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Case Report

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Trichosporon Asahii as a Causal Agent of Invasive Fungal Infection in a Pediatric Oncology Patient

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Abstract

We present the case of a pediatric patient with acute myeloid leukemia who developed an invasive fungal infection caused by Trichosporon asahii following hematopoietic stem cell transplantation. The infection occurred despite antifungal prophylaxis with caspofungin and was confirmed by blood cultures and MALDI-TOF identification. Treatment with liposomal amphotericin B and isavuconazole led to clinical improvement. This case highlights the importance of early recognition of rare fungal pathogens intrinsically resistant to echinocandins and the need to consider breakthrough infections in immunocompromised patients under antifungal prophylaxis.

Keywords: Prolonged fever, Leukemia, Trichosporon Asahii, Liposomal Amphotericin B, Isavoconazole

Introduction

The spectrum of fungal infections in pediatric oncology units has evolved over time. The introduction of antifungal agents such as azoles in the 1980s led to a decline in invasive infections caused by the genus Trichosporon [1].

However, these pathogens are increasingly emerging as breakthrough invasive fungal infections (IFI), particularly in hematologic patients who, due to multiple risk factors for invasive fungal disease, are placed on antifungal prophylaxis [2]. Echinocandins, frequently chosen for prophylaxis, inherently select for microorganisms such as Trichosporon, which are intrinsically resistant to these agents [1–6]. Within this genus, Trichosporon asahii accounts for more than half of infections, which are associated with high mortality rates [2,3].

These infections are becoming increasingly frequent and are often inappropriately managed under the diagnosis of treatment-resistant Candida or Aspergillus fungal infection [3,4], leading to delays in diagnosis and, consequently, increased morbidity and mortality among our patients [4].

Specifically, this case is particularly relevant due to its occurrence in the early post–hematopoietic stem cell transplantation period, despite ongoing echinocandin prophylaxis, and the subsequent need to escalate therapy to isavuconazole. These features highlight both the rarity and the clinical complexity of managing emerging Trichosporon asahii infections in pediatric patients with hematologic malignancies.

Case Presentation

The patient was an adolescent female diagnosed with acute myeloid leukemia with NUP214/DEK-CAN fusion, who underwent an allogeneic hematopoietic stem cell transplantation (HSCT) from a haploidentical sibling donor. There was no relevant family history or significant past medical history for the patient. There were no complications during the infusion of hematopoietic stem cells (day 0) or in the preceding days. She had been receiving antifungal prophylaxis with caspofungin since the initiation of conditioning, in accordance with the unit's protocol.

She developed febrile spikes (day +1), prompting collection of blood cultures and initiation of empiric antibiotic therapy with piperacillin-tazobactam, amikacin, and teicoplanin. Although initially afebrile, fever recurred, leading to a switch to meropenem-linezolid (day +3). Chest CT, β-D-glucan, and galactomannan assays were unremarkable, and antibiotic therapy was discontinued after 10 days (day +9).

Forty-eight hours later, high-grade fever recurred with hemodynamic deterioration, necessitating reinitiation of antibiotic therapy (day +11). Given the persistence of fever and after having ruled out a bacterial infection, a fungal infection is suspected as the underlying cause, despite negative test results. Therefore, antifungal therapy is switched from prophylactic to therapeutic dosing, and empiric liposomal amphotericin B therapy was initiated after obtaining blood cultures (day +16).

A total of eight blood cultures-drawn from peripheral blood and multiple intravascular sites-were collected within the following 12 hours (day +16). All were processed according to the protocols of the Spanish Society of Infectious Diseases and Clinical Microbiology, using the Virtuo® system (BioMérieux, Lyon, France). Of these eight cultures, only the one drawn from the peripheral catheter was positive after 37 hours of incubation. Gram staining revealed yeast-like fungal elements, and subculture on appropriate media allowed identification (Trichosporon asahii) and antifungal susceptibility testing. Identification was performed using MALDI Biotyper® mass spectrometry (Bruker Daltonics, Karlsruhe, Germany), and susceptibility testing was conducted with the Sensititre YeastOne® system (Thermo Fisher Scientific, Waltham, USA). Six days later, two additional blood cultures grew the same organism.

In light of the partial clinical improvement and the uncommon nature of the microorganism, a consultation with the microbiology service was requested. Following a review of the literature and drawing upon their experience, they recommended the addition of isavuconazole, among other azoles (day +16). A few days later (day +21) all central venous catheters

were removed. Dissemination workup revealed both pulmonary (Figure 1) (day +32) and splenic involvement (day +35).

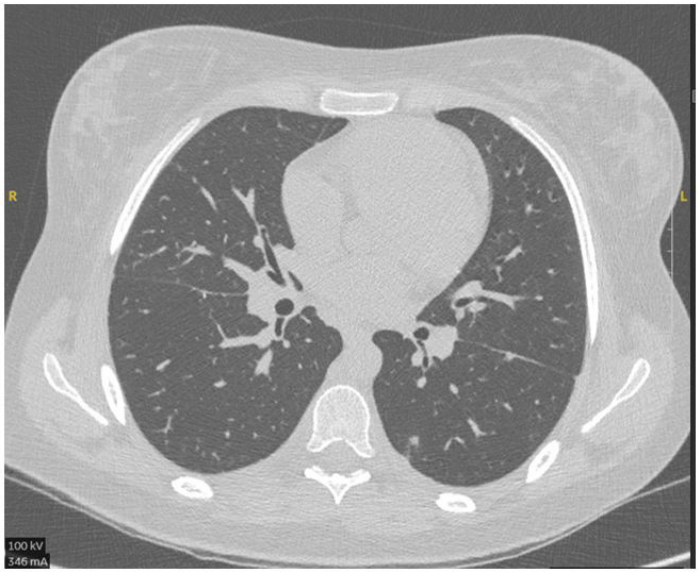

Figure 1: Chest CT scan. Patchy ground-glass opacities in the right upper lobe (RUL) and right lower lobe (RLL). Ground-glass pseudonodule in the superior segment of the left lower lobe (LLL). Small nodule abutting the left pleura and another in the major fissure suggestive of lymphadenopathy. Posterior basal subpleural pseudonodular thickening in the right lung associated with pleuropulmonary strands. Thickening (left pleural effusion) associated with posterior basal atelectasis and pleuropulmonary strands.

Liposomal amphotericin B was discontinued after two weeks of apyrexia, and isavuconazole was stopped on day +126 post-transplant, with the patient remaining asymptomatic.

Conclusion

The genus Trichosporon ranks second in frequency among fungal infections in immunocompromised patients. Within this genus, several species have been identified, with T. asahii being the most common.

It is a yeast-like fungus capable of developing hyphae and pseudohyphae. It is widely distributed in nature and is occasionally found on the skin as well as in the oral and gastrointestinal microbiota. Infection is typically endogenous, arising from such colonization, producing cutaneous disease in immunocompetent individuals and systemic disease in immunocompromised hosts. The most well-known cases occur in oncohematologic patients with profound neutropenia; other presentations include pneumonia, urinary tract infection, brain abscesses, meningitis, and peritonitis [3-7].

Among predisposing factors, hematologic malignancies are of particular relevance, as is the presence of indwelling medical devices [1-3]. There is no specific biomarker; however, galactomannan and β-D-glucan assays may occasionally yield positive results [8]. Macroscopically, the colonies are typically cream-colored with a cerebriform appearance [9], and microscopically, the yeast may produce blastoconidia, arthroconidia, and hyphae [2]. The treatment of choice for fungemia is azole therapy, as this genus is intrinsically resistant to echinocandins [1-6].

This case underscores the importance of a multidisciplinary approach, encouraging close collaboration between pediatric oncologists, microbiology specialists, and, in some instances, adult oncologists. Such coordinated efforts provide broader expertise and should be emphasized as they significantly improve diagnostic accuracy and therapeutic decision-making in these rare but life-threatening infections.

Acknowledgements

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Conflict of Interest

There are none

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