Scientific Research Publishing

Modern Research in Inflammation, 2026, 15(2), 21-26
https://www.scirp.org/journal/mri
ISSN Online: 2169-9690
ISSN Print: 2169-9682

Fever and Cavitary Lung Lesion in an Adolescent: A Case of Histoplasmosis

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How to cite this paper: Foster, C. and Osmond, Z. (2026) Fever and Cavitary Lung Lesion in an Adolescent: A Case of Histoplasmosis. *Modern Research in Inflammation*, **15**, 21-26.
https://doi.org/10.4236/mri.2026.152002

Received: April 28, 2026
Accepted: May 26, 2026
Published: May 29, 2026

Abstract

Background: Cavitary pulmonary lesions in pediatric patients are uncommon and have a broad differential diagnosis, including bacterial necrotizing pneumonia, tuberculosis, septic emboli, and fungal infections. Histoplasmosis is an endemic fungal infection that typically causes mild respiratory illness in children, and cavitary disease in immunocompetent hosts is rare. *Case Presentation*: A 13-year-old previously healthy female presented with fever, cough, chest pain, and shortness of breath. Initial evaluation revealed leukocytosis, tachycardia, and an elevated D-dimer. Chest imaging demonstrated a left upper lobe cavitary lesion with an additional area of consolidation. Pulmonary embolism was excluded on chest CTA, and she was started on empiric broad-spectrum intravenous antibiotics for presumed severe pneumonia. Septic emboli were subsequently ruled out with echocardiography and negative blood cultures. Despite treatment, she remained intermittently febrile. Extensive infectious evaluation was negative for tuberculosis and other common bacterial and viral etiologies. On hospital day 6, urine *Histoplasma* antigen returned positive, confirming acute pulmonary histoplasmosis. Antibacterial therapy was discontinued, and antifungal therapy was initiated. Because of evidence of disseminated infection, the patient was transferred for a higher level of care. *Conclusion*: This case highlights the importance of considering histoplasmosis in pediatric patients with cavitary lung lesions, particularly in endemic regions. Early use of urine antigen testing can expedite diagnosis, avoid unnecessary invasive procedures, and enable timely, targeted therapy.

Keywords

Cavitary Lung Lesion, Histoplasmosis, Immunocompetent, Pediatric, Urine Antigen Testing

1. Introduction

Histoplasmosis is caused by the dimorphic fungus *Histoplasma capsulatum*, which

is endemic to the Mississippi and Ohio river valleys and grows in soil enriched by bird or bat droppings [1]. In the United States, the average annual incidence is estimated at 1 to 2 cases per 100,000 population, though in highly endemic areas 60% to 90% of long-term residents show serologic evidence of prior exposure [2]. The large majority of infections are asymptomatic or self-limited. When symptoms do occur, they are typically nonspecific, including fever, fatigue, and cough, findings that overlap with nearly every common cause of pediatric respiratory illness and make histoplasmosis difficult to distinguish from more familiar diagnoses [1] [2].

Data on histoplasmosis in children remain sparse relative to adults. A structured review of 83 pediatric cases, published between 2000 and 2019 found a mean age at presentation of 9.5 ± 5.5 years, with roughly two-thirds of affected children immunocompromised, where disseminated disease predominated, and overall mortality was 11% [3]. A separate single-center review of 73 children in an endemic area similarly found that most presented with pulmonary disease, with a minority progressing to dissemination [4]. Because the bulk of available pediatric data comes from immunocompromised patients, disease in an immunocompetent child, as in the case reported here, is comparatively underdescribed.

Cavitary lung lesions add a further layer of diagnostic difficulty. In immunocompetent children, cavitation is uncommon and is more often attributed to necrotizing bacterial pneumonia, lung abscess, or tuberculosis than to endemic fungal infection [5], so histoplasmosis is rarely an early consideration even in patients from endemic areas. The stakes of missing it are meaningful: dissemination beyond the lungs can produce hepatosplenomegaly, painful oral ulcers, and skin findings such as erythema nodosum, and it carries a mortality risk that is substantially higher than that of localized pulmonary disease [3]. This case illustrates how a young, immunocompetent patient without a clear exposure history was ultimately diagnosed with cavitary pulmonary histoplasmosis, and underscores the diagnostic value of rapid antigen testing in that process.

2. Case Presentation

A 13-year-old obese female with no significant past medical history presented to the emergency department (ED) with chest pain, cough and shortness of breath of approximately four weeks duration without significant fever or other symptoms. Her symptoms had been continuing despite conservative therapy, such as bed rest and extra oral fluids. On admission, she denied recent weight loss, night sweats, travel out of state or country, exposure to sick contacts, or known exposure to bird or bat droppings. She also reported nothing to indicate immunoincompetency, such as immunosuppressant drug use, recreational drug use, autoimmune conditions or any history of disseminated illness. The initial differential diagnosis included pericarditis, pulmonary embolism, sepsis, viral infection, and pneumonia. On presentation, she was febrile (102.5˚F) and tachycardic (heart rate 149 bpm). Physical examination revealed a well-appearing adolescent with scattered

rhonchi on lung auscultation.

The remainder of the examination was unremarkable, and a respiratory viral panel was negative. Initial laboratory evaluation revealed leukocytosis and an elevated D-dimer level (Table 1). A chest radiograph showed prominence of the upper mediastinum, which the radiologist felt was a possible artifact and recommended repeat two-view imaging (Figure 1). Given the elevated D-dimer and concern for pulmonary embolism, a chest computed tomography angiography (CTA) was obtained. No pulmonary embolism was identified, but the CTA demonstrated two consolidative lesions in the left upper lobe: an inferior cavitary lesion measuring 3.3 cm in maximal diameter, and a superior lesion that was nearly completely solid (Figure 2). These findings prompted admission to the pediatric service for further evaluation and management.

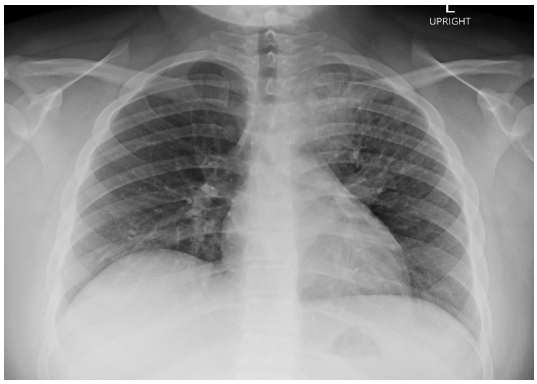

Figure 1. Persistent cavitary lesion in the left perihilar region.

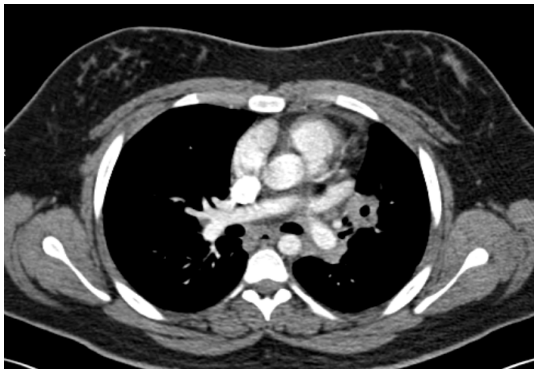

Figure 2. Consolidative appearing foci within the left upper lobe.

Table 1. Key laboratory findings on admission and prior to transfer.

	Hospital Day 1 (6 - 25)	Hospital Day 5 (6 - 29)	Normal value range
WBC (K/cm^2)	18.2 H	13.7 H	4.0 - 13.0
HgB (g/dL)	12.9 L	12.0 L	13.5 - 15.5
Platelet count (K/cm^2)	468 H	491 H	145 - 368
D-dimer (ng/mL)	1887 H	Did not repeat	0 - 500

Following admission, the patient was started on empiric intravenous ceftriaxone, vancomycin, and azithromycin for presumed necrotizing community-acquired pneumonia. She completed a 5-day course of azithromycin, while ceftriaxone and vancomycin were continued. Although her respiratory symptoms gradually improved, she remained intermittently febrile, with a maximum temperature of 100.8˚F after 6 days of intravenous antibiotic therapy.

Because of the cavitary pulmonary lesion, an extensive diagnostic evaluation was pursued. Tuberculosis was excluded with a negative QuantiFERON-TB Gold assay, two negative acid-fast bacilli sputum cultures, and a negative tuberculin skin test, allowing airborne isolation precautions to be discontinued. Blood cultures remained negative throughout hospitalization. Additional testing was obtained for endemic fungal infections, including *Histoplasma*, *Blastomyces*, *Coccidioides*, and *Aspergillus* species, as well as *Bartonella henselae* given a history of significant cat exposure. HIV and *Toxoplasma gondii* testing were negative.

Transthoracic echocardiography showed no evidence of infective endocarditis, and with blood cultures remaining sterile, septic emboli were considered unlikely. Chest CT had incidentally demonstrated hepatomegaly, but liver function tests and a viral hepatitis panel were within normal limits.

On hospital day 6, urine *Histoplasma* galactomannan antigen returned positive at 1.5 ng/mL (reference < 0.2 ng/mL), establishing the diagnosis of pulmonary histoplasmosis. Broad-spectrum antibacterial therapy was discontinued, and antifungal treatment with itraconazole 200 mg was initiated. However, given concern for hepatic involvement due to the hepatomegaly noted on recent CT, liposomal amphotericin B 300 mg was administered instead. Bronchoscopy was deferred given the microbiologic diagnosis and the patient's clinical improvement.

Following initiation of targeted antifungal therapy, the patient was transferred to a higher level of care given the anticipated need for prolonged antifungal treatment and evidence of dissemination.

3. Discussion

Cavitary lung lesions are uncommon in pediatric patients and present a significant diagnostic challenge given the broad range of potential etiologies. In immunocompetent children, these lesions are most often associated with necrotizing bacterial pneumonia, lung abscess or tuberculosis [5]. Because of this overlap, initial evaluation typically requires an extensive work-up to exclude life-threatening conditions before less common etiologies are considered. The clinical and radiographic picture in this case added to diagnostic uncertainty, since fever, cough, leukocytosis, and elevated inflammatory markers are nonspecific findings shared by many of these competing diagnoses.

Even in endemic areas such as the Ohio and Mississippi River valleys, histoplasmosis is typically asymptomatic or produces only mild fever and fatigue. Dissemination, including cavitary lung disease, is a markedly atypical presentation, particularly in a pediatric patient [3]. Recognizing it requires a high index of sus-

picion, and in this case histoplasmosis was only added to the differential after more common causes, like necrotizing pneumonia and tuberculosis, had been largely excluded.

Definitive diagnosis here was achieved through urine *Histoplasma* antigen testing, which returned positive at 1.5 ng/mL (reference < 0.2 ng/mL) and confirmed acute pulmonary histoplasmosis. This noninvasive test was central to establishing the diagnosis and meaningfully changed clinical management. In the largest contemporary pediatric series, diagnostic yield was highest when antigen testing was used alongside other modalities [4]. Importantly, it helped avoid more invasive procedures, such as bronchoscopy with bronchoalveolar lavage or lung biopsy, that are often pursued when a cavitary pulmonary lesion remains undiagnosed.

This case underscores the importance of keeping cavitary pulmonary lesions in children on a broad differential, particularly in patients from or with exposure to endemic regions. Early consideration of fungal infections such as histoplasmosis, paired with rapid antigen-based testing, can shorten time to diagnosis, reduce reliance on invasive procedures, and allow for prompt initiation of appropriate antifungal therapy [3] [4].

4. Conclusion

Histoplasmosis, along with other endemic fungal infections, should remain part of the differential diagnosis for a child presenting with a cavitary pulmonary lesion, even in the absence of a clear exposure history or classic risk factors. Urine antigen testing is a fast, noninvasive tool that can establish the diagnosis early, sparing patients more invasive procedures such as bronchoscopy or biopsy while allowing targeted antifungal therapy to begin without delay.

Acknowledgements

The authors thank Dr. Shilpa Singh and Dr. Huda Adam for their excellent clinical decision-making in the care of this patient, and the entire clinical team at Saint Anthony's Hospital for their contributions to her care.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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