Case Report

Beyond Antibiotic Failure: A Case of Strongyloides and Coccidioidomycosis Coinfection Presenting with Pulmonary Eosinophilia

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Case Report

Beyond Antibiotic Failure: A Case of Strongyloides and Coccidioidomycosis Coinfection Presenting with Pulmonary Eosinophilia

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Abstract

Background: Overlapping endemic infections often present with non-specific systemic features, which could initially lead to delayed recognition and inappropriate treatment. *Strongyloides stercoralis* and *Coccidioides spp.* are rarely encountered together, yet both may cause pulmonary disease, constitutional symptoms, and eosinophilia, complicating diagnosis. Corticosteroid exposure in particular can unmask severe strongyloidiasis, highlighting the importance of early detection. **Case Presentation:** We present the case of a 30-year-old man from the Dominican Republic with recent travel to Brazil and Mexico, who presented with a 3-week history of fever, cough, myalgias, rash, and 13-pound weight loss. Initial treatment for presumed asthma exacerbation and bacterial pneumonia with corticosteroids and multiple antibiotics failed to relieve symptoms. Laboratory evaluation revealed marked eosinophilia (absolute eosinophil count 3,400/μL) and elevated inflammatory markers. Chest CT demonstrated diffuse bilateral tree-in-bud and micronodular opacities. Bronchoalveolar lavage contained 44% eosinophils. Serologic testing was positive for *Strongyloides* IgG, *Coccidioides* IgM/IgG, and β-D-glucan. The patient improved with ivermectin and fluconazole but experienced a relapse of coccidioidomycosis after antifungal discontinuation, requiring reinitiation of long-term azole therapy. **Discussion:** Coinfection with *Strongyloides stercoralis* and *Coccidioides* spp. poses a difficult diagnosis due to overlapping respiratory and systemic manifestations that could mimic common bacterial, fungal or allergic processes. Corticosteroid exposure can precipitate Strongyloides hyperinfection while promoting fungal proliferation, worsening disease severity. Recognition of eosinophilia in patients with a compatible travel history should prompt evaluation for parasitic and fungal etiologies. This case emphasizes the need for early serologic testing and targeted therapy while providing close follow-up to prevent relapses and complications in overlapping endemic infections. **Conclusion:** This case shows the difficulty of diagnosing overlapping infections like *Strongyloides stercoralis* and *Coccidioides*, which can easily be mistaken for bacterial pneumonia. It highlights the risk of giving corticosteroids before ruling out parasitic diseases and stresses the value of screening those at risk. The patient's relapse after stopping treatment reflects the chronic nature of coccidioidomycosis and the need for close follow-up. Clinicians should keep an open, exposure-based approach when evaluating unexplained pulmonary symptoms, especially in people from endemic areas. This case underscores the importance of broad differentials, timely diagnosis, and long-term monitoring in patients with complex overlapping infections.

Keywords: strongyloides stercoralis; coccidioidomycosis; coinfection; eosinophilia; corticosteroids; hyperinfection; endemic infections

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Introduction

Infectious diseases with non-specific systemic symptoms and pulmonary involvement present with significant diagnostic challenges, especially in patients with extensive travel history and prior immunosuppression. This report describes a young man with Concomitant Strongyloides stercoralis infection and primary coccidioides, emphasising the complexity of overlapping endemic infections.

Strongyloides Stercoralis is a soil-transmitted helminth endemic in tropical and subtropical regions, including the Caribbean and South America. Its unique ability for auto-infection allows persistence for decades, often asymptomatically, until immunosuppression, most notably corticosteroid use, precipitates Hyperinfection Syndrome or disease dissemination with mortality rates approaching 70-90 %. [1][2]. Even short-term corticosteroid use for asthma or inflammatory conditions has been reported to trigger severe disease.[3]. Because chronic infection is usually silent, many cases are only recognised once complications arise.[4].

Coccidioidomycosis, caused by *Coccidioides* species, is a fungal infection endemic to arid regions of the Americas, including Mexico, Brazil, and the southwestern United States. Primary infection follows the inhalation of anthroconidia and may initially present as a mild respiratory illness. However, in a subset of individuals, it can evolve into a subacute pulmonary syndrome with systemic features. [5]. Host immune factors, including T-cell-mediated responses, largely determine disease trajectory, with immunocompromised hosts at risk for disease dissemination.[6]. Misdiagnosis is common as early symptoms overlap with community-acquired pneumonia, leading to delayed recognition[7].

The coexistence of these two infections is rare but clinically significant. Both share overlapping features, such as fever, cough, rash, constitutional symptoms, and eosinophilia, making the diagnosis challenging. In this patient, initial corticosteroid use for presumed asthma likely worsened an unrecognised Strongyloides infection, while travel exposure predisposed him to the coccidioides infection. His poor response to multiple antibiotics emphasized the importance of broadening the differential beyond bacterial pneumonia in patients with endemic exposures.

Thus, this case emphasizes three key lessons: the first being to maintain suspicion for parasitic and fungal infections when antibiotics fail in patients with endemic exposure regions. Secondly, to avoid unprotected corticosteroid use in patients at risk of Strongyloides and lastly to plan long-term follow-up, as co-infections may relapse or disseminate despite initial therapy.

Case Presentation

A 30-year-old man from the Dominican Republic with a history of childhood asthma presented with a 3-week history of fever, cough, diffuse myalgias, and progressive fatigue. His symptoms began shortly after attending a large work-related meeting in January. Initial outpatient management with albuterol, prednisone, and doxycycline for presumed asthma exacerbation with atypical pneumonia failed to produce improvement. He subsequently received levofloxacin, which he discontinued after 4 days due to the development of a generalized rash, and later a 5-day course of amoxicillin and azithromycin without clinical benefit.

At presentation to the emergency department, he reported persistent daily fevers up to 39.1°C, ongoing fatigue, intermittent urticarial-type rashes, and diffuse muscle aches. He also endorsed four episodes of hemoptysis, 21 days of nausea, vomiting, and diarrhea, unintentional weight loss of approximately 6 kg, night sweats, arthralgias, and high anxiety. He denied recent sick contacts, genitourinary symptoms, or hot-tub exposure. His travel history included a recent trip to Brazil and a trip to Mexico within the prior year. He lived with his wife, worked primarily in an office setting with occasional soil exposure through agricultural samples, and reported smoking cigars occasionally and consuming around 10 alcoholic drinks per week. He denied illicit drug use.

On examination, temperature was 37.7°C, blood pressure 130/89 mmHg, heart rate 100 beats/min, respiratory rate 18 breaths/min, and oxygen saturation 96% on room air. He appeared anxious and tremulous but was not in respiratory distress. Lung and cardiovascular examinations

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were unremarkable. Photographs of his rash revealed transient erythematous, urticarial lesions on the neck and trunk. The remainder of the physical examination was within normal limits.

Laboratory studies demonstrated a white blood cell count of 11.7 ×10³/μL, hemoglobin 12.1 g/dL, and platelet count 443 ×10³/μL. Chemistry panel was normal except for mildly elevated alanine aminotransferase. C-reactive protein was 7.6 mg/dL, and procalcitonin was minimally elevated at 0.3 ng/mL. Notably, there was marked eosinophilia with an absolute eosinophil count of 3,400/μL. Autoimmune serologies revealed elevated complement (C3, C4), elevated immunoglobulin levels, and a positive antinuclear antibody (1:160, speckled pattern).

Chest radiography demonstrated no focal consolidation or pulmonary edema. Computed tomography (CT) of the chest revealed diffuse bilateral tree-in-bud and micronodular opacities, predominantly in the lower lobes, along with focal consolidation at the left lung base (Figure 1,2). Bronchoscopy with bronchoalveolar lavage (BAL) revealed 44% eosinophils; cytology showed no malignant cells. Respiratory cultures were negative.

Further microbiologic and serologic testing demonstrated a positive Strongyloides IgG antibody, positive Coccidioides IgM and IgG, and a positive β-D-glucan assay. Tests for Legionella, Aspergillus, Cryptococcus, and HIV were negative.

The patient was diagnosed with concomitant Strongyloides infection and pulmonary coccidioidomycosis. He was treated with oral ivermectin for two days and fluconazole 800 mg daily for three months. His symptoms and chest imaging improved significantly, and coccidioidal serologies initially normalized. However, one month after discontinuation of fluconazole, he developed cervical lymphadenopathy with recurrence of elevated coccidioidal titers (1:32), raising concern for disseminated disease. Long-term azole therapy was reinitiated, with ongoing follow-up through infectious disease services.

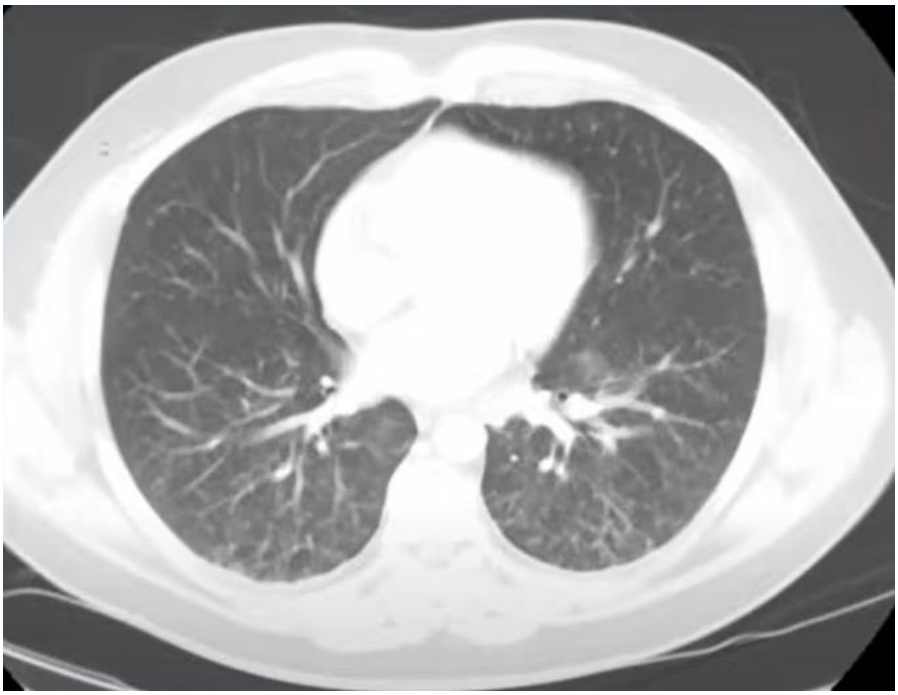

Figure 1. Axial view of Computed tomography (CT) of the chest reveals diffuse bilateral tree-in-bud and micronodular opacities.

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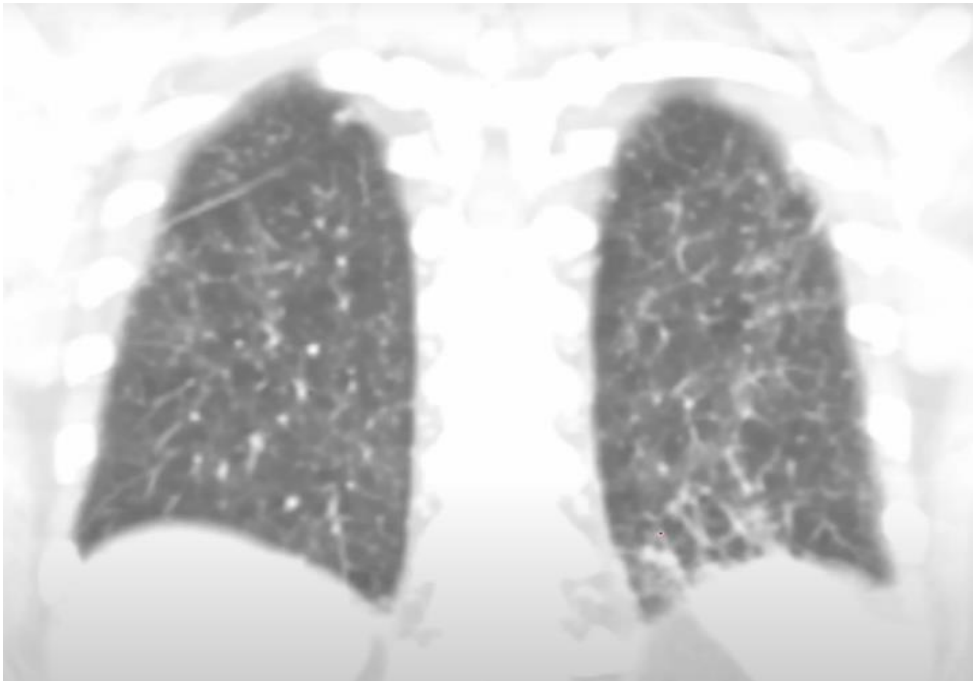

Figure 2. Coronal view of Computed Tomography (CT) of the chest, revealing diffuse bilateral tree-in-bud and micronodular opacities.

Discussion

Coinfection with *Strongyloidiasis* and *Coccidioidomycosis* is exceedingly rare, yet represents a clinically important diagnostic challenge due to overlapping pulmonary, systemic, and immunologic manifestations. Both infections may present with fever, cough, rash, constitutional symptoms, and eosinophilia, frequently mimicking more common conditions such as bacterial pneumonia, hypersensitivity syndromes, or autoimmune disease, leading to delayed diagnosis and inappropriate therapy [8,9].

A key feature in this case is the presence of marked eosinophilia, which should prompt consideration of parasitic infections, particularly in individuals with epidemiologic exposure. *Strongyloides stercoralis* is unique among helminths due to its capacity for autoinfection, enabling chronic infection that may persist for decades in asymptomatic individuals [8]. However, disruption of host immunity—most notably through corticosteroid exposure—can precipitate hyperinfection syndrome or disseminated disease, with mortality rates reported as high as 70–90% [9,10]. Corticosteroids suppress eosinophilic and Th2-mediated immune responses and have also been shown to directly enhance larval proliferation, accelerating the autoinfective cycle [10]. In this patient, early administration of prednisone for presumed asthma likely contributed to worsening of an unrecognized *Strongyloides* infection.

Simultaneously, *Coccidioides* spp., endemic to arid regions of the Americas, including Mexico and parts of Central and South America, are acquired via inhalation of arthroconidia and primarily affect the lungs [11]. While many infections are self-limited, approximately 5–10% progress to complicated pulmonary disease or dissemination, particularly in immunocompromised individuals [11]. The clinical presentation of primary pulmonary coccidioidomycosis often resembles community-acquired pneumonia, with cough, fever, fatigue, and characteristic imaging findings such as nodules or tree-in-bud opacities [12]. In this case, the patient's travel history to endemic regions likely predisposed him to acute coccidioidal infection, further complicating the clinical picture.

The coexistence of these infections may also have immunologic implications. Helminth infections such as *Strongyloides* are known to skew the immune response toward a Th2 phenotype,

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potentially impairing effective antifungal immunity, which relies more heavily on Th1-mediated responses [13]. This immune modulation may increase susceptibility to fungal infections or alter their clinical course, although direct evidence in *Strongyloides–Coccidioides* coinfection remains limited.

Another important diagnostic clue in this case was the elevated β-D-glucan level, which, although nonspecific, supports the presence of invasive fungal infection. However, β-D-glucan may also be elevated in parasitic infections, including strongyloidiasis, which can confound interpretation [14]. Thus, reliance on a single biomarker is insufficient, and a combination of serologic testing, imaging, and clinical context is essential for accurate diagnosis.

The patient's relapse of coccidioidomycosis following discontinuation of antifungal therapy underscores the chronic and potentially relapsing nature of this infection. Current guidelines recommend prolonged azole therapy for patients with severe or disseminated disease, often extending for months to years depending on clinical and serologic response [11]. Rising complement fixation titers, as seen in this patient, are indicative of active or progressive disease and warrant reinitiation of therapy [11].

This case highlights several critical clinical considerations. First, eosinophilia in the setting of pulmonary symptoms and relevant travel history should prompt evaluation for parasitic infections prior to initiating corticosteroids. Second, empiric corticosteroid therapy in undifferentiated respiratory illness carries significant risk in patients with potential latent strongyloidiasis and should be avoided unless parasitic infection has been reasonably excluded or empirically treated [9,10. Third, clinicians must maintain a broad, exposure-based differential diagnosis when initial therapies fail, particularly in patients with international travel or residence in endemic regions.

Finally, this case emphasizes the importance of longitudinal follow-up in patients with endemic fungal infections, as relapse may occur despite initial clinical improvement. Coordinated care involving infectious disease specialists is essential to guide duration of therapy, monitor serologic trends, and detect complications early.

Conclusion

This case represents the complexity of diagnosing overlapping endemic infections in otherwise healthy young patients and underscores the dangers of premature corticosteroid use in unrecognized parasitic disease. The coexistence of *Strongyloides stercoralis* and *Coccidioides* infections created a diagnostic challenge, as both conditions present with similar symptoms that easily masquerade as bacterial pneumonia. The patient's relapse after antifungal discontinuation further emphasizes the chronic and relapsing nature of coccidioidomycosis.

Clinicians should ***adopt a broad, exposure-informed differential diagnosis*** when evaluating patients with unexplained pulmonary and systemic symptoms, particularly in those from or with travel to endemic regions. Screening for *Strongyloides* prior to corticosteroid therapy in at-risk individuals is a simple yet potentially lifesaving step. Finally, the case reinforces the importance of long-term vigilance, as dual infections may follow an unpredictable course requiring prolonged therapy and careful follow-up.

In an increasingly interconnected world, where global travel and immunosuppressive therapies are common, recognizing and anticipating such overlapping infections is not only a matter of clinical acumen but also a safeguard against preventable morbidity and mortality.

Informed Consent Statement: Written informed consent was obtained from the patient prior to the initiation of this study and the drafting of the manuscript. The participant was provided with a detailed explanation of the nature, purpose, and scope of the research, including the intent for academic dissemination and publication. All relevant aspects of participation were discussed, including the use of de-identified clinical data, potential risks and benefits, and the voluntary nature of participation. The patient was given the opportunity to ask questions, all of which were addressed satisfactorily, and was informed of their right to decline participation or withdraw consent at any time without any impact on their medical care. The patient explicitly consented to the inclusion and publication of their clinical information, images (if applicable), and relevant data in a de-identified

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manner to ensure confidentiality and privacy. All procedures were conducted in accordance with applicable ethical standards and institutional guidelines governing human subject research.

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