

Talaromyces marneffei Infection in Non-Endemic Areas: Two Case Reports, Diagnostic Insights, and Literature Review

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Purpose: Talaromycosis is an invasive fungal infection caused by the pathogenic fungus *Talaromyces marneffei* (*T. marneffei*), prevalent in Southeast Asia and Southern China. This disease is rare in non-endemic areas and primarily affects the respiratory system with atypical manifestations. Therefore, the condition is often misdiagnosed as other respiratory infections. This article presents two cases of talaromycosis in non-endemic areas, aiming to provide diagnostic and therapeutic references for this rare fungal infection.

Case Presentation: Two cases of talaromycosis in non-endemic areas were retrospectively analyzed. The first case involved a human immunodeficiency virus (HIV)-infected patient with tracheobronchial talaromycosis. Bronchoscopy revealed multiple nodular neoplasms in the trachea and bilateral main bronchi. Histopathological examination of the biopsy tissue showed histiocyte aggregation and intracellular pathogens. *T. marneffei* was identified by microbial culture of bronchoalveolar lavage fluid (BALF). The second case was an HIV-negative patient with pulmonary talaromycosis. Bronchoscopy revealed mucosal congestion and edema, and *T. marneffei* was detected by metagenomics next-generation sequencing (mNGS) of BALF.

Conclusion: Talaromycosis warrants more clinical attention in both HIV-negative individuals and non-endemic areas. In addition, clinicians should improve diagnostic recognition of this disease for timely management. Bronchoscopy combined with mNGS can facilitate early diagnosis of talaromycosis, particularly in culture-negative cases where conventional methods fail. This strategy directly addresses a major diagnostic challenge and improves patient prognosis.

Keywords: talaromycosis, bronchoscopy, metagenomics next-generation sequencing

Introduction

Talaromycosis, caused by the thermally dimorphic fungus *Talaromyces marneffei* (*T. marneffei*), is a life-threatening fungal infection endemic to tropical and subtropical regions of Asia, including China, Thailand, Vietnam, and India.¹ This disease poses a substantial public health burden, with an estimated 17,300 cases and 4,900 associated deaths occur annually.² Previous studies have indicated that the total mortality rate of talaromycosis is 22.7% in endemic areas, compared to 15.2% in non-endemic areas.³ Although the prevalence of talaromycosis in the general population is unknown, a meta-analysis⁴ documented a pooled prevalence of 3.6% among people living with Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome (HIV/AIDS) in Asia. Vietnam reported the highest prevalence (6.4%), followed by Thailand (3.9%), China (3.3%), India (3.2%), and Malaysia (2.1%). In China, the prevalence of talaromycosis in southern regions (15.0%) is significantly higher than that in other parts of the country (0.3%). Notably, approximately 99.4% of talaromycosis cases in China are concentrated in the southern regions, predominantly in Guangxi (42.8%) and Guangdong (40.6%) provinces.⁵

Traditionally, talaromycosis has been considered an endemic mycosis. However, increasing population mobility has led to a growing number of cases being reported in non-endemic areas.⁶ Talaromycosis has been documented across 21

provinces and cities in China and reported in 34 countries globally.⁷ In addition, while talaromycosis primarily affects HIV-positive patients, its incidence is rising among HIV-negative individuals, largely attributable to the increased use of organ transplantation, novel anti-cancer targeted therapies, and immunosuppressive drugs.⁸ Consequently, clinicians outside traditional endemic areas must recognize the changing epidemiology of talaromycosis and update their prevention and diagnostic strategies accordingly. For instance, prompt screening for *T. marneffei* in high-risk individuals—such as returning travelers from endemic areas or immunocompromised patients—is crucial for early diagnosis and improved prognosis.

Currently, clinical studies and reports on talaromycosis in non-endemic areas and HIV-negative individuals are still limited, as well as a lack of established standard antifungal regimens for this patient population. Given this limited data, further research in these settings is needed. This retrospective study presents and analyzes two cases of talaromycosis from non-endemic areas. The first case involved an HIV-positive patient with tracheobronchial involvement; the other talaromycosis case was an HIV-negative patient diagnosed by metagenomics next-generation sequencing (mNGS). Our findings aim to provide valuable diagnostic and therapeutic references for managing this disease in non-endemic areas.

Case Presentation

Case 1

A 43-year-old female who previously resided in southern China for several years presented with a persistent dry cough for over two months. Initial chest computed tomography (CT) was performed on May 7, 2025, revealing bilateral diffuse nodular lesions, left hilar enlargement, and mediastinal lymphadenopathy. One week later, the patient developed a fever with a peak temperature of 39°C. The patient was hospitalized at another institution (medical records unavailable), and routine preoperative infectious disease screening revealed a positive result for HIV antibody testing. The patient received empirical treatment with oral antibiotics (cefixime and moxifloxacin) and antifungal therapy (fluconazole). The treatment effectively resolved the fever, but the cough remained. Traditional Chinese herbal medicine was self-administered for 10 days, with no improvement in cough. The patient was admitted to our hospital on June 16 due to a worsening cough accompanied by new-onset sputum production for three days. Physical examination on admission revealed palpable enlarged lymph nodes in the right cervical region, and bilateral moist rales on lung auscultation. The laboratory findings are summarized in Table 1. HIV antibody testing was positive, with a signal-to-cutoff ratio of 5.07 S/CO (normal range 0–1), and an HIV viral load of 1292 copies/mL (lower limit of detection of 20 copies/mL). Contrast-enhanced CT

Table 1 Laboratory Examination Results of the Two Cases

Test Items (units)	Case 1	Case 2	Reference Value
WBC ($10^9/L$)	8.53	7.76	3.5–9.5
HGB (g/L)	100	140	Female: 115–150
PLT ($10^9/L$)	334	206	125–350
NEU ($10^9/L$)	7.63	4.98	1.8–6.3
LYM ($10^9/L$)	0.49	1.38	1.1–3.2
ESR (mm/h)	113	73	0–15
CRP (mg/L)	98.1	31.2	0–6
ALT (U/L)	37.2	13.8	9–50
AST (U/L)	196.7	20.2	15–40
LDH (IU/L)	370.3	245.4	120–250
ALB (g/L)	33.09	45.73	40–55
GLO (g/L)	40.06	29.74	20–40
Cr ($\mu\text{mol/L}$)	47.6	44	Female: 41–81
Fib (g/L)	5.19	5.25	2–4
CD4 ⁺ T (cells/ μL)	28	ND	555–1440
D-dimmer ($\mu\text{g/mL}$)	10.81	0.49	<0.5

(Continued)

Table 1 (Continued).

Test Items (units)	Case 1	Case 2	Reference Value
G-test (pg/mL)	166.31	<37.5	<90
GM-test (ug/L)	ND	0.18	<0.85
HIV viral load (copies/mL)	1292	ND	<20

Abbreviations: ALB, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CD4⁺T, CD4⁺T lymphocyte count; Cr, serum creatinine; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; Fib, Fibrinogen; G test, serum (1,3)-beta-D-glucan assay; GM test, galactomannan assay; GLO, serum globulin; LDH, lactate dehydrogenase; HGB, hemoglobin; LYM, lymphocyte; ND, Not Done; NEU, neutrophils; PLT, platelet; WBC, white blood cell.

(Figure 1) demonstrated persistent bilateral miliary nodules, left hilar enlargement, and enlarged mediastinal lymph nodes. Bronchoscopy performed on June 18 revealed multiple scattered nodular neoplastic lesions with contact-induced bleeding in the trachea, left main bronchus, segmental bronchi, and the orifice of the right main bronchus. In addition, mucosal congestion and edema were observed in the left main bronchus, as well as purulent secretions in the right bronchial lumen. Diagnostic procedures were conducted in the left bronchus. Specifically, bronchoalveolar lavage fluid (BALF) was collected for microbiological analysis, bronchial brushing was performed for smear microscopy and liquid-based cytology (LBC), and suspected neoplastic tissues were biopsied for histopathological examination. The results were as follows: *Mycobacterium tuberculosis* DNA (TB-DNA) detection and multiplex infectious pathogens detection were negative, smear microscopy and microbial culture of two BALF samples were positive for *T. marneffei*, and LBC examination (Figure 2A and B) revealed intracellular blue-staining granular organisms. Histopathological examination (Figure 2D–F) of the biopsy tissue revealed chronic squamous mucosal inflammation with histiocyte aggregation, featuring numerous spherical to oval pathogens within the histiocyte cytoplasm. The specific staining results were: Acid-fast bacilli stain (-), Periodic acid-Schiff stain (PAS; +), and Grocott's methenamine silver stain (GMS; +). The patient was diagnosed with talaromycosis, and amphotericin B cholesteryl sulfate complex (150 mg × 10 days, daily through an intravenous pump) was administered starting June 25 as induction therapy. However, the therapy was discontinued due to refractory hypokalemia, which was attributed to drug-related adverse effects. Following treatment cessation and potassium supplementation, the serum potassium levels returned to within the reference range. The treatment regimen was adjusted to oral voriconazole (200 mg twice daily) for continued induction therapy. Antiretroviral therapy (bictegravir/emtricitabine/tenofovir alafenamide, one tablet per day) was administered concomitantly with antifungal therapy. The clinical symptoms improved, but the patient declined a follow-up chest CT scan to evaluate pulmonary lesions. The patient was discharged on July 15 and received oral voriconazole as consolidation therapy at a dose of 200 mg twice daily. At the time of writing this article, the patient remains asymptomatic and is being followed up.

Case 2

A 60-year-old female with a 2-year history of chronic obstructive pulmonary disease on maintenance inhaled corticosteroid therapy presented with worsening cough with sputum production, chest tightness, and wheezing for two weeks. The patient was hospitalized at another institution and was administered intravenous antibiotics and corticosteroid therapy. Her symptoms transiently improved, followed by rapid symptom relapse and clinical deterioration. The patient was admitted to our hospital on March 20, 2025. Initial laboratory findings are detailed in Table 1. A chest CT scan (Figure 3A–C) performed on March 22 revealed multiple patchy and nodular opacities and enlarged mediastinal lymph nodes. Bronchoscopy performed on March 27 revealed mucosal congestion, edema, and purulent secretions. BALF samples were collected for TB-DNA detection, multiplex infectious pathogen detection, and microbial culture, which yielded negative results. No pathogen was identified on smear microscopy. LBC examination indicated abundant neutrophils with the absence of malignant cells. BALF sample was subjected to mNGS, yielding positive results for *T. marneffei* (10 sequence reads), *Rhinovirus C* (42 sequence reads), and *Epstein-Barr virus* (11447 sequence reads). Based on the radiographic findings, *T. marneffei* was considered the most likely causative pathogen after excluding other

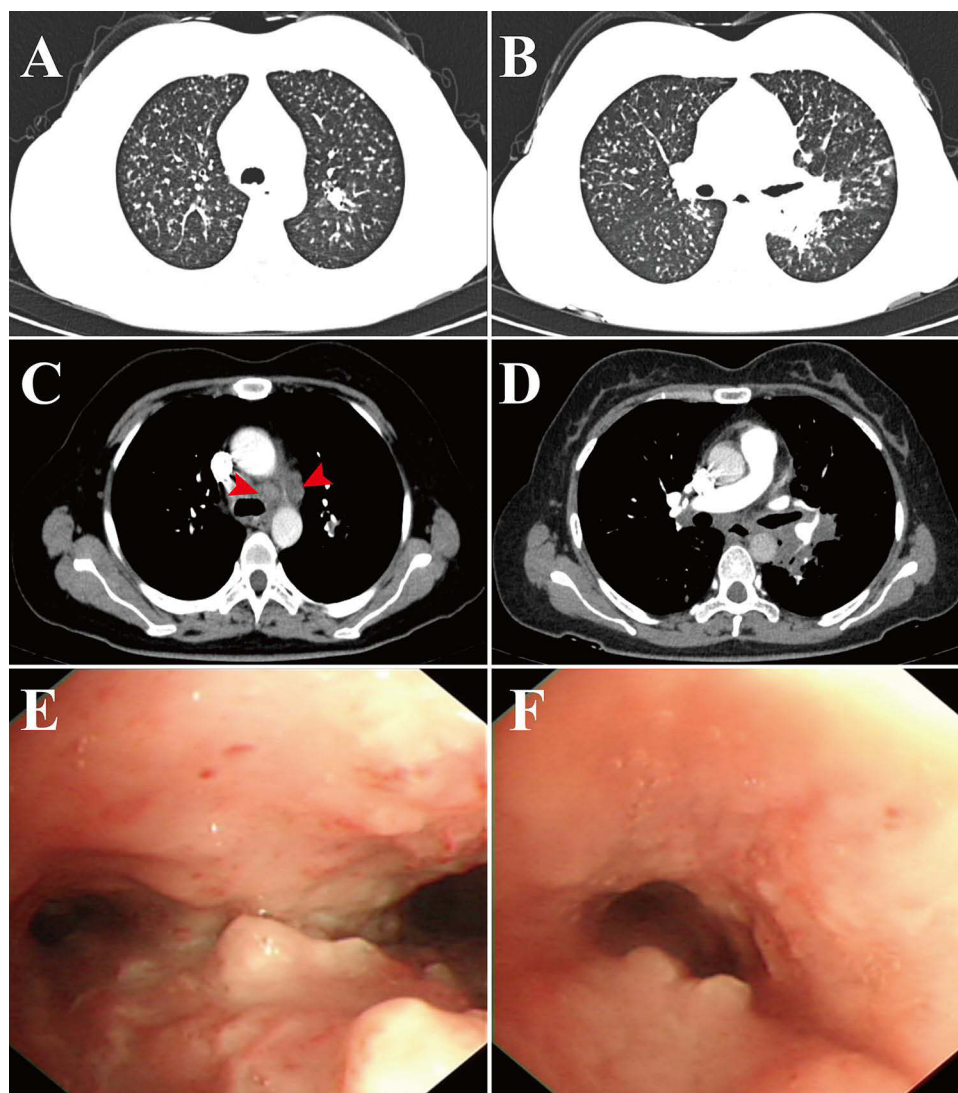


Figure 1 Radiology and bronchoscopy findings. (A and B) Chest CT scans reveal bilateral miliary nodular opacities. (C) Chest CT reveals enlarged mediastinal lymph nodes (red arrows). (D) Chest CT reveals left hilar enlargement. (E) Bronchoscopy showed nodular neoplastic lesions superior to the tracheal carina. (F) Bronchoscopy showed nodular neoplastic lesions in the left main bronchus.

potential etiologies, and diagnostic antifungal therapy was initiated. Intravenous voriconazole was administered at a loading dose of 300 mg twice daily for the first day, followed by 200 mg twice daily for five days. Subsequently, the therapy was switched to an oral formulation during the induction phase. Follow-up chest CT scan demonstrated significant resolution of the pulmonary lesions. Consolidation therapy with oral voriconazole (200 mg twice daily) was administered for 10 weeks. Finally, a repeat chest CT scan was performed on July 25, revealing complete resolution of the pulmonary lesions (Figure 3D–F). Considering the resolution of clinical symptoms, the antifungal therapy was discontinued, and the patient was followed up.

Discussion

T. marneffei is the only thermally dimorphic fungus in the *Talaromyces* species capable of causing disseminated infections in mammals, including humans. Talaromycosis is an invasive fungal infection caused by this pathogen.⁹ The condition predominantly affects immunocompromised individuals, particularly those infected with HIV. In Chinese endemic regions, approximately 88% of talaromycosis cases have been detected in individuals infected with HIV.⁵ Specifically, the prevalence of talaromycosis in HIV-positive individuals is 16.1%.¹⁰ Co-infection with HIV has elevated

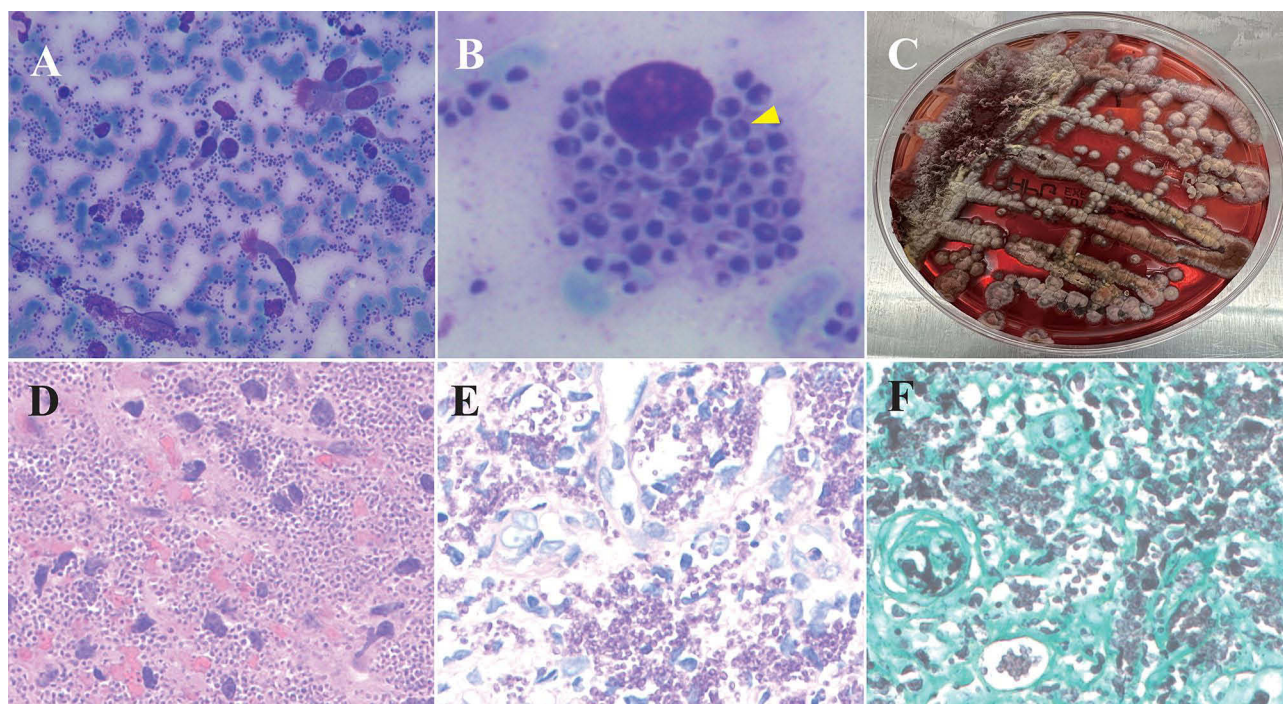


Figure 2 Pathological findings in Case 1. (A and B) LBC examination (Diff-Quik stain, 400×): (A) Intracellular blue-staining granular microorganisms; (B) Intracellular yeast-like fungal cells (yellow arrow). (C) Fungal culture: *T. marneffei* colonies isolated from BALF. Histopathology examination: (D) Chronic inflammation with histiocyte aggregation and pathogens in the histiocyte cytoplasm (hematoxylin and eosin stain, 400×); (E) Periodic acid-Schiff stain (400×) demonstrating numerous intracellular yeast-like fungi; (F) Grocott's methenamine silver stain (400×) showing *T. marneffei* cells.

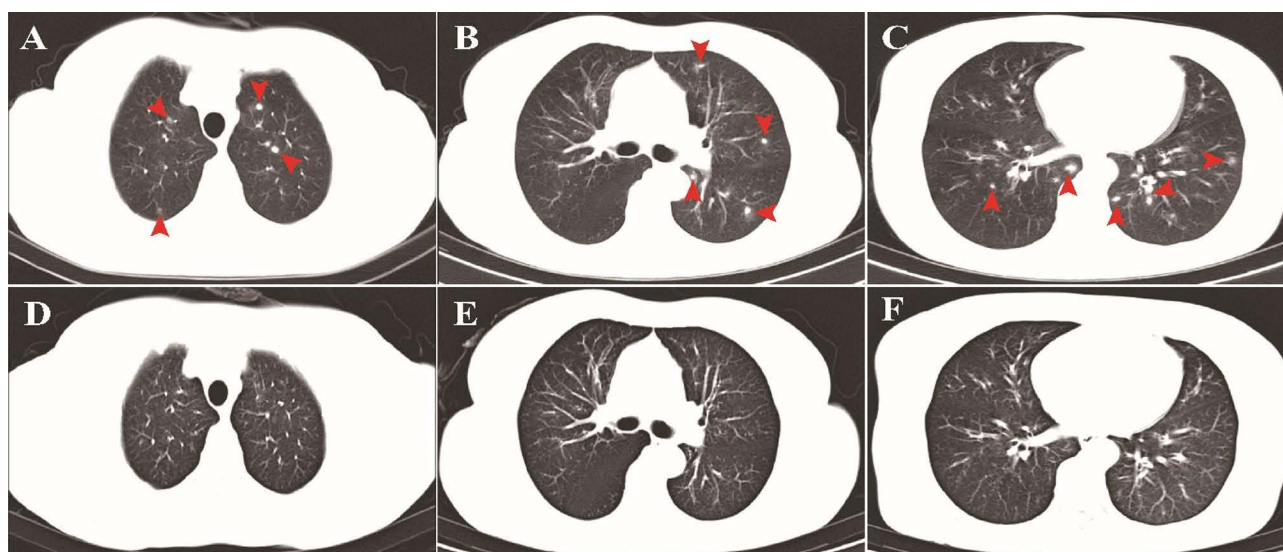


Figure 3 Serial chest CT imaging in Case 2. (A–C) Baseline scans showing multiple patchy and nodular opacities (red arrows) prior to antifungal therapy. (D–F) Follow-up scans after administration of voriconazole for 12 weeks showing complete resolution of pulmonary lesions.

T. marneffei from a rare opportunistic pathogen to the third most common cause of opportunistic infections among HIV-positive patients in endemic areas.¹¹ In recent years, the incidence of talaromycosis has decreased with improved HIV epidemic control through effective antiretroviral therapy and HIV transmission prevention measures.¹² While talaromycosis is rare in non-endemic areas, the increased population mobility and international travel have led to a growing number of cases in these areas. Some talaromycosis cases were reported in non-endemic areas after traveling to endemic areas.^{6,13,14} Furthermore, a growing number of HIV-negative individuals with primary or secondary immunosuppressive

conditions infected with *T. marneffei* are being documented.⁸ Published articles reported talaromycosis in patients with adult-onset immunodeficiency syndrome (e.g. anti-gamma interferon autoantibodies)¹⁵ and organ transplant recipients.¹⁶ Consequently, the changing epidemiology of talaromycosis warrants more clinical attention, and physicians in non-endemic areas should increase their awareness of this fungal infection.

Talaromycosis in humans is probably caused by inhaling *T. marneffei* conidia, resulting in disseminated infection commonly involving the lungs, skin, bone marrow, lymph nodes, and liver.^{1,11} The clinical manifestations of talaromycosis include constitutional symptoms (fever, weight loss, and fatigue), anemia, lymphadenopathy, cutaneous lesions, and respiratory or gastrointestinal abnormalities. Pulmonary involvement occurs in approximately 95% of talaromycosis cases,¹⁷ manifesting as respiratory symptoms such as fever, cough, sputum production, and dyspnea. Chest CT scan of pulmonary talaromycosis exhibit consolidations, patchy opacity, mass opacity, nodular opacities, mediastinal lymphadenopathy, and pleural effusion.¹⁸ Moreover, pulmonary talaromycosis is characterized by non-specific respiratory infection features and is often misdiagnosed as tuberculosis or other respiratory infections. Talaromycosis has a high mortality rate, and establishing an accurate diagnosis directly correlates with reduced mortality rates and improved outcomes. Furthermore, recent diagnostic advances have highlighted the utility of BALF in diagnosing talaromycosis, with fungal culture and mNGS of BALF demonstrating particular diagnostic value.¹⁹ Additional bronchoscopic diagnostic procedures, including transbronchial biopsy and needle aspiration, in combination with mNGS, have also been proven as effective diagnostic methods for this disease.^{20,21} Consequently, bronchoscopy plays a critical role in early differentiation and diagnosis of pulmonary talaromycosis.

Currently, clinical reports on tracheobronchial involvement in talaromycosis remain scarce. The present case report illustrates an unusual manifestation of talaromycosis with tracheobronchial involvement. Notably, obtaining airway specimens through bronchoscopy for histopathological examination and fungal culture is crucial for definitive diagnosis. A previous study revealed that respiratory tract specimens, including mucosal tissue and BALF, yielded the highest positivity rates for *T. marneffei* isolation compared with the diagnosis methods of routine culture and pathological examinations.²² The bronchoscopy findings of Case 1 revealed multiple nodular neoplasms in the airways, with morphological similarities to airway tumors or tracheal tuberculosis. Histopathological examination of biopsy tissue for Case 1 demonstrated histiocyte aggregation and *T. marneffei* cells within histiocyte cytoplasm, which is a frequent finding in HIV-positive patients with talaromycosis. This distinctive histopathological feature serves as a crucial diagnostic marker to differentiate talaromycosis from both airway tumors and tuberculous lesions. In Case 1, intracellular pathogens appeared as oval or spherical forms, and specific stains of PAS and GMS yielded positive results. The characteristic transverse midline septum was observed in only a small number of pathogen cells. Based on these features, *T. marneffei* infection was considered and then confirmed by BALF microbial cultures.

As an emerging and advanced diagnostic method, mNGS employs unbiased high-throughput sequencing of nucleic acids extracted from samples within a short time to comprehensively identify pathogens.²³ This method has been widely adopted for infectious disease diagnosis in clinical settings due to its rapid processing time and accuracy. Previous studies demonstrated that mNGS has excellent sensitivity and specificity for diagnosing talaromycosis (97.22% and 100.00%, respectively).²⁴ In the second case of an HIV-negative patient with talaromycosis, conventional microbial cultures yielded negative results. However, mNGS of BALF sample effectively identified *T. marneffei*. This definitive diagnosis enabled prompt treatment, which resulted in a favorable prognosis. This highlights the particular diagnostic value of mNGS in culture-negative cases.

Talaromycosis carries a mortality rate approaching 30%. However, the risk of death can be significantly reduced if these patients are treated with appropriate antifungal therapy.¹ The antifungal therapy of talaromycosis consists of induction, consolidation, and maintenance phases. Current international guidelines recommend amphotericin B (liposomal amphotericin B given at 3–5 mg/kg daily, or deoxycholate amphotericin B at 0.7 mg/kg daily) as first-line antifungal drugs for induction therapy, followed by oral itraconazole (200 mg twice daily) for consolidation therapy.²⁵ Induction therapy was given for two weeks, and consolidation therapy was maintained for 10 weeks. In case of medication unavailability, voriconazole serves as an effective alternative for both induction and consolidation therapy.^{26,27} HIV-positive patients with CD4⁺ T-cell counts of less than 100 cells/ μ L require maintenance therapy with oral itraconazole (200 mg daily) until the CD4⁺ T-cell counts exceed 100 cells/ μ L and remain stable for over 6 months.¹¹

Nonetheless, preventive administration of itraconazole (200 mg orally daily) against systemic fungal infections, such as talaromycosis, in HIV-positive individuals with severe immunosuppression ($CD4^+$ T-cell count < 200 cells/ μ L) is not widely adopted.

In this study, Case 1 was initially treated with amphotericin B cholesteryl sulfate complex for induction therapy; however, the treatment was discontinued due to refractory hypokalemia, a well-documented adverse effect of this drug that frequently necessitates treatment discontinuation. Ultimately, both patients received voriconazole as the antifungal agent. Serial monitoring of liver and kidney function throughout the treatment course demonstrated no drug-related adverse effects, and both patients achieved significant clinical improvements after antifungal therapy. This is evidenced by the repeated chest CT scans for Case 2, which demonstrated remarkable resolution of lung lesions after 12 weeks of voriconazole therapy. These findings highlight the favorable efficacy and safety profile of voriconazole for treating talaromycosis. Notably, no standardized treatment protocol has been established for HIV-negative patients with talaromycosis, who are typically managed following guidelines designed for HIV-positive individuals. From the perspective of antifungal treatment in HIV-negative patients with talaromycosis, the successful outcomes in Case 2 provide crucial clinical evidence supporting voriconazole monotherapy as an effective and well-tolerated regimen for both induction and consolidation phases.

Conclusion

Talaromycosis is a potentially fatal endemic mycosis whose epidemiological characteristics are no longer limited to geographical endemicity and HIV-positive individuals. Cases in non-endemic areas and HIV-negative individuals warrant increasing attention. The respiratory system is the most frequently affected in talaromycosis, and early diagnosis can be significantly improved by combining bronchoscopy with mNGS.

Data Sharing Statement

The data are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

The study was reviewed and approved by the Ethics Committee at Yichang Central People's Hospital (ethics approval number: 2025-236-01). Written informed consent for participation in this study was obtained from all participants. Yichang Central People's Hospital and The First College of Clinical Medical Science, China Three Gorges University has approved the publication of the case details in this study.

Consent for Publication

Written informed consent was obtained from all participants for the publication of the details of their medical case and any accompanying images.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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