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INTRODUCTION

Trichosporon is a genus of yeast-like fungi. It is perhaps most widely known as the cause of white piedra, a benign superficial mycosis seen in immunocompetent individuals in tropical and subtropical regions. However, the incidence of invasive trichosporonosis has increased in immunocompromised patients, most notably those with hematologic malignancies. The following case illustrates characteristic features of trichosporonosis fungemia.

CASE

A 24-year-old black woman with acute myeloid leukemia (AML) recalcitrant to 2 prior courses of chemotherapy was admitted for high-dose cytarabine. She had neutropenic fever and remained febrile despite standard empiric antimicrobial coverage with piperacillin/tazobactam and anidulafungin. Computed tomography scans for dyspnea and worsening fever revealed paranasal sinus mucosal disease, pulmonary infiltrates, and axillary lymphadenopathy. Anidulafungin was switched to amphotericin to cover potential mucormycosis. A fungal smear endoscopically obtained from the left maxillary sinus revealed budding yeast. Blood cultures confirmed growth of *Trichosporon asahii*, after which her antifungal regimen was changed to fluconazole.

Five days after the first positive blood culture, red-to-violaceous papulo-nodules and pustules developed on the patient's face, neck, trunk, and extremities (Fig 1, *A* and *B*). A skin biopsy and tissue culture were obtained from the forearm. Step sections and a periodic acid-Schiff stain highlighted a small focus of suppurative inflammation along with periodic acid-Schiff–positive spores and pseudohyphae (Fig 2, *A*).

Abbreviation used:

AML: acute myeloid leukemia

A tissue culture confirmed *T. asahii* in the skin. *T. asahii* was visualized on peripheral blood smear (Fig 2, *B*) and grew from 8 separate blood cultures despite treatment with fluconazole. The patient decompensated and was transferred to the medical intensive care unit, at which point voriconazole was initiated. After this change in therapy and slow recovery of her blood counts, the patient improved over 1 month with resolution of her fever, dyspnea, and rash. A repeat bone marrow biopsy confirmed marrow recovery and complete remission of AML.

DISCUSSION

Over the past decade, the taxonomy of the genus *Trichosporon* has been revised on the basis of molecular data. The formerly named *T. beigelii* (or *T. cutaneum*) now corresponds to 6 different species that can lead to invasive fungal infections. These include *T. asahii*, *T. asteroids*, *T. cutaneum*, *T. inkin*, *T. mucoides*, and *T. ovoides*.[1,2] The incidence of invasive trichosporonosis has risen in large part due to the increased use of intensive cytotoxic therapy, allogenic blood stem cell transplantation, and immunosuppressive therapy.[1-3] Most cases present in patients with hematologic malignancy, commonly AML, during periods of profound neutropenia[1,2] In fact, trichosporonosis is second only to *Candida* species as the most frequent cause of fungemia in patients with hematologic malignancy.[1] Most cases occur as a breakthrough infection, despite standard

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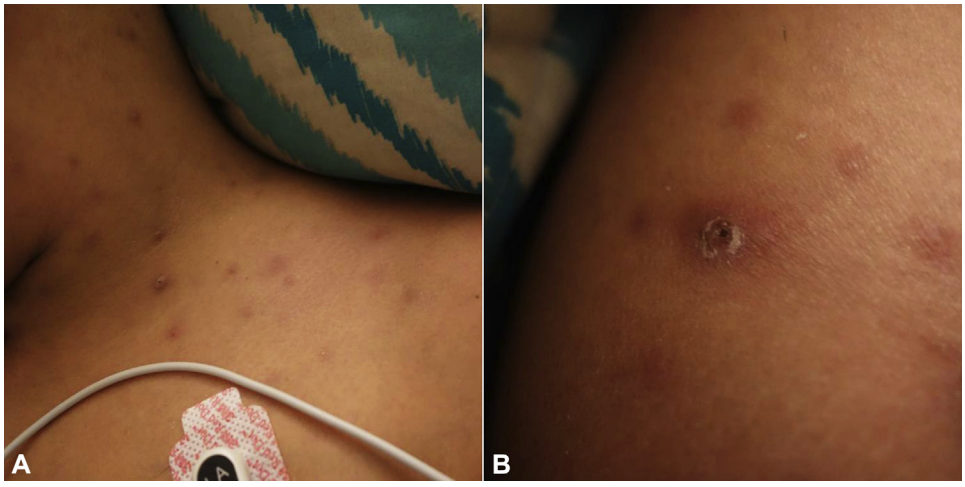

Fig 1. **A** and **B**, Clinical presentation of trichosporonosis. Pink-to-red edematous papules and pustules present on neck and upper chest.

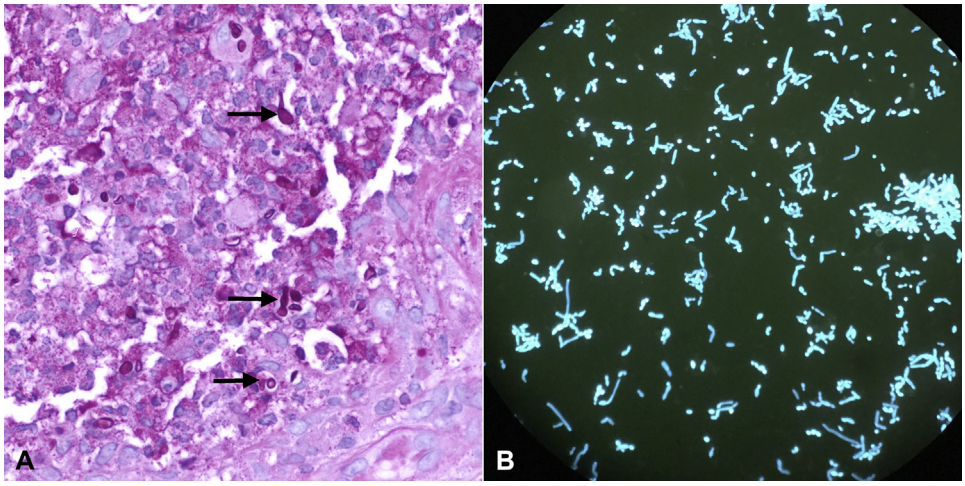

Fig 2. Histopathologic examination of samples from patient with trichosporonosis. **A**, PAS-positive spores and pseudohyphae. **B**, Potassium hydroxide preparation and calcofluor white fungal stain of peripheral blood smear revealed presence of numerous pseudohyphae. (**A**, PAS stain; **B**, calcofluor white stain; original magnifications: **A**, ×400; **B**, ×200.) *PAS*, Periodic acid-Schiff.

prophylactic antifungal regimens.[4] Mortality rates are in excess of 80%.[3]

Dermatologic findings of invasive trichosporonosis might include erythematous papules, bullae, ulcerations, or necrosis.[5] On histopathology, dermal pseudohyphae and yeast forms can be identified.[1,3] Other features include renal failure, pulmonary infiltrates, and chronic liver disease.[1] Trichosporonosis must be distinguished from candidiasis, which shares similar clinical and morphologic features in the disseminated form.

Optimal therapy for disseminated *Trichosporon* is not established. Multidrug resistance has been reported with amphotericin, echinocandins, flucytosine, fluconazole, and itraconazole.[1-3,6] Clinical and in vitro evidence suggests susceptibility to newer triazoles including voriconazole, posaconazole, and ravuconazole.[2,6] Resolution of trichosporonosis was associated with neutrophil recovery, mitigation of hyperglycemia, and use of azole-containing antifungal therapies in a retrospective study of patients with hematologic malignancies.[4]

Dermatologists and dermatopathologists should be aware of the similarities in presentation between invasive trichosporonosis and *Candida* and their differences in treatment susceptibility. Early detection of trichosporonosis via recognition of clinical, histologic, and systemic features can aid in rapid

treatment and improved outcomes for this rare and fatal infection. Prognosis remains poor without immune reconstitution.[1]

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