectrum antibiotics were also identified in the same study as predominant risk factors for invasive *Trichosporon* infections. Our patient is a known farmer, had long-standing COPD, and had recently been admitted to the ICU with broad-spectrum antibiotics use which made him susceptible to *Trichosporon* fungemia.

Patients with *T. asahii* infections have a wide range of non-specific clinical manifestations. In over 140 cases reported, fungemia was the most common presentation followed by urinary tract infection and respiratory tract infection. Disseminated infection, skin infection, peritonitis, and meningoencephalitis were other reported infections [1]. Our patient had sepsis with septic shock and fungemia. Fungemia caused by *T. asahii* was associated with a very high mortality rate (30.3%) [1].

The literature contains very few reports of invasive *T. asahii* infections in immunocompetent patients. John R. reported a case of a widespread *T. asahii* infection leading to lethal septic shock in patients who were non-neutropenic immunocompetent [4]. A young healthy male with meningoencephalitis and pneumonia due to *T. asahii* infection was reported by Rastogi VL [5]. There were six cases of multidrug-resistant *T. asahii* infection reported by Wolf *et al*. in non-neutropenic patients. Only one patient had underlying COPD as a risk factor [6].

Trichosporon species express an intrinsic resistance to the echinocandins and poor responsiveness to the polyenes [9,10]. Patients with hematological conditions respond well to voriconazole-based therapy [11]. The ESCMID/ECMM guidelines also recommend the use of voriconazole for the treatment of trichosporosis [9]. We used voriconazole and the patient responded well with resolving sepsis and septic shock. A repeat blood culture was negative for *T. asahii* after the treatment.

CONCLUSION

Invasive Trichosporonosis is rare in immunocompetent patients which makes it difficult to suspect and treat. A high index of suspicion in patients with risk factors is important in identifying and treating it promptly as mortality of untreated patients is very high.

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