

Navigating Trichosporon Asahii Infections in Pediatric Leukemia: First Reported Case in Central and Eastern Europe Case Report and Literature Review

Kamila Mozga¹, Kacper Ponikowski¹, Ewelina Wieczorek¹, Kacper Wrzosek¹, Rafał Burek¹, Paulina Deleszkiewicz², Katarzyna Karska³, Monika Lejman⁴, Joanna Zawitkowska¹ 

¹Student Scientific Society of the Department of Pediatric Hematology, Oncology and Transplantology, Medical University of Lublin, Lublin, Poland; ²Department of Pediatric Hematology, Oncology and Transplantology, University Children's Hospital in Lublin, Lublin, Poland; ³Department of Pediatric Hematology, Oncology and Transplantology, Medical University of Lublin, Lublin, Poland; ⁴Independent Laboratory of Genetic Diagnostics, Medical University of Lublin, Lublin, Poland

Correspondence: Joanna Zawitkowska, Department of Pediatric Hematology, Oncology and Transplantology, Medical University of Lublin, Lublin, Poland, Tel +48 507-365-635, Email joanna.zawitkowska@umlub.edu.pl

Abstract: Invasive fungal infections pose significant challenges in the management of immunocompromised patients, particularly those undergoing treatment for hematologic malignancies. *Trichosporon asahii* is a rare but severe cause of invasive trichosporonosis, associated with mortality rates. Effective management is complicated by its resistance to echinocandins and reduced susceptibility to polyenes, necessitating azole-based therapy. This paper aims to illustrate the diagnostic challenges associated with *Trichosporon* infections, analyze the complexities of treatment, review and synthesize known risk factors, and highlight the need for improved clinical management. It addresses the clinical and therapeutic difficulties involved in diagnosing and treating *Trichosporon asahii* infections in pediatric and adult hemato-oncologic patients. We present the first case of a 14-year-old female with T-cell acute lymphoblastic leukemia who developed a disseminated *Trichosporon asahii* infection during chemotherapy in Central and Eastern Europe. Initial symptoms included joint pain, fever, and neutropenia. The diagnosis was confirmed through synovial fluid and urine cultures. Despite initial treatment with voriconazole (70 days) and liposomal amphotericin B (309 days), the infection progressed, involving the lungs, liver, kidneys, and spleen. The patient transitioned to isavuconazole (232 days intravenous then orally), along with extensive supportive care, which eventually controlled the infection. However, the patient experienced significant complications, including joint contractures and prolonged hospitalization. Maintenance therapy was carefully adjusted to minimize adverse effects while ensuring disease control. The patient was followed monthly through September 2024, resulting in a successful outcome. A literature review highlighted neutropenia, antibiotic use, central venous catheters, and corticosteroid therapy as significant risk factors for invasive trichosporonosis. The diagnostic challenge stems from its resemblance to *Candida* and other yeasts, necessitating advanced methods like matrix-assisted laser desorption/ionization time-of-flight mass spectrometry for accurate identification. Voriconazole remains the first-line treatment, with combination therapy using liposomal amphotericin B as a salvage approach. This case underscores the critical importance of early recognition and targeted management of *Trichosporon* infections in immunocompromised patients. Enhanced diagnostic techniques and tailored antifungal strategies are imperative to improve outcomes. This case provides new insight for pediatric hematology practice by demonstrating that early use of advanced diagnostic methods and timely adjustment to azole-focused antifungal therapy are critical for controlling disseminated *Trichosporon asahii* infections, even in severely immunocompromised children.

Keywords: invasive fungal infections, precursor T-cell acute lymphoblastic leukemia, trichosporon asachii, trichosporonosis, isavuconazole, antifungal therapy

Introduction

Despite advances in medical science and pharmacotherapy, invasive fungal infections (IFI) are a major cause of morbidity and mortality for immunocompromised patients. These infections may present either de novo or as high-risk complications in patients with hematologic malignancies undergoing antifungal therapy.^{1,2} *Trichosporon* species are opportunistic, yeast-like organisms from the Basidiomycota group, commonly found in the environment. They are also part of the human mycobiome, colonizing the skin, perigenital areas, and gastrointestinal tract. However, under certain conditions, it can become pathogenic to humans.³ *Trichosporon* is capable of causing a broad range of infections, including white piedra, hypersensitivity pneumonitis, and, the most severe manifestation, an invasive trichosporonosis (IT) with an alarmingly high mortality rate of approximately 80%.⁴ Disseminated form of trichosporonosis can involve numerous organs, manifesting as conditions such as brain abscess, meningitis, endophthalmitis, pneumonia, lymphadenopathy, endocarditis, arthritis, esophagitis, liver and splenic abscess, or even uterine infections.⁵ *Trichosporon asahii* (*T. asahii*) is the predominant pathogen responsible for IT, most commonly isolated from blood (41%), urine (28%), and surgical wound specimens (12.5%) compared to other *Trichosporon* species.⁶

The occurrence of *T. asahii* infections in the pediatric population is exceptionally uncommon, accounting for only 4% of all documented non-*Candida* fungal infections. The vast majority of reported cases (92%) originate in Asia, underscoring the geographic constraints of current epidemiological data. In the current literature, there is an absence of comparative studies evaluating the incidence of *T. asahii* infections among pediatric patients with leukemia.⁷ IFIs are a common complication in children undergoing cancer treatment. It is the second most frequent yeast infection after candidiasis, causing fungemia in patients with hematologic malignancies.⁸ Pediatric patients with leukemia are at the highest risk of developing IFI. According to current evidence, *T. asahii* presents substantial antifungal resistance, and no standardized, pathogen-specific therapeutic regimen has yet been established. Voriconazole (VOR) remains the recommended first-line agent for invasive trichosporonosis, with an initial dosing of 6 mg/kg intravenously every 12 hours during the first 24–48 hours, followed by 4 mg/kg every 12 hours, or transition to an oral regimen (100–200 mg every 12 hours) for maintenance therapy. To enhance therapeutic efficacy, the addition of amphotericin B (LAmB) may be considered; however, monitoring of the patient's clinical trajectory and emerging antifungal resistance is imperative during the course of treatment.⁹

Herein, we describe a case of a patient with T-cell acute lymphoblastic leukemia (T-ALL) who developed a *T. asahii* infection during chemotherapy. The aim of our study is to highlight the insufficient cases of *T. asahii* infections in pediatric patients with leukemia and provide initial insights into the management and challenges associated with this rare fungal infection in immunocompromised individuals.

Case Presentation

On 26 November 2021, a 14-year-and-4-month-old patient was admitted to the Hematology-Oncology clinic due to an X-ray-identified 124x79x169 mm (RLxAPxCC) mass in the mediastinal region, along with morphological abnormalities showing 31% atypical cells.

Medical history was as follows: a month-long period of weakness, low-grade fevers, fatigue, and occasional chest pain. Since 2015, the patient has been treated for epilepsy and managed with lamotrigine and levetiracetam.

After admission, bone marrow was collected, and T-ALL was diagnosed based on myelogram and immunophenotype.

The patient's family history was notable for epilepsy, and no malignancies were reported. Social and economic conditions were described as stable and favorable. The patient reported spending a significant portion of her free time in the countryside, frequently engaging with animals.

The patient was treated according to the treatment protocol for children and adolescents with T-ALL currently applicable in Poland. The therapy plan for this child consisted of the following: an induction of remission (protocol IA: steroids, vincristine (VCR), daunorubicin (four doses), and pegaspargase L-asparaginase (PEG L-ASP) (two doses); protocol IB: 6-Mercaptopurine (6-MP), high-dose methotrexate (HD-MTX), and methotrexate (MTX) intrathecal (i.th.); consolidation: arabinoside cytosine (ARA-C), cyclophosphamide (CPM), and 6-MP), protocol M (MTX 5 g/m² infusion (four doses), i.th. MTX (four doses), and 6-MP), protocol II (dexamethasone, VCR (four doses), doxorubicin (four

doses), PEG L-ASP (one dose), ARA-C, CPM, and thioguanine), and maintenance therapy (oral 6-MP every day and MTXone day per week).

The child was categorized into a non-high-risk group. Supportive care included intravenous (i.v.) mycamine for antifungal prophylaxis and oral co-trimoxazole to prevent the infection of *Pneumocystis jirovecii* (*P. jirovecii*). Figure 1 shows the progression of antifungal treatment.

The treatment started on November 26, 2021. The patient underwent induction chemotherapy, including Protocols IA and IB, but did not proceed to Protocol M. Chemotherapy was discontinued after 56 days due to multiple complications, including the initiation of dialysis therapy (November 29, 2021), generalized edema with prominent swelling around the ankle joints (December 2, 2021), stomatitis (December 4, 2021), pain in the neck, throat, and limbs (December 7, 2021), infection with *Klebsiella pneumoniae* Extended Spectrum Beta-lactamases positive (ESBL+) (December 12, 2021), significant lower gastrointestinal bleeding (December 22, 2021), and laparotomy performed to investigate the cause of internal bleeding (December 30, 2021), shock and epileptic seizure associated with Posterior Reversible Encephalopathy Syndrome (PRES) (January 1, 2022), asymptomatic COVID-19 infection (January 17, 2022), hyperbilirubinemia (January 31, 2022), and profound bone marrow aplasia (February 5, 2022).

On February 11, 2022 (the 78th day of therapy and the 41st day of treatment with LAmB 3.5 mg/kg body weight daily i.v.), the patient reported severe pain in both knee joints and the right elbow joint, accompanied by a fever of up to 38°C. Physical examination revealed swelling and redness in these areas. Laboratory tests included a complete blood count: WBC $0.07 \times 10^3/\mu\text{L}$, RBC $1.78 \times 10^6/\mu\text{L}$, PCT 2290 ng/mL, and CRP 16.75 mg/dL.

Imaging studies revealed multiple findings. Computed tomography (CT) of the thorax showed nonspecific pulmonary changes. Imaging of the knee joints, including magnetic resonance imaging (MRI), CT, X-ray, and ultrasonography (USG), identified increased joint effusion in both knees with associated inflammatory changes. Abdominal USG detected a significant volume of free fluid in the small pelvis and lower abdomen, measuring approximately 118×59×63 mm. The liver was enlarged (162 mm in the anterior axillary line) but showed no focal lesions. The gallbladder was deformed with a hyperechoic, thickened wall (up to 4.3 mm). The pancreas exhibited mild head enlargement (up to 31 mm), discreetly heterogeneous texture, blurred margins, and mildly increased vascular flow. The kidneys were in their typical locations, with increased cortical echogenicity and dimensions of 119 mm (left kidney) and 118 mm (right kidney) in the long axis. A small anechoic or severely hypoechoic area (12×6 mm) was noted at the upper pole of the right kidney, likely representing fluid within the right adrenal gland. Brain MRI showed the resolution of changes associated with PRES.

Arthrocentesis was performed on the knee joints, and synovial fluid samples were sent for microbiological analysis (Figure 2). After seven days, *T. asahii* was identified. Additional microbial samples were collected from urine, stool,

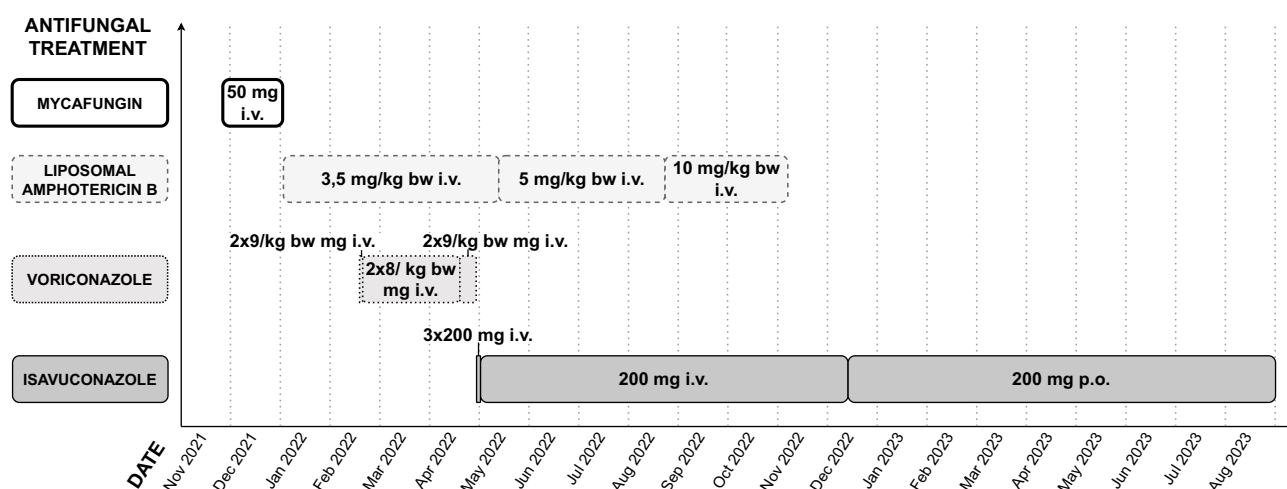


Figure 1 Progress of antifungal treatment. All dosages presented in the figure are administered daily. In the beginning, mycalfungin was included for antifungal prophylaxis. Due to the detection of a subpleural lesion in the right lung on computed tomography, the treatment was changed to LAmB. After the identification of *T. asahii*, VOR was added to the treatment, followed by ISA. Both VOR and ISA were initiated with loading doses. Due to subtherapeutic VOR plasma concentrations, the dosage was subsequently increased. The antifungal therapy was continued until the end of August 2023.



Figure 2 Post-Arthrotomy Right Knee with Intra-Articular Drain and Muscle Wasting. The image shows the right knee joint after arthrotomy (15.02.2022). A drain is placed within the joint. There are stretch marks on the skin above the knee and significant muscle thinning of the thigh and lower leg.

bone marrow, and cerebrospinal fluid (CSF). A urine culture also tested positive for *T. asahii*. VOR (8 mg/kg body weight i.v. once daily) was initiated, slightly reducing inflammatory markers and causing gradual clinical improvement. Cancer treatment continued until day 56 of Protocol I.

On March 23, 2022, a chest CT scan revealed the progression of a fungal infection (Figure 3). However, a follow-up chest CT on April 7, 2022, showed significant regression of the largest pulmonary changes previously described, while smaller changes persisted. New changes were also observed in different areas of the lungs.

Five days later, an abdominal USG identified new focal lesions in the liver and left kidney (Figure 3). This finding prompted an increase in the antifungal medication dosage of LAmB to 5 mg/kg body weight i.v. Despite this adjustment, follow-up imaging conducted a week later revealed further progression of the fungal infection, with additional focal

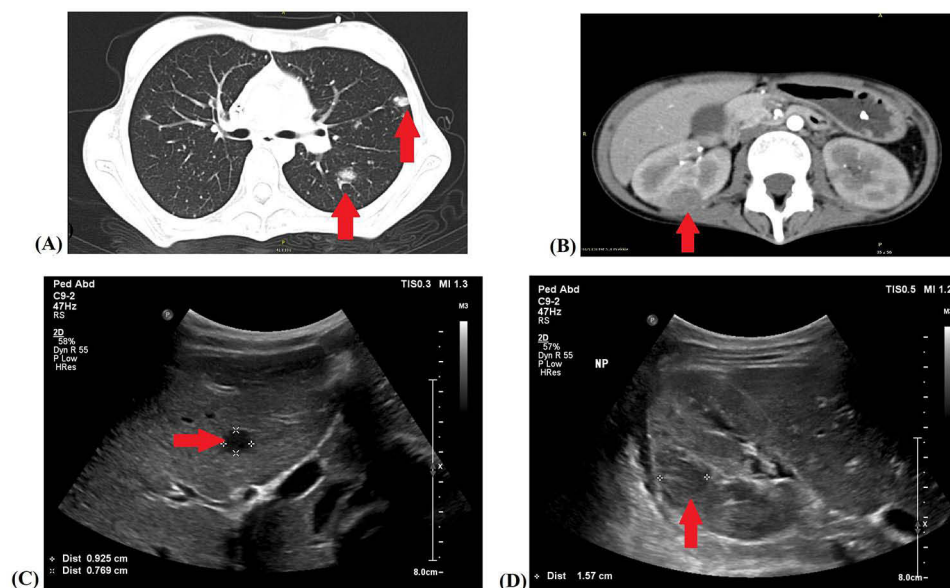


Figure 3 Findings of: axial sections of computed tomography (A and B) and abdominal USG (C and D). (A) Axial CT image of the chest (23.03.2022) shows multiple diffuse nodular infiltrates (red arrows) in both lungs, measuring up to 15 mm in diameter. (B) Axial CT image of the abdomen (13.04.2022) reveals a heterogeneously enhancing lesion (red arrow) in the mid-section of the right kidney, measuring $24 \times 16 \times 18$ mm, with a distorted renal architecture in the region of the lesion. (C) Abdominal USG (23.04.2022) reveals a liver with scattered hypoechoic lesions. The largest lesion (red arrow), measuring $9 \times 8 \times 10$ mm, is located in segment IV. (D) Abdominal USG (23.04.2022) reveals a hypoechoic lesion (red arrow) in the middle part of the right kidney measuring $22 \times 18 \times 16$ mm. Adjacent to it, there is another lesion of similar morphology, approximately 9 mm in diameter.

lesions observed in the lungs, both kidneys, and the spleen (Figure 3). Testing the concentration of VOR in plasma showed it to be below the therapeutic range, which prompted an increase in dosage.

Despite this adjustment, an abdominal USG performed on April 26, 2022, showed no regression of the lesions. Consequently, the decision was made to switch from VOR to isavuconazole (ISA) at a dosage of 200 mg i.v. three times daily for two days, then once daily.

An abdominal USG performed on July 1, 2022, revealed partial regression of focal lesions in the internal organs. Complete resolution of the changes in the kidneys, liver, and spleen was observed on an abdominal USG conducted on November 3, 2022. This prompted the discontinuation of LAmB, while ISA was continued at the same dosage. However, a thoracic CT scan performed one month later still demonstrated multiple disseminated lesions, measuring approximately 4 mm. Blood cultures did not identify any pathogens, including *T. asahii*. Antifungal therapy with ISA was continued, transitioning from intravenous to oral administration (P.O.) on December 14, 2022. This adjustment facilitated the patient's discharge home, enabling the continuation of remission therapy.

The patient required extensive supportive care to address complications during treatment. This included multiple courses of broad-spectrum antibiotic therapy to manage infections and the administration of immunoglobulins to bolster her immune response. Additionally, cryoprecipitate was provided to support her clinical needs, highlighting the critical and multifaceted nature of her care plan. These measures were essential in stabilizing her condition and enabling her to continue her treatment regimen.

During hospitalization, between March and April 2022, the patient developed knee joint contractures, which impair upright mobility. To address this limitation, a structured physical rehabilitation program was initiated. By August 2022, the patient had progressed from wheelchair dependence to ambulating with the assistance of a walker. Attempts at independent walking began around December 2022.

The patient's clinical improvement enabled thoracoscopy for biopsy in July 2023, prompted by suspicion of a persistent pulmonary fungal infection. Two pleural lesions were excised for histopathological and microbiological analysis during the procedure. The surgery and postoperative course were uneventful. Histopathological evaluation revealed fibrous changes, with no evidence of fungal growth. The patient was subsequently discharged in good general condition.

The patient remained under regular follow-up in the Hematology-Oncology Clinic. Chemotherapy treatment began on May 10, 2022, with maintenance therapy involving 6-MP and MTX at a reduced dosage to mitigate the risk of hepatotoxicity observed earlier. Adjustments were made over time, with the 6-MP dosage gradually increasing to 100% by August, while the MTX dosage was adjusted to 75% before being reduced to 50% in September due to poor tolerance. This carefully managed therapy concluded on November 30, 2023, marking the end of an extended period of treatment tailored to balance efficacy with the patient's ability to tolerate the drugs.

In December 2024, bone marrow aspirates were collected, and comprehensive testing, including MDR PCR negative analysis, was performed. The patient continued regular follow-up in the Hematology-Oncology Clinic. At the most recent visit in June 2025, bone marrow sampling was performed, and MDR PCR testing was negative, confirming remission of T-ALL.

A detailed timeline visualization, including disease progression, diagnostic milestones, and treatment interventions, is shown in Figure 4.

To the best of our knowledge, this represents the first documented case of T-ALL remission achieved following only 56 days of chemotherapy.

Review and Discussion

With significant advancements in managing bacterial infections in immunocompromised patients, fungal infections have emerged as a leading cause of morbidity and mortality in this population. While the early use of empiric antibiotics has markedly reduced the mortality rates associated with bacterial infections, fungal infections remain a substantial threat. Among the most frequently identified invasive fungal pathogens are species of *Candida*, *Aspergillus*, *Cryptococcus*, and *Pneumocystis*.¹⁰ Notably, *T. asahii*, once primarily linked to superficial infections, has become a significant opportunistic pathogen responsible for systemic infections in immunocompromised individuals.¹¹ The *T. asahii* species was only

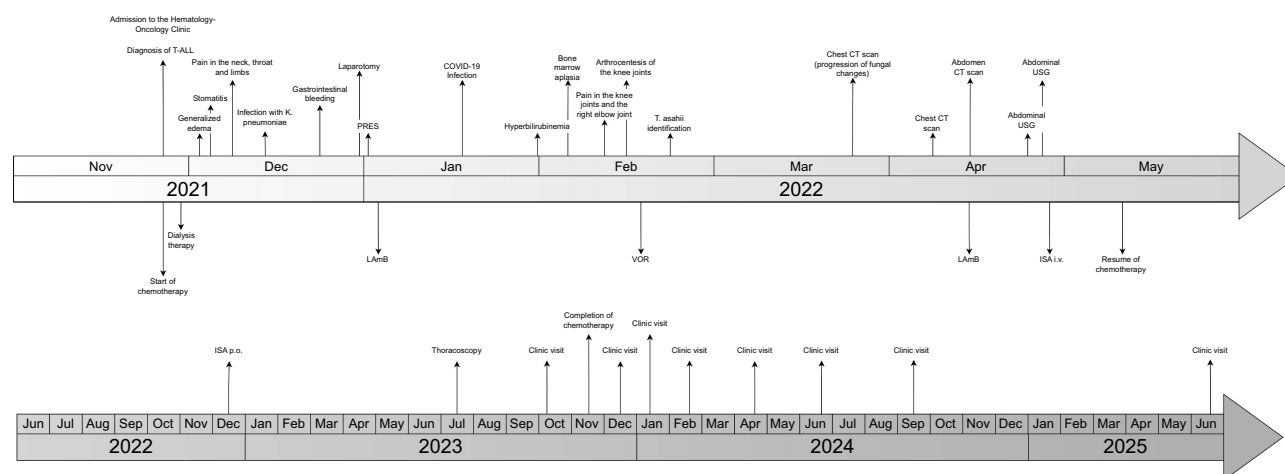


Figure 4 Timeline illustrating the clinical course of the case. Summary of the key stages of the patient's diagnostic and therapeutic process, covering the period from the admission to the Hematology-Oncology Clinic in November 2021 to the last clinic visit in June 2025.

Abbreviations: T-ALL, T-cell acute lymphoblastic leukemia; PRES, Posterior Reversible Encephalopathy Syndrome; LAmB, liposomal amphotericin B; VOR, voriconazole; CT, computed tomography; USG, ultrasonography; ISA, isavuconazole; i.v., intravenous; P.O., oral administration.

distinguished from *Trichosporon beigelii* in 1992 by Gueho et al.¹² This recent differentiation explains the relatively low number of reported infection cases worldwide, especially among pediatric patients. However, the number of documented cases in adults and children has been increasing in recent years. This is likely due to advancements in the diagnosis of infectious diseases.¹³ *Trichosporon* spp. are commonly found in nature, with their distribution primarily concentrated in tropical and temperate regions. In humans, they can sometimes be part of the microbiota in the gastrointestinal tract and oral cavity, as well as temporarily colonize the respiratory system and skin.¹⁴ As a result, endogenous infection might be one of the routes through which IT is acquired.¹⁵ Notably, 77% of reported cases of *T. asahii* infection originate from Asia. Our patient represents the first documented case of this fungal infection in Central and Eastern Europe. Li et al highlight the lack of crucial clinical information, such as the infection process and the sources of pathogens.¹⁶ These details would be crucial for understanding the transmission mechanisms and implementing preventive measures against infection. Interestingly, our patient frequently spends time in the countryside, which could have potentially served as an environmental reservoir for the pathogen. However, due to the absence of specific data on the routes and sources of infection in other case reports, this hypothesis cannot be confirmed.

The primary risk factor for trichosporonosis is neutropenia resulting from cytotoxic chemotherapy. According to a review covering 19 published cases of invasive *T. asahii* infection in pediatric patients with hematological malignancies, neutropenia was an underlying condition in 96% of cases.¹⁷ Broad-spectrum antibiotic use, invasive medical devices such as central venous catheters (CVCs), and intensive care unit hospitalization are also consistently implicated as major contributors.^{6,18} Antibiotic usage and invasive device application were documented in 46.4% and 44.3% of cases, respectively, among the 140 patients diagnosed with *T. asahii* infections.¹⁶ Ruan et al reported that among 19 disseminated trichosporonosis cases, 94.7% involved the usage of CVCs, and 89.5% required broad-spectrum antibiotics.⁶ Similarly, Kontoyannis et al analyzed 17 patients with trichosporonosis and found that 70.6% had a documented history of CVC use.¹⁸ The analysis of six clinical cases within the pediatric population revealed a higher prevalence of 100% for both antibiotic usage and CVC placement among the affected patients. Additional risk factors include prolonged or high-dose corticosteroid therapy exceeding 10 days, organ transplantation, prosthetic valve implantation, and peritoneal dialysis.^{19,20} Our case aligned closely with available literature as it exhibited the majority of common risk factors, including neutropenia, antibiotic exposure, the use of CVC, and prolonged corticosteroid therapy.

In immunosuppressed patients, *T. asahii* infection often begins with acute fever and can progress to multi-organ failure.²⁰ Some cases resemble *Candida* infections,²¹ with cutaneous manifestations such as maculopapular or pustular lesions, which are sometimes necrotic. Skin involvement was reported in 22% of 32 pediatric cases with hematological diseases in a study by Kourtis et al.⁸ Disseminated *Trichosporon* infection in the general population typically affects the

skin, lungs, liver, spleen, brain, and eyes.¹³ Pulmonary involvement is a major contributor to morbidity and mortality.²² Our patient initially exhibited multiple lung nodules, followed by liver and kidney involvement. Unlike most cases, there were no skin lesions, and the first symptom was severe knee pain impairing mobility, a presentation not previously described in the literature.

Because of the rapid clinical deterioration associated with disseminated trichosporonosis, the diagnosis should be considered in febrile individuals with neutropenia and a rash, as early recognition and prompt treatment may improve clinical outcomes.²⁰ *T. asahii* is a urease-positive, nonencapsulated yeast that can form both hyaline septate hyphae and pseudohyphae, as well as cylinder-shaped arthroconidia, distinguishing it from other budding yeasts such as *Candida* and *Cryptococcus* or *Histoplasma capsulatum*.¹⁵ A urease-positive yeast that forms arthroconidia can often be presumptively identified as a *Trichosporon* species.²³ However, direct examination seldom yields a definitive diagnosis, as arthroconidia are rarely observed, and *T. asahii* can resemble *Candida* histologically.⁸ Despite this similarity, *T. asahii* exhibits thinner hyphae and pseudohyphae, and it stains faintly with the Gomori methenamine silver (GMS) stain. Clinically, cutaneous involvement, such as maculopapular or pustular lesions, may suggest trichosporonosis, although similar lesions can also occur in disseminated candidiasis.²⁴ The species of *Trichosporon* can be identified by standard laboratory procedures, such as morphological identification and the API 32C system.²⁵ Reliable identification now relies on sequencing the Internal Transcribed Spacer (ITS) region, the Intergenic Spacer 1 (IGS1) region, or the D1/D2 domain of ribosomal DNA, which are commonly used for species-level identification in various organisms, including fungi and other microorganisms. Matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) offers a validated and dependable alternative.²⁶ It is considered the reference method for species identification of *Trichosporon* isolates.¹³

Although intracellular persistence of *T. asahii* within macrophages has not been clearly demonstrated in the literature, interactions between the pathogen and mononuclear phagocytes appear to play an important role in host–pathogen dynamics. Experimental studies show that macrophages can inhibit the growth of *T. asahii*, and their fungicidal activity increases under stimulation with macrophage colony-stimulating factor (M-CSF), suggesting that these cells actively participate in controlling infection rather than serving as a long-term intracellular reservoir.²⁷ In contrast, the ability of *T. asahii* to form biofilms composed of hyphae, pseudohyphae, and arthroconidia, together with the presence of highly drug-tolerant persister cells within these structures, may create functional reservoirs that sustain infection despite systemic antifungal therapy.^{28–30} Biofilm formation on central venous catheters significantly reduces susceptibility to azoles and contributes to persistent fungemia, often necessitating catheter removal for successful treatment.²⁹ These biological features are likely to contribute to delayed clinical response and dissemination to multiple organs, underscoring the importance of aggressive source control, imaging surveillance, and prolonged antifungal therapy.

A review of the adult cases summarized in Table 1 shows that *T. asahii* infection in adults with ALL is equally uncommon and similarly severe. All reported patients presented with profound neutropenia and rapidly developed bloodstream infection, paralleling the pattern observed in pediatric cases. Treatment regimens in adults were heterogeneous, yet outcomes remained poor, with two of three cases being fatal.^{23,31,32} These findings reinforce the critical role of neutrophil recovery and early azole-based therapy in improving survival and highlight the need for standardized management strategies across age groups.

In patients with acute lymphoblastic leukemia, antifungal prophylaxis is crucial. Among the 14 analyzed cases, seven patients received echinocandins (Table 2). One of them was treated with caspofungin, and six, including our patient, received micafungin. For the remaining eight cases, the antifungal agent used was unspecified. Echinocandins are frequently administered to pediatric leukemia patients to protect against *Candida*, *Aspergillus*, and *P. jirovecii*.³³ However, they lack activity against *Trichosporon*, and severe infections have been documented in cases where echinocandins were used prophylactically.³⁴ This trend was also observed in the cases we reviewed. Therefore, some authors suggested that children with prolonged neutropenia and multiple risk factors, such as mucositis, CVC use, and repeated antibacterial treatment, may benefit from mold-active azole prophylaxis.^{35–37} Evidence remains limited; however, posaconazole prophylaxis has been associated with reduced incidence of breakthrough infections compared with echinocandin-based strategies.^{38,39} Its use in younger patients is constrained by age-related pharmacokinetic variability and formulation limitations. Further pediatric data are needed to define optimal antifungal prophylaxis in high-risk leukemic patients.^{40,41}

Table 1 Characteristics of Adult Patients with *T. asahii* Infection and ALL

Publication	Patient's Age	Patient's Gender	Job	Neutropenia Presence	Isolation Place	Symptoms	Organs Involved	Antifungal Prophylaxis	Empirical Treatment	Targeted Treatment	Treatment Outcome	Country
Chan-Tack 2005 ²³	43	M	Farmer	Present	Blood, urine, skin, bone marrow	Fever, splenomegaly, multiple red papular lesions	Skin, lungs, spleen	NA	LAmB	VOR	Died	USA
Feugray et al 2019 ³¹	66	M	NA	Present	Blood	Fever, skin lesions	Skin	NA	CAS	LAmB + VOR then LAmB then LAmB + ISA	Alive	France
Földes et al 2008 ³²	45	F	NA	Present	Blood	Fever	NA	NA	FLU	VOR	Died	Romania

Abbreviations: M, male; F, female; CAS, caspofungin; VOR, voriconazole; LAmB, amphotericin B; FLU, fluconazole; NA, not applicable.

Table 2 Characteristics of Pediatric Patients with *T. asahii* Infection and ALL

Publication	Patient's Age	Patient's Gender	Neutropenia Presence	Isolation Place	Symptoms	Organs Involved	Antifungal Prophylaxis	Empirical Treatment	Targeted Treatment	Treatment Outcome	Country
Giacomozzi et al 2023 ⁴²	2	M	Present	Blood	Fever	Spleen, kidneys	NA	CAS	VOR then CAS	Alive	Chile
Mahoney et al 2022 ²²	2	F	Present	Skin	Fever, weight loss, rash	Kidneys, liver, lung, and spleen, skin	CAS	LAmB	LAmB +VOR	Died	USA
Present case	14	F	Present	Synovial fluid, urine	Fever, knee, and elbow joints pain	Spleen, kidneys, lungs	MCF	LAmB	VOR + LAmB then ISA + LAmB	Alive	Poland
Galligan et al 2019 ²⁴	18	M	Present	Blood	Paponodular rash	Skin, lungs, spleen	MCF	NA	LAmB + VOR Then VOR	Died	USA
Nguyen et al 2017 ²⁰	10	M	Present	Blood	Asymptomatic rash, fever	Lungs, heart, kidneys, spleen, pancreas, lymph nodes	MCF	LAmB	VOR	Died	USA
Karapinar et al 2015 ⁴³	5	F	Present	Blood	Fever, maculopapular lesions	Skin, lungs	NA	CAS	VOR	Alive	Turkey
Maxfield et al 2015 ⁴⁴	3	F	Present	Blood	Fever, maculopapular, respiratory failure	Lungs, skin	NA	NA	VOR Then MCF Then VOR + LAmB	Died	USA
Thibeault et al 2008 ⁴⁵	11	M	Present	Blood	Fever	Kidneys, spleen, liver, gastrointestinal tract, lungs	NA	LAmB	VOR	Died	Canada
Tsuji et al 2008 ⁴⁶	16	M	Not present	Blood, urine	Fever, Graft versus host disease (gvhd)	NA	NA	MCF	VOR	Died	Japan
Foster et al 2017 ¹⁷	10	M	Present	Blood, lungs	Fever	Heart, spleen, kidney, lymph nodes, pancreas, gallbladder, thymus, adrenal gland	MCF	LAmB	LAmB + VOR	Died	USA

(Continued)

Table 2 (Continued).

Publication	Patient's Age	Patient's Gender	Neutropenia Presence	Isolation Place	Symptoms	Organs Involved	Antifungal Prophylaxis	Empirical Treatment	Targeted Treatment	Treatment Outcome	Country
Foster et al 2017 ¹⁷	15	F	Present	Skin	Fever, subcutaneous nodules	Liver, kidneys	MCF	LAmB	LAmB + VOR	Alive	USA
Foster et al 2017 ¹⁷	8	F	Present	Skin	Fever, skin nodules	Skin, lungs	MCF	LAmB + VOR	VOR	Alive	USA
Antachopoulos et al 2005 ⁴⁷	13	M	Present	Blood, lungs	Fever, paponodular lesions, later liver and kidney dysfunction, respiratory distress	Skin, lungs, kidneys, liver	NA	LAmB	LAmB + VOR	Died	Greece
Itoh et al 1996 ⁴⁸	5	F	Present	Blood, skin	Skin lesions, fever	Skin	NA	NA	MCZ	Died	Japan

Abbreviations: M, male; F, female; CAS, caspofungin; VOR, voriconazole; LAmB, amphotericin B; MCF, micafungin; ISA, isavuconazole; MCZ, miconazole; NA, not applicable.

From the viewpoint of leukemia-directed therapy, our case also highlights the limitations of intensive chemotherapy in profoundly immunocompromised patients. Recent therapeutic advances illustrate how targeted approaches may reduce the cumulative burden of cytotoxic response. For instance, the Phase III Children's Oncology Group AALL1331 trial demonstrated that replacing consolidation chemotherapy with the bispecific T-cell engager blinatumomab (anti-CD19/CD3) in relapsed B-cell precursor ALL significantly improved disease-free survival while reducing severe chemotherapy-related toxicity.^{49,50} Similarly, the anti-CD38 monoclonal antibody daratumumab has transformed outcomes in heavily pretreated multiple myeloma by delivering durable responses with a higher safety profile compared with conventional regimens.^{51,52} Although blinatumomab is specific to CD19-positive B-cell disease and therefore not directly applicable to T-ALL, these therapeutic advances exemplify a broader shift toward targeted immunotherapies capable of preserving antileukemic efficacy while minimizing systemic toxicity. In the future, analogous antigen-directed strategies for T-cell leukemias may allow reduction of the intensity and duration of profoundly myelosuppressive chemotherapy, potentially lowering the risk of life-threatening opportunistic fungal infections such as disseminated *T. asahii*.

Targeted treatment typically involves fad, often in combination with LAmB, as this pairing demonstrates synergistic antifungal effects both in vitro and in vivo.^{53,54} VOR is recommended by the European Society of Clinical Microbiology and Infectious Diseases and the European Confederation of Medical Mycology (ESCMID/ECMM) based on in vitro sensitivity data for *T. asahii* and favorable in vivo clinical outcomes.³⁵ Studies have shown VOR to be more effective than other antifungal agents, including fluconazole, itraconazole, and LAmB.^{6,14,34} The initially measured VOR level of our patient was subtherapeutic, as treatment was initiated with the lowest recommended pediatric dose given the patient's clinical condition and the need to minimize the risk of adverse effects. Considering the substantial pharmacokinetic variability of VOR in children, therapeutic drug monitoring was essential to allow safe dose optimization. Despite dose adjustment and continuation of VOR and LAmB combination therapy, the clinical response remained insufficient, leading to a switch to ISA. This agent, previously untested in pediatric patients with *T. asahii* infection, had demonstrated efficacy in a single case report and in vitro studies. The use of ISA represents a promising alternative to VOR in treating *T. asahii* infections.³¹ In addition to antifungal therapy, removing the central venous catheter and addressing underlying immunosuppression are critical for successful treatment.⁵⁵ Maxfield et al highlighted that patients who recovered from Trichosporon infections were no longer neutropenic during recovery.⁴⁴ Similarly, our patient was brought out of immunosuppression, which likely contributed significantly to the efficacy of antifungal treatments. Among the children with ALL analyzed, our patient is the fifth reported pediatric survivor of *T. asahii* infection globally.

Despite the valuable clinical insights provided by this report, several limitations should be acknowledged. As this is a single case study, the findings cannot be generalized to broader pediatric leukemia populations. Moreover, molecular analysis of *Trichosporon asahii* was not performed, which limits the ability to fully characterize the strain and its antifungal susceptibility profile. Nevertheless, this report highlights the importance of early recognition, targeted antifungal therapy, and the need for multicenter data collection to improve evidence-based management of rare fungal infections in immunocompromised children.

Conclusion

T. asahii has become a significant opportunistic pathogen, particularly in immunocompromised patients. Once linked to superficial infections, it now poses a serious threat due to its potential to cause systemic infections, especially in those undergoing chemotherapy or severe immunosuppression. Early diagnosis and targeted treatment are critical, as trichosporonosis can rapidly worsen. Effective management requires recognizing key risk factors, such as neutropenia, invasive devices, and prior antibiotic use. Advanced molecular and spectrometric techniques are vital for accurate identification, ensuring appropriate therapy. Echinocandins, while common for antifungal prophylaxis, lack activity against Trichosporon, highlighting the need for tailored strategies. VOR, often combined with LAmB, is the treatment of choice, with alternatives like ISA showing promise for resistant cases. Successful outcomes depend on antifungal therapy and managing conditions like neutropenia and immunosuppression. Our pediatric case underscores the importance of a comprehensive, adaptive approach to treating this life-threatening infection.

Ethics and Consent Statement

Written informed consent in two languages was obtained from the parent of the patient for publication. An institutional approval was not required for publishing the case details.

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Disclosure

The authors report no conflicts of interest in this work.

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