Cureus
Part of SPRINGER NATURE

Open Access Case Report

Coinfection With Disseminated Histoplasmosis and Extrapulmonary Tuberculosis in a Pediatric Patient: A Case Report

Cesar Alas [1], Marvin J. Menjivar-Vallecillo [2, 3], Sendy Ruiz [4], Luis Romero Reyes [5]

1. Quantitative Biomedical Science Program, Dartmouth College, Hanover, USA 2. GIMUNICAH, Faculty of Medicine, Pontificia Universidad Católica de Honduras, San Pedro Sula, HND 3. Research, Sapiens Wisdom Research, San Pedro Sula, HND 4. Faculty of Medicine, Pontificia Universidad Católica de Honduras, San Pedro Sula, HND 5. Pediatric Infectious Diseases, Hospital Mario Catarino Rivas, San Pedro Sula, HND

Corresponding author: Marvin J. Menjivar-Vallecillo, marvinmenjivarv04@gmail.com

Review began 10/07/2026
Review ended 11/08/2026
Published 11/13/2026

© Copyright 2026
Alas et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119611

Abstract

Coinfection with Histoplasma capsulatum and extrapulmonary Mycobacterium tuberculosis is rare in pediatric patients without previously confirmed severe immunodeficiency. We report the case of a 9-year-old boy from Honduras who presented with prolonged fever, weight loss, generalized lymphadenopathy, and abdominal distension. One year earlier, he had been hospitalized for bicytopenia and treated with antibiotics and intravenous steroids. Further investigations confirmed extrapulmonary tuberculosis via lymph node biopsy and disseminated histoplasmosis through special stains and a positive urinary antigen test. The patient was treated with amphotericin B followed by oral itraconazole, along with antituberculous therapy and supportive care, achieving a favorable clinical outcome. This case highlights the diagnostic challenges of fungal-mycobacterial coinfections in complex pediatric hosts and emphasizes the importance of early detection, combined diagnostic testing, and a multidisciplinary approach to optimize clinical outcomes and reduce morbidity and mortality.

Categories: Other, Pediatrics, Pulmonology
Keywords: coinfection, histoplasmosis, lymphadenopathy, pediatric, tuberculosis

Introduction

Histoplasmosis is an endemic mycosis caused by the dimorphic fungus Histoplasma capsulatum, whose distribution is widely recognized across North, Central, and South America [1,2]. In endemic regions, it is estimated that over 500,000 infections occur annually, although many are subclinical or go undiagnosed, contributing to a potential underestimation of its true incidence [2,3].

Although H. capsulatum is historically recognized in these regions, there is growing evidence of its significant presence across Latin America and Central America. In Honduras, recent studies estimate an incidence of approximately 4.3 cases per 100,000 inhabitants, with between 430 and 500 cases annually and a reported mortality of up to 255 deaths per year, particularly among people living with HIV [4]. Multicenter study conducted between 2015 and 2019 in Honduras, Nicaragua, and Panama found that 32% of Honduran patients with HIV and suspected opportunistic infections tested positive for histoplasmosis via urinary antigen detection, the highest percentage among the countries evaluated [5]. Environmental presence of the fungus has also been detected in neighboring countries such as Guatemala, El Salvador, and Nicaragua, as well as in forested and cavernous areas of western Honduras [6].

Infection occurs through the inhalation of spores, which lodge in the lungs and can remain latent within macrophages due to the granulomatous immune response [1]. In most individuals, the infection is localized or asymptomatic, but 5-10% of cases may progress to progressive disseminated histoplasmosis (PDH), particularly when the host immune response is impaired [1,7].

There are two pathogenic varieties for humans: H. capsulatum var. capsulatum, which predominates in the Americas, and H. capsulatum var. duboisii, primarily associated with cases in Africa [7]. Diagnosis is based on rapid antigen tests, cultures, and histopathological studies. Treatment of progressive disseminated histoplasmosis (PDH) begins with amphotericin B in severe cases, followed by itraconazole [1,8].

In systemic fungal infections, it is important to consider coinfection with extrapulmonary tuberculosis. Although it is often associated with low CD4+ lymphocyte counts (<200 cells/μL), it can also occur in young or vulnerable hosts following prior immunosuppressive exposures. Overlapping symptoms, such as fever, weight loss, night sweats, lymphadenopathy, and cytopenias, complicate clinical and radiological differentiation. Extrapulmonary tuberculosis frequently affects lymph nodes, serous surfaces, the liver, spleen, and the central nervous system [8,9].

How to cite this article
Alas C, Menjivar-Vallecillo M J, Ruiz S, et al. (November 13, 2026) Coinfection With Disseminated Histoplasmosis and Extrapulmonary Tuberculosis in a Pediatric Patient: A Case Report. Cureus 18(11): e119611. DOI 10.7759/cureus.119611

This rarity is likely explained by the fact that adequate host immune defenses usually control at least one of the two infections before dissemination occurs [10]. However, in children, whose immune response is still developing, defense against these intracellular pathogens may be insufficient, favoring simultaneous dissemination [11]. The clinical similarity and nonspecificity of radiological findings complicate diagnosis, contributing to its underestimation. Concurrent presentation represents a major clinical challenge that requires a high index of suspicion, particularly in endemic areas.

We present the case of a pediatric patient from a rural area in Honduras, with prior systemic steroid exposure and ongoing investigation for underlying immunodeficiency, who developed a severe coinfection with disseminated histoplasmosis and extrapulmonary tuberculosis with central nervous system involvement. This report highlights the diagnostic approach and multidisciplinary management of dual intracellular co-infection in a complex pediatric host.

Case Presentation

A 9-year-old male patient from a rural area in the Santa Bárbara department of Honduras presented to the emergency department. His mother has a history of pulmonary tuberculosis diagnosed in 2024 (under active treatment), while his father has no relevant medical history. The patient denied recent travel or contact with animals, and his main activity was attending school.

In April 2023, the patient was hospitalized for one week due to bicytopenia of undetermined etiology. He received intravenous corticosteroids and antibiotics, with subsequent clinical resolution. He remained under monthly follow-up by the Pediatric Hematology-Oncology Department, with normal blood counts during follow-up. In August 2024, he was readmitted for bilateral cervical lymphadenopathy; biopsy confirmed tuberculoid granulomatous lymphadenitis, establishing the diagnosis of extrapulmonary tuberculosis and initiating antituberculous therapy. On February 13, 2025 (hospital day 1), while in the continuation phase of antituberculous treatment, the patient presented to the emergency department with a 15-day history of high fever, a 3-kg weight loss, cervical lymphadenopathy (1-3 cm), and painful abdominal distension. Abdominal ultrasonography revealed mesenteric adenitis. Laboratory tests showed leukopenia (white blood cell count: 3.2×10^9/L) and anemia (hemoglobin: 7.7 g/dL), with a platelet count of 177×10^9/L. Mild hepatomegaly was also noted, prompting admission for evaluation of a suspected hematologic disorder (Table *1*, Figure *1*).

Test Name	Day 1 (Admission)	Day 3–5 (Clinical Deterioration and ICU)	Day 7–16 (Critical / Diagnostic Phase)	Unit of Measurement	Reference Range
Complete Blood Count (CBC)					
White Blood Cells (WBC)	3.2	1.7	3.7	10^9/L	5.0 – 10.5
Hemoglobin (HGB)	7.7	5.8	9.5	g/dL	12.0 – 18.0
Platelet Count (PLT)	177	4	130	10^9/L	130 – 400
Inflammatory & Metabolic Markers					
C-Reactive Protein (CRP)	—	24.0	192.0	mg/L	0.0 – 6.0
Interleukin-6 (IL-6)	—	—	34.4	pg/mL	0.0 – 10.0
Serum Potassium (K+)	—	1.45	2.70	mmol/L	3.4 – 5.1
Fasting Glucose	—	45* / 219	—	mg/dL	70 – 100
Bicarbonate (HCO3-)	—	15.5	—	mmol/L	22.0 – 26.0
Chemistry & Coagulation					
Aspartate Aminotransferase (AST/GOT)	—	—	164.4	U/L	0.0 – 38.0
Alanine Aminotransferase (ALT/GPT)	—	—	117.2	U/L	0.0 – 41.0
Total Bilirubin	—	—	6.1	mg/dL	0.2 – 1.0
D-Dimer	—	—	3,106.6	ng/mL	0 – 500
Prothrombin Time (INR)	—	—	2.71	N/A	0.87 – 1.20
Microbiological & Molecular Tests					
Histoplasma Urinary Antigen	—	—	Positive (50 ng/mL)	N/A	Negative
Urine TB-LAM Test	—	—	Positive	N/A	Negative
GeneXpert MTB/RIF (Urine)	—	—	Detected (No RIF resistance)	N/A	Not Detected
Dengue Rapid Test (NS1)	—	Positive (Day 3)	—	N/A	Negative
Blood Culture	—	Negative (at 6 days)	—	N/A	Negative

TABLE 1: Summary of Relevant Laboratory Investigations

Note: Values represent findings during the acute phase and critical clinical progression of hospitalization.

Abbreviations: ALT/GPT: alanine aminotransferase; AST/GOT: aspartate aminotransferase; CBC: complete blood count; CRP: C-reactive protein; HGB: hemoglobin; IL-6: interleukin-6; MTB/RIF: Mycobacterium tuberculosis / rifampicin; N/A: not applicable; PLT: platelets; TB-LAM: tuberculosis lipoarabinomannan; WBC: white blood cells.

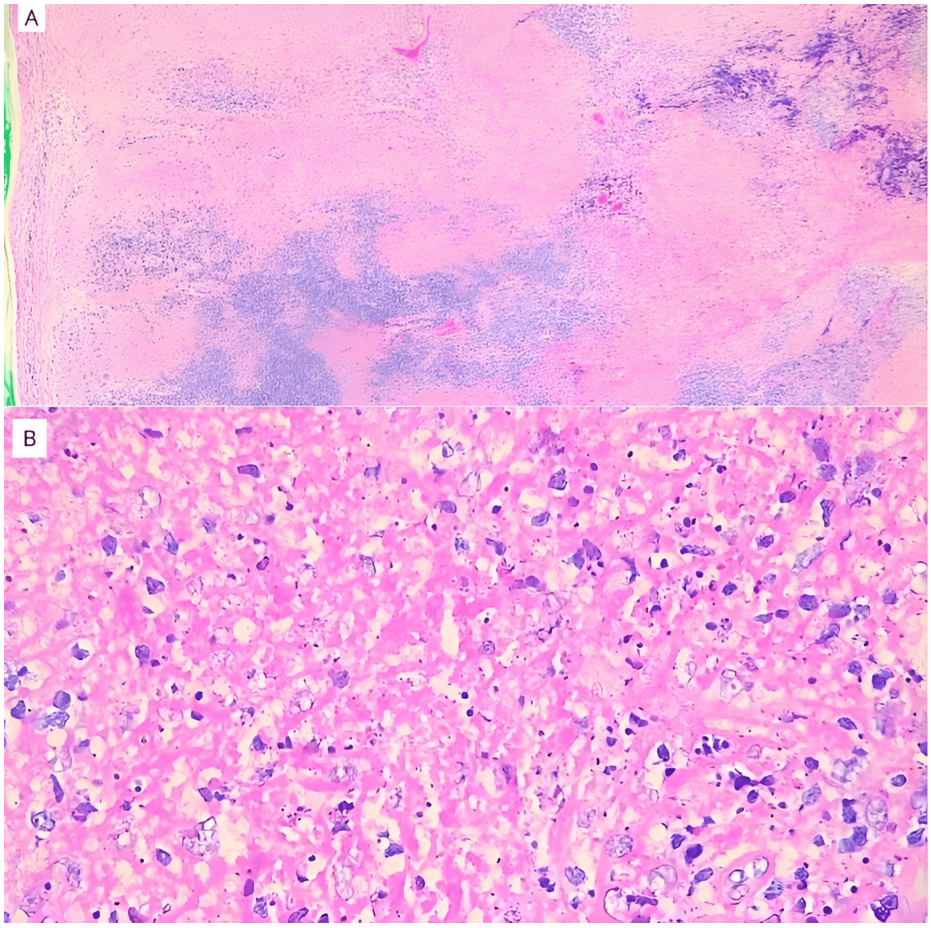


FIGURE 1: Chronic non-caseating granulomatous lymphadenitis in a cervical lymph node (H&E).

(A) Panoramic view of the cervical lymph node showing loss of normal architecture and multiple non-caseating granulomas (H&E, 100x). (B) Higher magnification view showing epithelioid histiocytes and scant residual lymphoid tissue, without caseous necrosis (H&E, 400x).

Note: Non-caseating granulomatous histology is non-specific, and tissue acid-fast bacilli (AFB) stains/cultures were negative on this lymph node specimen. Definite confirmation of extrapulmonary tuberculosis was established via positive urine TB-LAM and urine GeneXpert MTB/RIF assays.

On February 15, 2025 (hospital day 3), dengue infection was confirmed by a rapid NS1 antigen test, and intravenous fluid therapy was initiated. However, persistent fever and clinical deterioration prompted empirical antibiotic treatment with ceftriaxone. On February 17, 2025 (hospital day 5), the patient developed severe metabolic disturbances, including hyponatremia, hypoglycemia, and metabolic acidosis, requiring transfer to the intensive care unit (Table *1*). On February 18, 2025 (hospital day 6), intravenous amphotericin B deoxycholate was initiated at 1 mg/kg/day because of suspected disseminated fungal infection. Repeat cervical lymph node biopsy specimens were obtained for histopathological examination, which revealed non-caseating granulomatous lymphadenitis with yeast-like structures. Additional investigations showed bilateral reticulonodular pulmonary infiltrates and a mild right-sided pleural effusion on chest computed tomography. Bone marrow aspiration revealed adequate cellularity without evidence of blasts (Figure *2*).

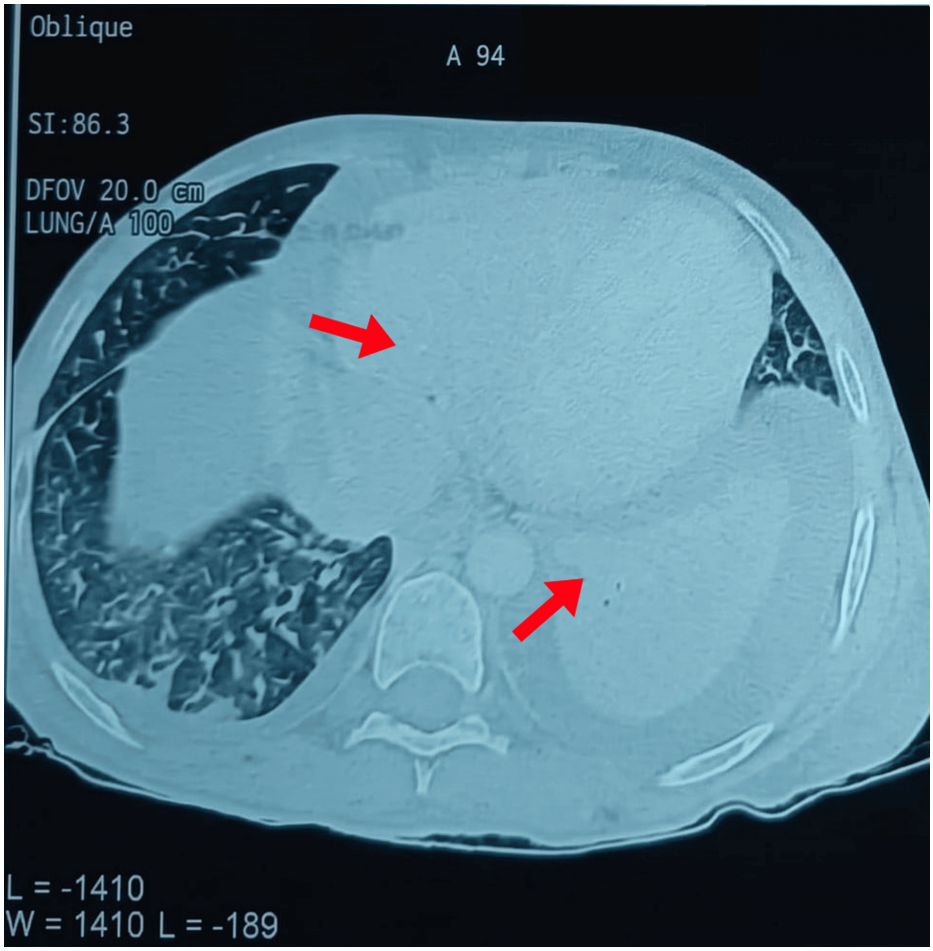


FIGURE 2: Chest CT scan showing mild right pleural effusion and pulmonary infiltrates.

Axial computed tomography (CT) scan of the chest showing a small homogeneous fluid collection in the right hemithorax (indicated in the image), associated with scattered parenchymal infiltrates. These findings are consistent with a mild pleural effusion and early pulmonary involvement, documented during the patient's hospitalization.

On February 19, 2025 (hospital day 7), the patient developed hemodynamic deterioration requiring vasopressor support and broad-spectrum antibiotic therapy with meropenem. Intravenous immunoglobulin (IVIG) therapy (administered over two days) was added due to severe clinical deterioration and high suspicion of underlying primary immunodeficiency.

On February 26, 2025 (hospital day 14), intravenous dexamethasone was initiated. On February 28, 2025 (hospital day 16), the patient developed seizures, and brain computed tomography revealed non-specific cortical and subcortical hypodense areas, raising concern for possible secondary neurological involvement in the context of systemic infection (Figure *3*). Anticonvulsant therapy with levetiracetam and oxcarbazepine was initiated. A diagnostic thoracentesis was also performed; pleural fluid analysis, including cytochemical examination, culture, and GeneXpert MTB/RIF testing, was negative for Mycobacterium tuberculosis. Toward the end of February, urine TB-LAM and urine GeneXpert MTB/RIF assays were positive, with the latter detecting M. tuberculosis without rifampicin resistance. These findings, considered alongside the patient's clinical history, supported the diagnosis of extrapulmonary tuberculosis. The non-caseating granulomatous findings on lymph node histopathology were not considered independently diagnostic (Table *1*).



FIGURE 3: Brain CT scan showing lesions consistent with secondary neurological involvement.

Sagittal non-contrast computed tomography (CT) scan of the brain demonstrating subtle cortical and subcortical hypodense areas. These findings are non-specific and may reflect localized inflammatory, ischemic, or post-ictal changes, which clinically correlated with the onset of seizures. Secondary neurological involvement by Histoplasma capsulatum or Mycobacterium tuberculosis was suspected based on systemic dissemination, although neuroimaging findings remain non-diagnostic on CT alone.

Confirmatory mycology and molecular laboratory testing established the diagnosis of co-infection. Grocott's methenamine silver (GMS) stain of the repeat cervical lymph node biopsy revealed loss of normal tissue architecture, extensive hyalinization, and non-caseating granulomatous lymphadenitis with intracellular and extracellular yeast-like structures measuring 2-5 µm with single budding, consistent with Histoplasma capsulatum (Figure *4*). Intracellular yeast structures were also observed on Giemsa stain. Disseminated disease was further confirmed by a positive urine Histoplasma antigen assay (50 ng/mL). Tuberculosis was confirmed by a positive urine TB-LAM assay and a positive GeneXpert MTB/RIF test for Mycobacterium tuberculosis, with no rifampicin resistance detected (Table *1*). Trimethoprim-sulfamethoxazole (TMP-SMX) was initiated for 17 days due to ongoing suspicion of primary immunodeficiency, and full antituberculous therapy was resumed. Comprehensive immunological testing (serum immunoglobulins, lymphocyte subsets, phagocyte assays) was unavailable due to local resource limitations. HIV serology was negative.

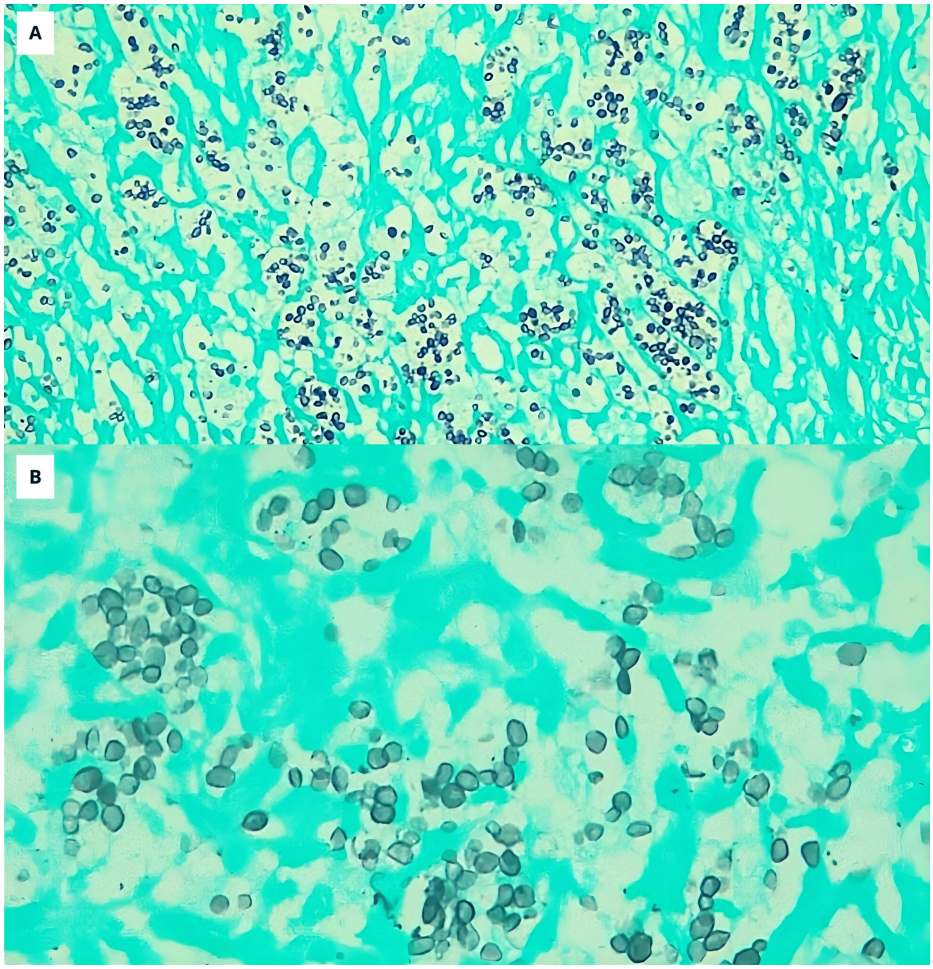


FIGURE 4: Mycological confirmation of Histoplasma capsulatum infection using Grocott stain.

(A) Panoramic view of the lymph node showing granulomas with intracellular yeast-like structures stained black against a greenish background (100x). (B) Higher magnification view showing round yeasts measuring 2–5 µm with single budding, consistent with Histoplasma capsulatum (400x). Disseminated infection was further supported by intracellular yeasts on Giemsa stain and confirmed via positive urine Histoplasma antigen assay (50 ng/mL).

Targeted antifungal management included intravenous amphotericin B deoxycholate at 1 mg/kg/day, followed by voriconazole and subsequently oral itraconazole, which was initiated on February 28, 2025. The available medical records did not allow the total duration of amphotericin B or voriconazole therapy to be established. Intravenous immunoglobulin (for two days) and intravenous dexamethasone were administered due to severe systemic inflammatory compromise and neurological involvement. At discharge, the patient was afebrile and hemodynamically stable on an outpatient regimen including oral itraconazole, antituberculous drugs, trimethoprim-sulfamethoxazole, anticonvulsants (levetiracetam and oxcarbazepine), and sucralfate, with ongoing multidisciplinary outpatient follow-up.

Discussion

Coinfection with Histoplasma capsulatum and Mycobacterium tuberculosis represents a rare condition in pediatric patients without confirmed prior immunodeficiency or known HIV infection. Both infections are endemic in tropical regions and share similar pathogenic and immunological mechanisms, which complicate differential diagnosis and may delay treatment initiation [6,10]. In the present case, the occurrence in a child without previously confirmed primary immunodeficiency and from a region not recognized as highly endemic in Honduras highlights the need for a high index of suspicion in the presence of prolonged febrile episodes, generalized lymphadenopathy, and cytopenias.

From a pathophysiological perspective, both H. capsulatum and M. tuberculosis are facultative intracellular pathogens capable of surviving within macrophages by inhibiting phagosome-lysosome fusion and modulating the inflammatory response [1,12]. In hosts with intact cell-mediated immunity, CD4+ T lymphocytes and the activation of cytokines such as IFN-γ and TNF-α promote granuloma formation that

contains the infection. However, in childhood, incomplete immune maturation and factors such as prior tuberculosis exposure or corticosteroid use can facilitate the dissemination of Histoplasma to visceral organs and the central nervous system [6,7].

The clinical presentation of this patient, which included persistent fever, weight loss, hepatomegaly, lymphadenopathy, and pancytopenia, mimicked malignant hematologic diseases and extrapulmonary tuberculosis, delaying the identification of disseminated mycosis [9]. In tuberculosis endemic regions, diagnostic suspicion focuses primarily on mycobacterial disease, often underestimating systemic mycoses [4]. Crucially, the lack of clinical response to broad-spectrum antibacterial therapy and ongoing antituberculous treatment, accompanied by progressive metabolic deterioration, served as the primary clinical triggers to prompt comprehensive diagnostic evaluation for a co-existing fungal etiology.

Diagnostic differentiation between Mycobacterium tuberculosis and Histoplasma capsulatum was established through independent microbiological, histopathological, and molecular findings, rather than histology alone, since non-caseating granulomatous lymphadenitis is a non-specific finding that may occur in both infections [1,12]. H. capsulatum was definitively identified by the presence of characteristic intracellular yeast structures in Grocott's methenamine silver (GMS) and Giemsa-stained lymph node tissue, supported by a positive urinary Histoplasma antigen test [1,5]. Importantly, the identification of H. capsulatum in lymph node tissue did not exclude concurrent tuberculosis, which was independently confirmed by positive urinary TB-LAM and GeneXpert MTB/RIF assays. The latter demonstrated the presence of M. tuberculosis complex DNA without evidence of rifampicin resistance, providing independent etiological evidence of tuberculosis [10].

The dual inflammatory response likely explains the severity of the clinical presentation. Both infections trigger a strong granulomatous reaction mediated by cytokines such as TNF-α and IFN-γ, but the persistence of antigens from both pathogens can lead to disorganized chronic inflammation and progressive tissue damage [12]. This phenomenon is associated with a higher risk of dissemination and systemic complications, including neurological, metabolic, and hepatosplenic involvement, all of which were observed in the described patient. Additionally, the interaction between intracellular infections may induce reciprocal immunomodulation, whereby the immune response against M. tuberculosis alters the control of H. capsulatum, favoring its persistence [10].

Antihistoplasma and antituberculous treatment regimens followed standard pediatric protocols adapted to institutional resources. Antituberculous therapy consisted of a standard four-drug regimen during the intensive phase (rifampicin 15 mg/kg/day, isoniazid 10 mg/kg/day, pyrazinamide 35 mg/kg/day, and ethambutol 20 mg/kg/day) for two months, followed by a two-drug continuation phase (rifampicin and isoniazid) planned for 10-12 months due to extrapulmonary dissemination [10]. For histoplasmosis, induction therapy was initiated with amphotericin B deoxycholate (1 mg/kg/day) for 14 days in the intensive care unit, given the severity of the disseminated infection and the need for prompt fungicidal treatment. Following clinical stabilization, the patient was transitioned to oral itraconazole maintenance therapy at an initial dose of 100 mg/day. However, following clinical relapse and considering the potential reduction in itraconazole exposure due to rifampicin-mediated hepatic enzyme induction, the itraconazole dose was increased to 200 mg/day, with a planned total antifungal treatment duration of 12 months [7,12]. Therapeutic drug monitoring (TDM) for itraconazole plasma levels was unavailable due to local laboratory constraints; thus, management relied on close clinical surveillance, relapse monitoring, and hepatic toxicity screening. Nevertheless, the potential rifampicin-itraconazole interaction remained a therapeutic challenge, as itraconazole plasma concentrations could not be measured and the adequacy of drug exposure following dose escalation could not be confirmed.

Empiric immunomodulatory and prophylactic therapies were required during the patient's critical ICU course. Intravenous immunoglobulin (IVIG) and high-dose dexamethasone were introduced to control overwhelming hyperinflammation secondary to severe systemic dissemination and suspected macrophage activation-like syndrome [7,12]. Concurrently, trimethoprim-sulfamethoxazole (TMP-SMX) was initiated empirically for broad-spectrum antimicrobial coverage and opportunistic infection prophylaxis given the clinical presentation of severe cytopenias and high suspicion of underlying impaired cell-mediated immunity. A main limitation of this case is that advanced immunological profiling (such as lymphocyte subset analysis, serum immunoglobulins, and oxidative burst assays) could not be performed during the acute phase due to local laboratory constraints, necessitating these aggressive empiric strategies to prevent fatal progression.

The patient's clinical recovery underscores the importance of timely diagnosis and targeted treatment in severe coinfections. However, this case highlights the limited availability of rapid mycological testing in rural areas of Honduras, contributing to the underdiagnosis of histoplasmosis [4,5]. Indeed, regional studies indicate that over 30% of Honduran patients with suspected opportunistic infections test positive for histoplasmosis, reflecting the hidden magnitude of the problem in Central America [6].

Seizures raised clinical suspicion of secondary central nervous system (CNS) involvement [1]. However, definitive confirmation of CNS histoplasmosis could not be established; advanced neuroimaging (MRI) and

cerebrospinal fluid (CSF) analysis were omitted due to severe hemodynamic instability and critical condition in the intensive care unit, along with institutional resource constraints. Thus, neurological findings reflect a plausible clinical manifestation of systemic dissemination rather than a proven organ-specific diagnosis.

Finally, this case highlights the urgent need to strengthen mycological surveillance in Central America, where histoplasmosis remains an underestimated cause of pediatric morbidity and mortality. Integrating antigen testing and molecular tools into national tuberculosis programs could improve detection and reduce misdiagnoses [6,7].

Conclusions

Coinfection with Histoplasma capsulatum and Mycobacterium tuberculosis represents a significant diagnostic challenge in complex pediatric hosts, particularly in the presence of prior immunosuppressive exposures or suspected underlying primary immunodeficiencies. The overlapping features of these infections often lead to a predominantly tuberculosis-oriented approach in endemic regions, potentially delaying the recognition of disseminated histoplasmosis. This case underscores the importance of maintaining a broad differential diagnosis in children presenting with prolonged fever, hematologic abnormalities, lymphadenopathy, non-specific neurological symptoms, and multisystem involvement. Integrating microbiological, mycological, and antigen-based diagnostic methods at an early stage is essential. Timely recognition of dual infections and prompt initiation of targeted therapy supported by multidisciplinary management are critical to minimize complications and optimize outcomes. Furthermore, the limited availability of fungal diagnostic tools in Central America highlights the urgent need to strengthen surveillance and diagnostic capacity for systemic mycoses in pediatrics.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Marvin J. Menjivar-Vallecillo, Cesar Alas

Acquisition, analysis, or interpretation of data: Marvin J. Menjivar-Vallecillo, Cesar Alas, Sendy Ruiz, Luis Romero Reyes

Drafting of the manuscript: Marvin J. Menjivar-Vallecillo, Cesar Alas

Critical review of the manuscript for important intellectual content: Marvin J. Menjivar-Vallecillo, Cesar Alas, Sendy Ruiz, Luis Romero Reyes

Supervision: Marvin J. Menjivar-Vallecillo, Cesar Alas, Sendy Ruiz, Luis Romero Reyes

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Li LX, Rajack ST, Ostrander D, et al.: Progressive disseminated histoplasmosis: the experience in one non-endemic medical center. Med Mycol. 2023, 61:myad115. 10.1093/mmy/myad115
2. Hajjeh RA: Disseminated histoplasmosis in persons infected with human immunodeficiency virus . Clin Infect Dis. 1995, 21:S108-S110. 10.1093/clinids/21.supplement_1.s108
3. CDC. Histoplasmosis. (2024). Accessed: November 10, 2026: https://www.cdc.gov/histoplasmosis/index.html.
4. Agudelo Higuita NI, Varela Bustillo D, Denning DW: Burden of serious fungal infections in Honduras . Mycoses. 2022, 65:429-439. 10.1111/myc.13432
5. Caceres DH, Arauz AB, Flores C, et al.: Implementation of rapid diagnostics assays for detection of histoplasmosis and cryptococcosis in Central American people living with HIV. Mycoses. 2021, 64:1396-1401. 10.1111/myc.13303
6. Adenis AA, Valdes A, Cropet C, et al.: Burden of HIV-associated histoplasmosis compared with tuberculosis in Latin America: a modelling study. Lancet Infect Dis. 2018, 18:1150-1159. 10.1016/S1473-3099(18)30354-2

7. Galgiani JN, Kauffman CA: Coccidioidomycosis and histoplasmosis in immunocompetent persons . N Engl J Med. 2024, 390:536-547. 10.1056/NEJMra2306821
8. Meintjes G, Maartens G: HIV-associated tuberculosis. N Engl J Med. 2024, 391:343-355. 10.1056/NEJMra2308181
9. Rodriguez-Takeuchi SY, Renjifo ME, Medina FJ: Extrapulmonary tuberculosis: pathophysiology and imaging findings. Radiographics. 2019, 39:2023-2037. 10.1148/rg.2019190109
10. Anot K, Sharma S, Gupta M, Kaur D: Disseminated histoplasmosis and tuberculosis: dual infection in a non-endemic region. BMJ Case Rep. 2020, 13:e235531. 10.1136/bcr-2020-235531
11. Nacher M, Couppié P, Epelboin L, Djossou F, Demar M, Adenis A: Disseminated histoplasmosis: fighting a neglected killer of patients with advanced HIV disease in Latin America. PLoS Pathog. 2020, 16:e1008449. 10.1371/journal.ppat.1008449
12. Kauffman CA: Histoplasmosis: a clinical and laboratory update . Clin Microbiol Rev. 2007, 20:115-132. 10.1128/CMR.00027-06