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Positive Syphilis Serology With Concurrent Disseminated *Talaromycosis marneffei* in a Patient With HIV: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCD Qihuan Wu
CDG Hong Li
CF Yuwei Xia
EF Fenfang Wang

Department of Infectious Diseases, The Affiliated Xiangshan Hospital of Wenzhou Medical University, Ningbo, Zhejiang, PR China

Corresponding Author: Fenfang Wang, Donggu Road, Dandong Street Ningbo, Zhejiang 315700 PR China
Phone: 86-13738880862, e-mail: wff8750@163.com

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Patient: Male, 37-year-old
Final Diagnosis: Disseminated *Talaromyces marneffei* co-infection with syphilis
Symptoms: Fever • rash
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Rare disease

Background: *Talaromyces marneffei* (TM) infection is an opportunistic infection that typically affects individuals with compromised immune systems. TM is an invasive fungal infection associated with high mortality in patients with advanced human immunodeficiency virus (HIV) disease. Syphilis, caused by *Treponema pallidum*, is a re-emerging infectious disease with diverse clinical manifestations. While primary syphilis often goes unnoticed, secondary syphilis is characterized by systemic symptoms and variable skin lesions, earning it the epithet "the great imitator." In areas where TM is non-endemic, misdiagnosis between these 2 diseases can occur.

Case Report: A 37-year-old male patient visited the dermatology department due to a rash. He was initially diagnosed with secondary syphilis based on positive serological tests for syphilis. However, his symptoms did not improve after ampicillin treatment. Instead, he developed fever and cough. It is regrettable that a skin rash biopsy was not performed to further clarify the patient's condition.

Through targeted next-generation sequencing (tNGS) and blood culture, he was diagnosed with concurrent disseminated TM infection. Despite continuous antifungal therapy, his blood cultures remained positive for 11 weeks. Fortunately, he ultimately had a good outcome.

Conclusions: Early detection results from tNGS facilitate timely clinical diagnosis of patients with HIV co-infected with multiple pathogens. When fungal drug susceptibility testing is not feasible, persistently positive blood culture results can be problematic; however, an extended duration of blood culture positivity may indicate the efficacy of the current treatment. It is essential to ensure an adequate duration of antifungal therapy and conduct a comprehensive post-treatment evaluation, as the current evidence is only derived from single cases. The use of blood culture positivity as a marker for treatment response still requires further validation with a larger number of cases.

Keywords: Talaromyces • Blood Culture • Syphilis
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953459

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Introduction

Talaromyces marneffei (TM), a thermally dimorphic fungus, causes life-threatening systemic mycosis, particularly in immunocompromised individuals [1,2].

In the environment (eg, at 25 °C), TM exists as a mold that produces infectious conidia. Upon inhalation by a susceptible host, the conidia convert into a unique yeast form at body temperature (37 °C), which reproduces by fission (not budding), leading to characteristic sausage-shaped, septate yeast cells visible in tissue specimens [3,4].

Infection typically starts in the lungs following inhalation of conidia. Dissemination occurs hematogenously, primarily affecting the reticuloendothelial system (liver, spleen, lymph nodes, bone marrow). The central role of cell-mediated immunity in host defense is evidenced by the extreme vulnerability of individuals with low CD4+ T cell counts, such as those with advanced HIV infection [5,6]. It is the only member in the genus causing systemic mycosis and is more prevalent in Southeast Asia [7-9].

Even after antifungal treatment, the mortality rate can still reach 30%. TM infection and human immunodeficiency virus (HIV) infection can synergistically lead to a further decline in immune function, resulting in increased mortality [10]. Studies have demonstrated that among patients with TM infection, a minority experience slow fungal clearance after antifungal therapy, leading to substantially prolonged hospital stays and a higher incidence of adverse drug reactions. Furthermore, the underlying mechanisms driving delayed TM eradication remain unclear [11].

Syphilis is an infectious disease caused by the bacterium *T. pallidum*, a microaerophilic spirochete that is transmitted through close contact with infected individuals [12]. Syphilis, often referred to as "the great imitator," frequently leads to misdiagnosis due to its diverse clinical manifestations. It typically presents with macular, maculopapular, or papular lesions, and, although rare, can also cause erythema multiforme-like lesions [13]. As a sexually transmitted disease, syphilis is also frequently observed among people living with HIV. Therefore, the co-occurrence of these 2 infections in people with HIV/AIDS is not rare.

Rapid and accurate diagnosis is crucial for survival, yet it remains challenging due to non-specific symptoms. Here, we report a case of TM that was misdiagnosed as syphilis because of an atypical rash.

Case Report

A 37-year-old man with a rash (**Figure 1**) for 20 days, a sore throat for 6 days, and fever was admitted to our hospital. Before admission, he had sought treatment at a local hospital for rashes that appeared on multiple body sites, including the chest, back, neck, face, and arms. Syphilis serology testing at that hospital returned positive results: TM particle agglutination was positive, and the toluidine red unheated serum test titer was 1: 32. He was considered to have syphilis and was transferred to our dermatology department for intramuscular

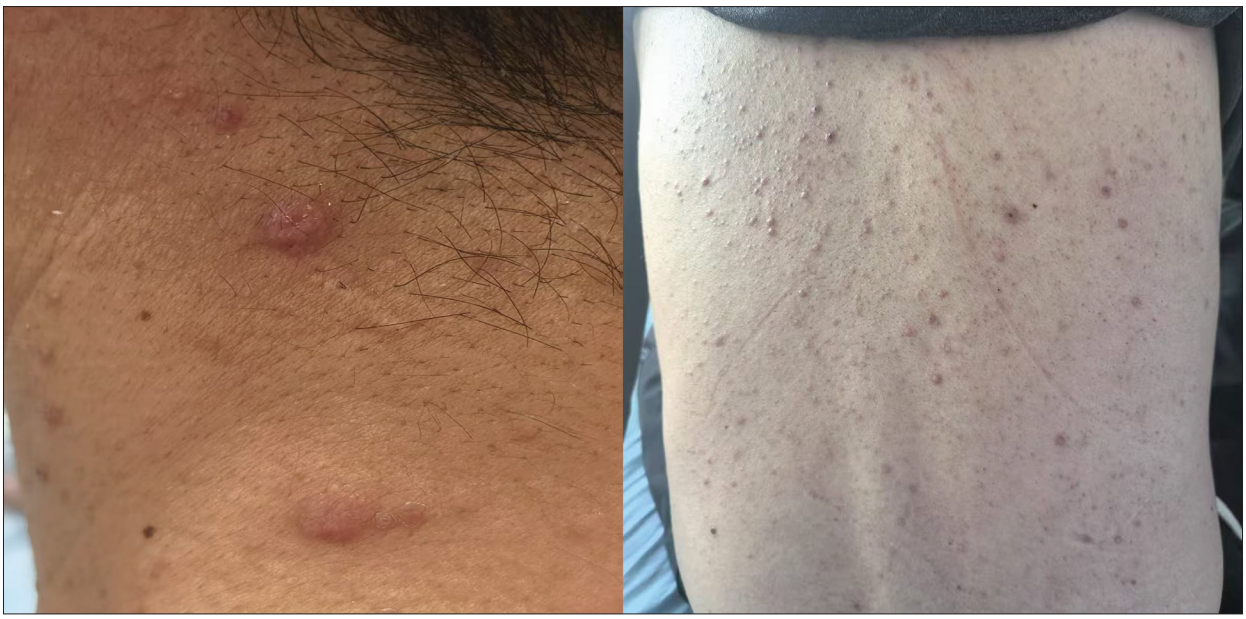

Figure 1. Scattered dark red rashes of varying sizes are visible on back and face, slightly raised above the skin surface, without ulceration or exudation. Some nodules have crusted in the center.

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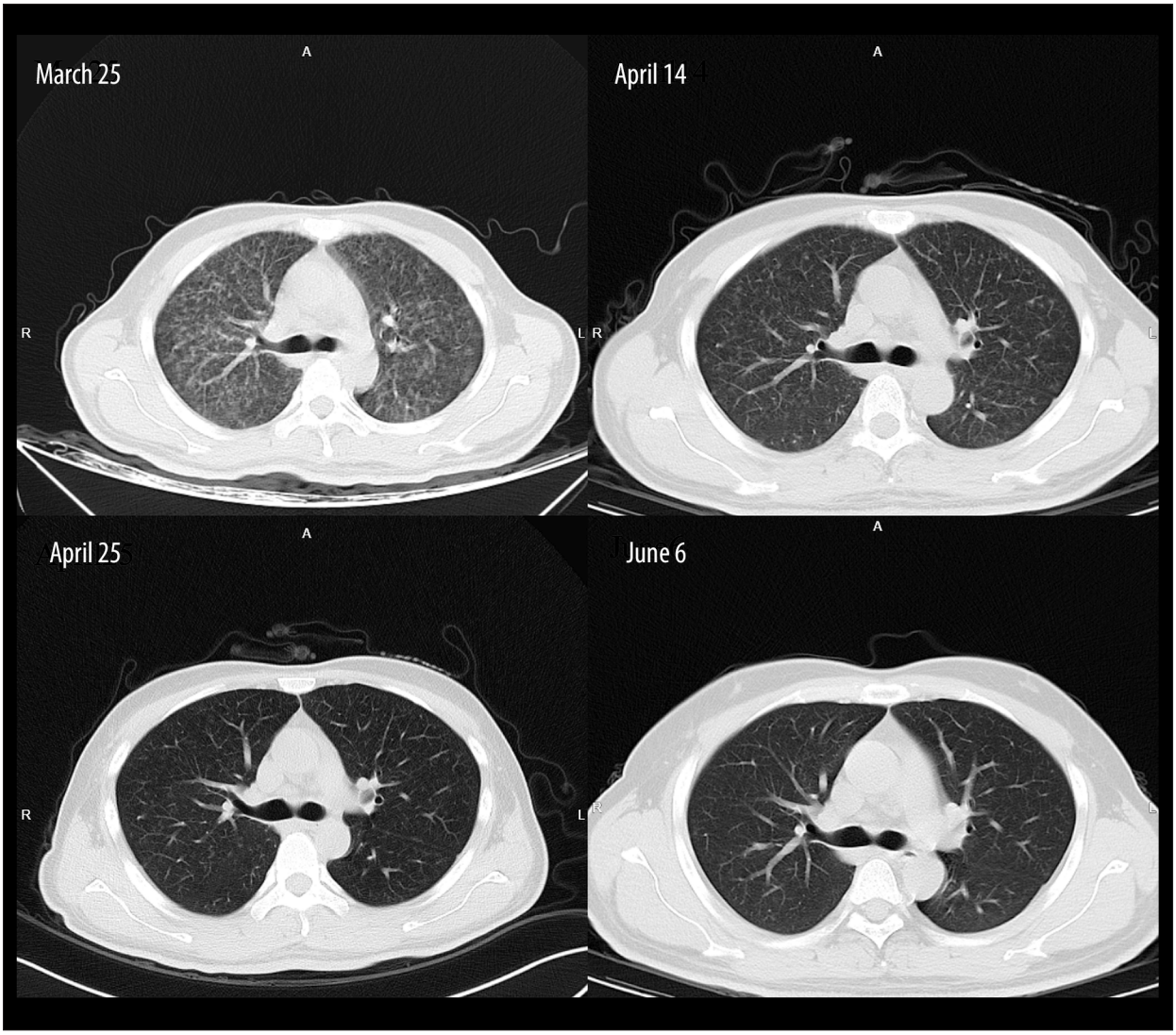

Figure 2. The signs of computed tomography imaging are miliary nodules, interstitial involvement, bilateral lung involvement, and involvement of multiple segments. The pulmonary shadows gradually absorb and dissipate as treatment progresses.

ampicillin therapy. During this period, there was no significant improvement in the rash, which progressively worsened and was accompanied by itching. At the same time, he also had a sore throat and fever. He visited the fever clinic, and preliminary screening showed HIV positivity. Chest computed tomography (CT) indicated diffuse lesions in both lungs (**Figure 2**). Therefore, he was admitted to the infectious disease department on March 25, 2025. He had a feverish appearance with somewhat subdued vitality. Numerous vesicles and white pseudomembranes or patches were observed in the oral cavity and pharynx. Extensive dark red maculopapular eruptions were scattered across the back and face, some with crusting. Coarse breath sounds were heard in both lungs; no dry or wet rales were detected. The second stage of syphilis presents as a disseminated nonpruritic, polymorphic maculopapular rash that involves the trunk, extremities, palms, and soles. Papules with central necrotic umbilication, which resemble molluscum contagiosum lesions, comprise the classic cutaneous manifestations of TM. However, the typical rash seen in our patient made it difficult to differentiate from secondary syphilis.

On March 25, considering the possibility of acquired immunodeficiency syndrome (AIDS)-related fungal infection, fluconazole 0.1g was administered for antifungal treatment. On March 27, purified protein derivative (PPD) was negative. Blood tNGS testing results showed TM and hepatitis B virus, but no *Treponema pallidum* was detected. Treatment was switched to voriconazole 0.2 g q12h for antifungal therapy, and tenofovir alafenamide fumarate was used for hepatitis B virus. Due to an extremely low CD4 cell count, sulfamethoxazole (SMZ) was added for prophylaxis against *Pneumocystis jirovecii* pneumonia (PJP). Concurrently, leukocyte-enhancing

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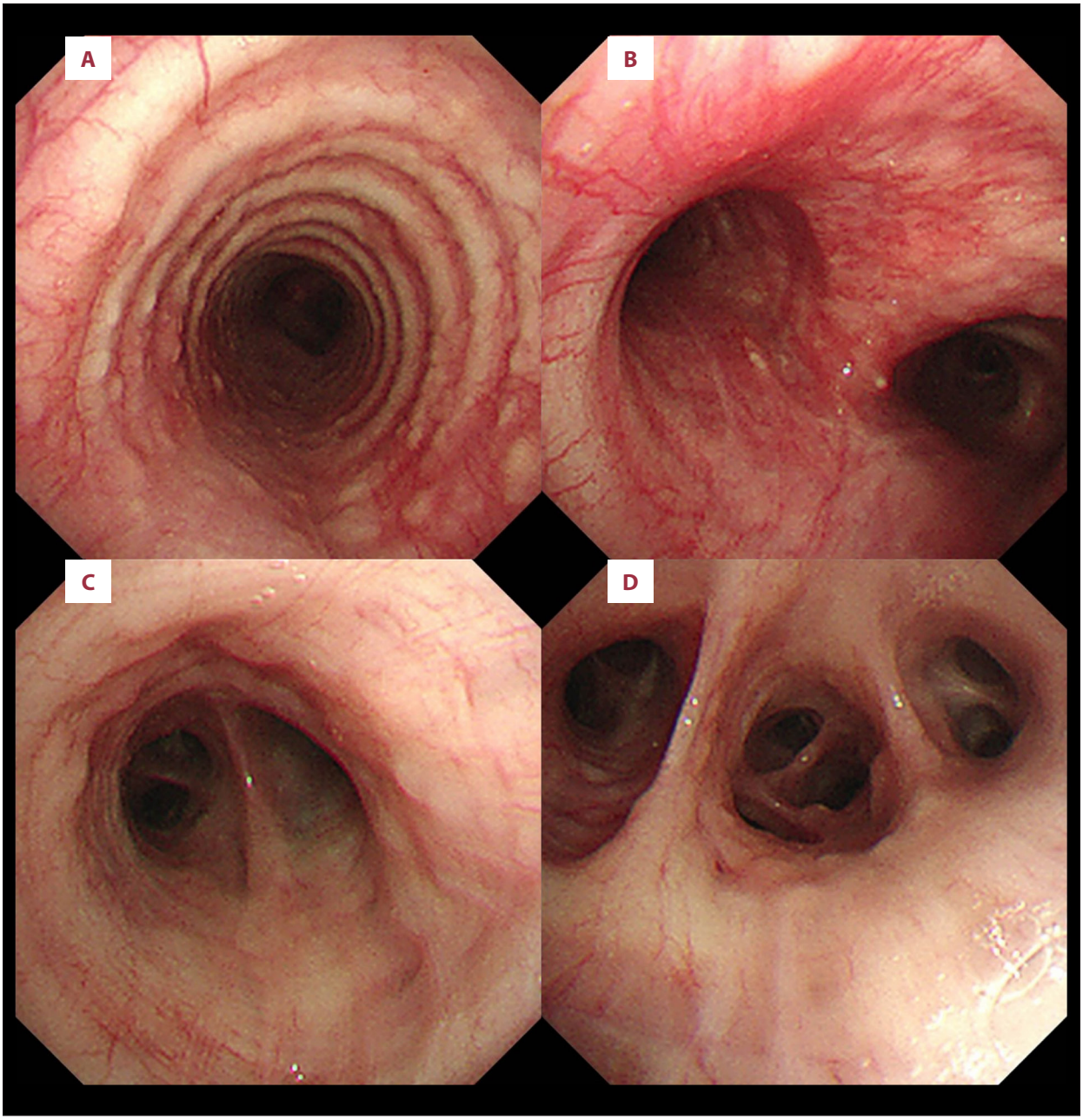

Figure 3. (**A**) Middle part of the trachea, (**B**) Tracheal carina, (**C**) Basal segment of the left lung, (**D**) Basal segment of the right lung. Multiple nodules in the trachea lumen. Congestion and edema of the mucosa of the tracheal carina. Nodular processes are seen in the basal segment of the left lung and the right lung.

agents, hepatoprotective agents, and other symptomatic supportive therapies were administered.

On March 29, bronchoscopy revealed multiple nodular elevations in the trachea and bronchi (**Figure 3**). Bronchoscopic brush biopsy showed no malignant cells. Bronchoscopic lavage fluid TNGS: Human parainfluenza virus (sequence 1615), *Talaromyces marneffei* (sequence 422570), *Pneumocystis jirovecii* (sequence 211284). *Treponema pallidum* was also not detected. The SMZ dosage was adjusted for the treatment of PJP. No *Treponema pallidum* was detected in either the blood TNGS or bronchoscopic lavage fluid TNGS; therefore, we concluded that he had positive syphilis serology with concurrent disseminated TM.

The first blood culture report, dated April 1, 2025, identified TM (**Figure 4**).

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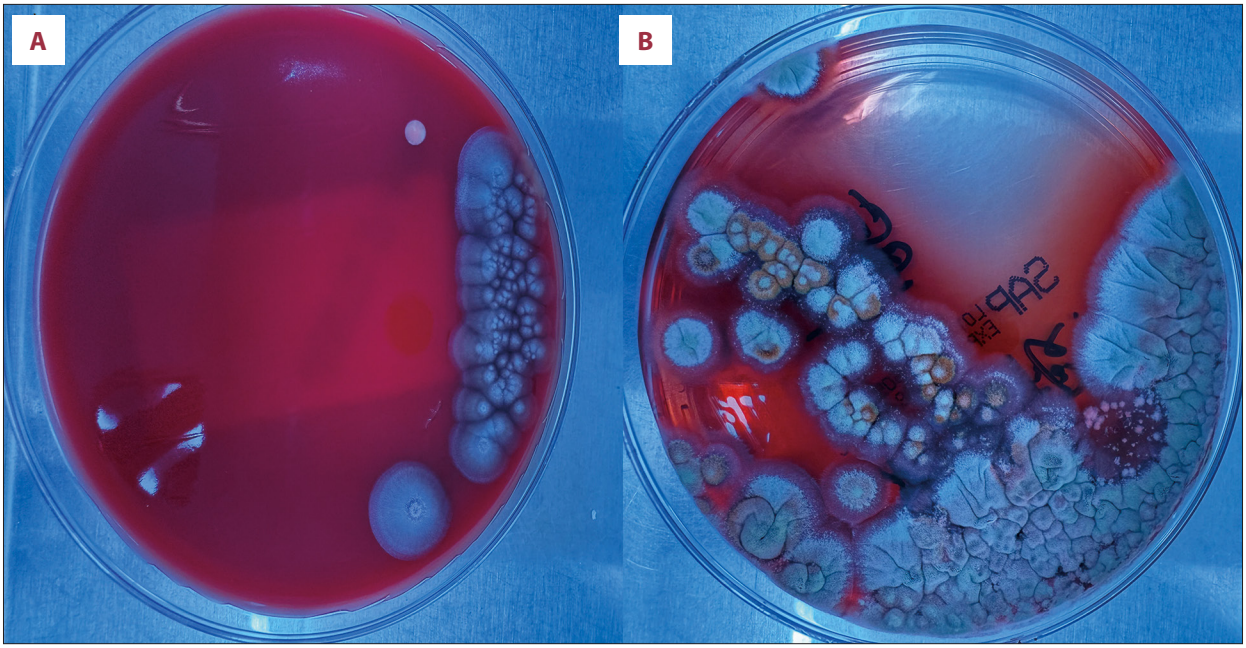

Figure 4. (**A**) At 25 °C, the blood plate shows grayish-white, dry colonies. (**B**) At 35 °C, grayish-white, dry colonies appear on the Sabouraud medium, with some colonies showing yellowish discoloration around the edges.

Blood culture for aerobic bacteria was reported positive at 35.5 hours, and specific bacterial species identification took 7 days. One week after the administration of voriconazole, the erythematous and swollen rash began to subside, and crusting occurred at some ulcerated sites. On April 10, follow-up liver function tests showed improvement, and antiviral therapy with BIKTARVY was added. Subsequently, clinical symptoms showed marked improvement. The patient was discharged after symptoms improved following treatment.

Six days later, he was readmitted with fever, which we considered to be an immune reconstitution reaction. Following anti-infective and anti-inflammatory steroid therapy, the patient's temperature normalized. Blood culture tests during the second admission continued to indicate the presence of TM. Following discharge, he continued taking bictegravir/emtricitabine/tenofovir (BIC/FTC/TAF), voriconazole, and SMZ. Over 20 days later, the patient discontinued voriconazole for 3 days due to lack of medication and was subsequently readmitted with fever. The blood culture remained positive. Consequently, we adjusted the treatment regimen to itraconazole for antifungal therapy. During subsequent outpatient follow-ups, the rash had largely resolved by week 8, leaving only minimal scarring. Unfortunately, the patient did not undergo a skin biopsy, so it is impossible to determine whether the rash was solely caused by secondary syphilis, disseminated talaromycosis, or a combination of both conditions. However, multiple blood culture results remained positive. The last positive report (24 June 2025) occurred approximately 11 weeks after the initial positive blood culture result. The culture finally turned negative on August 5, 2025. The patient achieved a good outcome. The final repeat TRUST test result decreased to 1: 8 (**Table 1**).

Discussion

TM was first isolated from the liver of wild Chinese bamboo rats by Capponi et al [14] in 1956 at the Pasteur Institute in Vietnam. TM mainly invades blood vessels, the mononuclear phagocytic cell system, and other reticuloendothelial systems. First, it invades the lungs from the upper respiratory tract, causing pulmonary infection. Depending on the patient's immune status, it can manifest as localized TM mycosis and disseminated TM disease. Due to the low immune function of AIDS patients, TM infection cannot be restricted, leading to widespread dissemination and mass reproduction of TM, which is highly prone to causing systemic disseminated infection [15-18].

This disease has a distinct regional distribution, occurring in Thailand, Vietnam, and southern China (including Hong Kong, Guangdong, and Guangxi). Its poor prognosis is usually associated with thrombocytopenia, sepsis, metabolic acidosis, and delayed diagnosis [19-22]. CD8 counts can independently predict prognosis regardless of CD4 counts [19]. Elevated serum aspartate aminotransferase (AST) was identified as an independent risk factor for poor outcomes in AIDS patients complicated with TM [23]. Moreover, the poor-outcome group had a significantly lower $CD4^+$ T cell count [24,25].

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Table 1. Combined timeline, clinical event, intervention and laboratory results.

Date	Key findings	Medications/interventions	Tmax (°C)	WBC (10^9/L)	Neutrophil (10^9/L)	Lymphocytes (10^9/L)	HB (g/L)
Mar 25, 2025		Fluconazole initiated (empirical antifungal therapy)	38.4	2.36	2.04	0.16	100
Mar 27, 2025	PPD test: Negative CD4 cell count: Low Blood TNGS: Talaromyces marneffei and hepatitis B virus detected	Fluconazole discontinued; switched to Voriconazole TAF SMZ added Leukocyte-enhancing agents added Hepatoprotective agents added	—	—	—	—	—
Apr 1, 2025	Bronchoscopic lavage fluid TNGS: *Human parainfluenza* virus (1615 reads) *Talaromyces marneffei* (422 570 reads) *Pneumocystis jirovecii* (211 284 reads) First blood culture report: *Talaromyces marneffei* identified (positive)	Voriconazole TAF continued SMZ dosage adjusted	—	—	—	—	—
Apr 7, 2025	Subsequent positive blood culture		—	2.21	1.33	0.47	117
Apr 10, 2025	Follow-up liver function tests: Improved	BIC/FTC/TAF added	—	—	—	—	—
Apr 14, 2025	Subsequent positive blood culture		—	1.20	0.77	0.27	104
Apr 22, 2025	Readmission (fever, nausea/vomiting)	Combination regimen: BIC/FTC/TAF Voriconazole SMZ Anti-inflammatory steroid therapy	39.9	1.43	0.97	0.22	109
May 1, 2025			36.8	2.86	2.13	0.29	101
Jun 14, 2025	Blood culture: Remained positive	Voriconazole discontinued Switched to Itraconazole BIC/FTC/TAF SMZ continued	—	—	—	—	—
Jun 24, 2025	Last positive blood culture		36.7	5.72	3.59	1.58	139
Aug 5, 2025	First negative blood culture		36.8	5.43	3.50	1.05	153

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Table 1 continued. Combined timeline, clinical event, intervention and laboratory results.

Date	PLT (10^9/L)	CRP (mg/L)	PCT (ng/mL)	ALT (U/L)	AST (U/L)	ALB (g/L)	Blood culture	Other key results
Mar 25, 2025	94	39.1	0.94	135	274	25.8	Positive (35.5h)	HBV DNA 1.28×10^8 IU/ml; HIV 210367 cps/ml
Mar 27, 2025	—	—	—	—	—	—	—	—
Apr 1, 2025	—	—	—	—	—	—	—	—
Apr 7, 2025	111	2.9	0.14	172	369	29.7	Positive (50h)	GM 4.62 pg/ml
Apr 10, 2025	—	—	—	—	—	—	—	—
Apr 14, 2025	135	1.4	—	99	148	29.0	Positive (61h)	GM 4.49 pg/ml
Apr 22, 2025	53	114.9	0.56	30	46	35.0	Positive (73h)	G test 388.17 pg/ml; GM 4.97 pg/ml; CD4+/CD8+ 0.02
May 1, 2025	206	12	0.08	65	56	31.1	Positive (104h)	—
Jun 14, 2025	—	—	—	—	—	—	—	—
Jun 24, 2025	228	0.5	—	274	124	44.8	Positive (146h)	G test < 37.5 pg/ml
Aug 5, 2025	215	—	—	17	19	46.3	Negative	HBV DNA 1.55×10^5 IU/ml; CD4+/CD8+ 0.13

For physicians in non-endemic areas, diagnosing and treating TM is a huge challenge. Due to insufficient physician understanding of the disease, some patients do not have typical rashes and only show lung disease shadows on CT scans, which can easily be misdiagnosed as bacteriologically-negative pulmonary tuberculosis, resulting in delayed treatment and a poor prognosis. Therefore, for HIV-infected individuals, routine monitoring of $CD4^+$ T cells and HIV viral load is essential. When CD4 levels fall below 100 cells/μL, vigilance for TM infection should be exercised, and early screening for galactomannan (GM) and gene sequencing is recommended.

Our patient presented with a rash as the initial symptom at the local hospital's dermatology department. Due to the lack of a typical umbilical pit-like rash and positive syphilis antibodies, secondary syphilis was initially considered. After 2 treatments with penicillin, the rash continued to progress. At the same time, he also had throat pain accompanied by herpes, low-grade fever, and other symptoms. The initial HIV screening result was positive, and TM was suspected based on the rash. Blood samples and bronchoalveolar lavage fluid were immediately sent for tNGS examination, and the diagnosis was AIDS combined with disseminated TM infection.

The diagnosis of this disease is based on blood culture as the gold standard. However, due to its slow growth and the long time needed for positive blood culture reports, there may be delays in the treatment of some critically ill patients. A previous report indicated that 24 (38.1%) patients were misdiagnosed as having pulmonary tuberculosis and 5 (7.9%) as having bacterial pneumonia, with a 105 days median time from onset to confirmed diagnosis (range, 11-912 days) [22]. To improve diagnostic efficiency, molecular methods such as polymerase chain reaction and metagenomic next-generation sequencing can be used to detect TM DNA, helping clinicians obtain laboratory evidence earlier and initiate timely treatment [26,27]. Compared to conventional methods, TNGS combines multiplex polymerase chain reaction technology and hybrid capture technology, delivering higher detection rates for mycobacteria, fungi, and rare pathogens [28]. In March 2024, the World Health Organization's latest tuberculosis diagnostic guidelines first included TNGS as a recommended method for *Mycobacterium tuberculosis* molecular drug susceptibility testing. This landmark recognition signals international endorsement of tNGS technology and provides an important reference framework for its application in diagnosing other invasive fungi such as TM [29].

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Currently, there is no consensus on the treatment of TM mycosis. Amphotericin B (AMB) followed by itraconazole (ITRA) is the main treatment strategy for talaromycosis [1], and voriconazole (VOR) is regarded as an alternative in patients with AMB intolerance [30,31]. However, clinical case studies on TM bloodstream infection are scarce [32]. For patients with persistent positive blood cultures, the efficacy of antifungal therapy is still unclear.

Currently, in vitro antifungal susceptibility testing for this fungus is not widely applied. Most hospitals only provide species identification results and cannot perform in vitro susceptibility testing, especially in areas where this disease is not endemic. For immunocompromised patients with concurrent infections by multiple pathogens who present with recurrent fever and delayed clearance of TM in blood, the lack of in vitro antifungal susceptibility testing can lead clinicians to have little confidence in the current antifungal treatment regimen. If there is an infection with drug-resistant strains, an incorrect treatment regimen can result in poor patient outcomes.

Persistent positive blood cultures can correspond to a high mortality rate, which is associated with elevated minimum inhibitory concentrations (MIC) of antifungal drugs in patients with AIDS complicated by TM infection [33,34].

Fifty percent of patients sterilized their bloodstream by 83.16 hours (range, 13-264 hours). A higher initial fungal burden was associated with longer time to sterilization [35]. The study demonstrated that among 190 patients with TM bloodstream infection, very few (5/190) exhibited delayed blood culture conversion to negative for more than 6 weeks, regardless of the antimicrobial therapy used. In the treatment of fungal infections, it is important to pay attention to the MIC of antifungal agents. Persistent blood culture positivity for over 2 weeks may indicate a higher MIC for fluconazole and voriconazole. Furthermore, when there is a poor response to treatment for talaromycosis in patients with AIDS, attention should be paid to the resistance of *T. marneffei* to antifungal drugs. It is also crucial to pay attention to the timing of antiretroviral therapy (ART) initiation. If ART is started too early (before TM infection is controlled), the immune system recovers rapidly, triggering a severe inflammatory response to TM and potentially inducing invasive respiratory syndrome. Therefore, in clinical practice, antifungal induction therapy is typically used first, followed by ART once the infection is initially under control, thereby balancing immune reconstruction with inflammatory risks. In our patient, we noticed that despite recurrent fever and persistent positive blood cultures, the time to blood culture positivity increased during antifungal treatment. This discovery gave us sufficient confidence throughout this lengthy treatment process, and it may potentially be regarded as one of the indicators for effective antifungal therapy in the future.

Conclusions

In summary, some specialists may lack sufficient knowledge of uncommon diseases outside their own field, which limits their clinical reasoning to some extent. The present case report shows that when rashes or systemic symptoms have atypical progression or fail to achieve the expected therapeutic response, it is necessary to re-evaluate the diagnosis for immunocompromised patients. Patients with syphilis, especially those with atypical rashes or poor treatment responses, should be tested for HIV simultaneously. For patients infected with HIV, a comprehensive assessment of their status regarding opportunistic infections is crucial. This can reduce or avoid misdiagnosis or missed diagnosis of these fungal infections, thereby preventing irreversible adverse outcomes. For immunocompromised patients suspected to have a fungal infection, TNGS testing is helpful for early diagnosis and earlier initiation of appropriate medication to improve prognosis compared to traditional pathogen culture.

The course of anti-TM treatment includes an induction period, a consolidation period, and a maintenance period. The total treatment duration for AIDS patients typically exceeds 6 months, and for severe cases or those with bone and central nervous system involvement it extends to over 1 year. The timing of treatment discontinuation for maintenance therapy is determined by $CD4^+$ T cell counts and HIV viral load. Close monitoring of immune parameters, mycological examinations, imaging studies, and organ function is essential. Standardized follow-up enables early detection of relapse, invasive respiratory syndrome, and adverse drug reactions, thereby improving the long-term prognosis.

In primary hospitals lacking relevant fungal drug susceptibility testing, can the time required for blood culture results to turn negative serve as a reference for clinicians? The present report involves only 1 case and lacks antifungal drug susceptibility testing data, which makes it impossible to draw any causal conclusions. Further research is needed to verify the hypothesis that using the time required for blood cultures to turn negative can guide clinical treatment.

Institution Where Work Was Done

The Affiliated Xiangshan Hospital of Wenzhou Medical University, Ningbo, Zhejiang, PR China.

Patient Permission/Consent Declarations

Written informed consent was obtained from the patient.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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