SKIN

BRIEF ARTICLE

Invasive Trichosporonosis in a Child Following Chemotherapy Induction

Valerie M. Foy[1], Emily McEldrew, DO[2], Chelsea Kesty, MD[2], Kristina Lim, DO[2], Kevin Liu, DO[2], Haider Asad, MD[3], Cynthia Bartus, MD[2]

[1] Philadelphia College of Osteopathic Medicine, Philadelphia, PA
[2] Lehigh Valley Health Network, Department of Dermatology, Allentown, PA
[3] Lehigh Valley Health Network, Department of Pathology, Allentown, PA

ABSTRACT

Trichosporonosis is an opportunistic fungal infection that commonly affects neutropenic, immunocompromised patients. We report a case of invasive trichosporonosis in a child with acute lymphoblastic leukemia following the induction of chemotherapeutic agents.

CASE REPORT

A 2-year-old Caucasian female presented to the hospital with fatigue and dyspnea on exertion. Her lab work revealed leukocytosis, anemia, and thrombocytopenia, and subsequent spinal tap was diagnostic of acute B cell lymphoblastic leukemia. The patient began induction chemotherapy with vincristine and mercaptopurine, together with stress dose hydrocortisone and trimethoprim-sulfamethoxazole. Two weeks later, she developed neutropenia, fevers, port site erythema, and new painful lesions on her legs. She was empirically started on vancomycin, cefepime and micafungin.

On physical exam, there were multiple tender hyperpigmented nodules scattered on the lower extremities (**Figure** 1). An MRI of bilateral lower legs was performed and demonstrated extensive infectious myositis with numerous foci of peripheral ring enhancement, concerning for developing abscesses. An MRI of her lumbar spine revealed multiple lesions, possible fluid collections or abscesses, in the lower posterior paraspinal muscles and subcutaneous soft tissues. A punch biopsy of her lower leg showed deep mixed suppurative and granulomatous inflammation with multiple yeast and hyphal forms (**Figure** 2). The tissue culture was positive for *Trichosporon asahii*. Notably, anaerobic/aerobic culture, acid-fast bacilli culture, blood culture, and bone marrow aspirate were all negative. The patient was started on amphotericin B for treatment of invasive trichosporonosis and her port site was surgically debrided. She was ultimately discharged on oral voriconazole monotherapy and has continued therapy for 8 months.

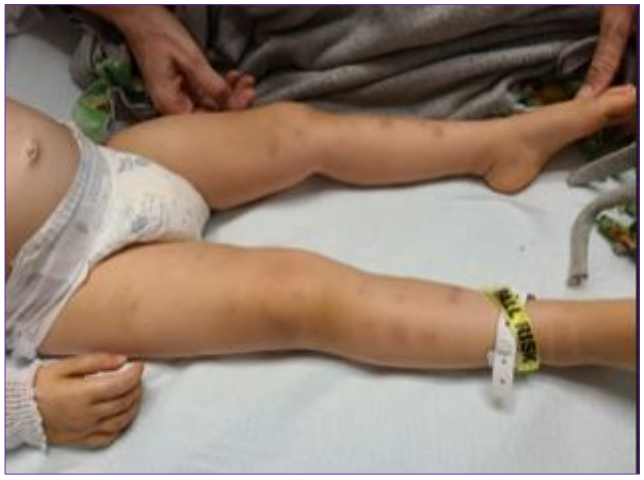

Figure 1. Hyperpigmented nodules scattered across the bilateral lower extremities.

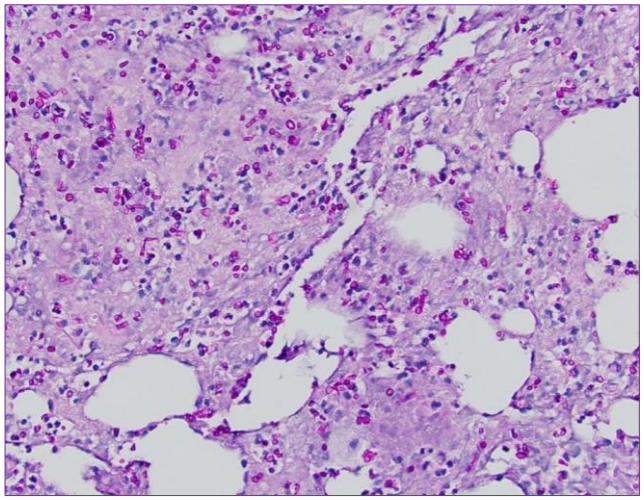

Figure 2. Punch biopsy of lower leg nodules showing deep mixed suppurative and granulomatous inflammation with multiple yeast and hyphal forms.

DISCUSSION

Trichosporonosis is an opportunistic fungal infection caused by *Trichosporon* species, with *T. asahii* most identified as the cause of disseminated infection.[1]

Trichosporon species are basidiomycetous yeast-like fungi that have been known to colonize the skin, gastrointestinal tract, and mucosal surfaces. Trichosporonosis primarily affects immunocompromised patients with neutropenia and may only cause allergic pneumonitis or superficial skin infections in immunocompetent hosts. [3] It is the second most common fungal infection in patients with hematologic malignancy, with Aspergillus being the leading cause.[1] There are several risk factors for invasive disease including solid organ transplant, HIV, extensive burns, invasive medical equipment, chronic kidney disease, cystic fibrosis, and peritoneal dialysis.[1] The mortality rate of disseminated disease is estimated to be 50-80%.[1]

Disseminated trichosporonosis typically presents with prolonged fever unresponsive to antibiotics, pulmonary infiltrates, acute renal failure, and hepatic and/or splenic abscesses.[2] Cutaneous manifestations include painless papules, pustules, nodules, ulcers, exophytic growths, and soft tissue infections. A morbilliform rash has previously been reported in the literature on two separate occasions.[4,5] Other systemic findings include brain abscesses, meningitis, endocarditis, and myositis. Histological examination will reveal budding yeasts that enhance with periodic acid methenamine silver staining; however, the diagnosis should be confirmed with a tissue culture. Blood and urine cultures may also be of additional clinical utility.

Voriconazole is the current drug of choice for disseminated trichosporonosis, as multiple studies suggest it is superior to other azoles, including fluconazole and itraconazole.[2] Other options for treatment include amphotericin B and flucytosine. Notably, echinocandins such as micafungin and caspofungin have no activity against *Trichosporon* species and are therefore not recommended.[2] In fact, careful consideration should be taken in deciding whether to start echinocandin prophylaxis,

given the risk of antifungal pressure selecting for pathogens associated with high mortality.[6] Ultimately, cure of trichosporonosis depends on source control with surgical intervention, adequate antifungal therapy, and reversal of neutropenia.

Conflict of Interest Disclosures: None

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Corresponding Author:
Valerie Foy
Philadelphia College of Osteopathic Medicine
Email: vf8787@pcom.edu

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