A case of disseminated histoplasmosis: lung abscess without pneumonia

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Introduction

Disseminated Histoplasmosis, a rare manifestation of a fungal infection, most commonly seen in patients with HIV/AIDS who are particularly susceptible to opportunistic infections. The infection results from inhaling spores of the fungus *Histoplasma capsulatum*. In regions where this fungus is endemic, such as parts of North and Central America, immunocompromised individuals are at heightened risk, which can lead to severe respiratory illness and disseminated infections if not promptly diagnosed and treated. [1]

Therefore, understanding its clinical presentation is crucial for healthcare providers to effectively manage and treat these vulnerable patients regardless of geographical setting. Here we present a case of disseminated histoplasmosis in a severely immunocompromised patient with no recent history of travel to endemic regions.

Case Presentation

This case report involves a 55-year-old male patient with past medical history of HIV/AIDS and substance use (marijuana) who presented to the emergency department (ED) with complaints of a productive cough for 2 weeks with yellow sputum, shortness of breath, and pleuritic chest pain. Past labs showed a HIV viral load of 60,508 and a CD4 count of 24 with non-compliance to HAART therapy. The patient denied fever, night sweats, sick contacts, or any recent travel outside of New York. Patient was born in Haiti, however immigrated to the US a long time ago.

Upon admission, the patient was tachycardic (HR: 129, pulse 133) and tachypneic (RR: 24), but afebrile. On physical exam, the patient appeared cachectic with temporal and generalized muscle wasting. Auscultation of the lungs was notable for diffuse crackles and decreased breath sounds on the left lung fields. The patient also endorsed weight loss , however, was unable to quantify how much and over how long.

Hospital Course

Initial Labs

- Labs: Elevated CRP, ESR, LDH; no leukocytosis (CBC 5.6)
- EKG: sinus tachycardia, preventricular contractions with normal QTc (432)
- The patient received lactated ringer bolus, an empiric antibiotic regimen of Vancomycin , Cefepime, and Metronidazole. The patient was also given acetaminophen, heparin, albuterol-ipratropium, and atovaquone for pneumocystis pneumonia prophylaxis.

Initial Imaging

- Initial chest x-ray showed a thick-walled left posterior cavitary opacity with air-fluid level and surrounding consolidated changes. Chest CT showed thick-walled left posterior subpleural 7.7 cm x 5.2 cm x 7.6 cm cavitary opacity with air-fluid level in surrounding consolidative changes involving left upper and lower lobes, diffuse right upper lobe ground-glass nodular opacities with peribronchial thickening, most consistent with infectious/inflammatory process.

Week 1-2

- Sputum culture, acid-fast bacillus culture x 3, Quantiferon, urine culture, blood culture, legionella, aspergillum IgG, HIV viral load/CD4 count, and a hepatitis panel was ordered
- Toxoplasma IgG/M, histoplasma Ag/galactomannan, cryptococcal Ag, and LFTs were ordered in the following days. Patient continued to remain febrile (Tmax 102.8F). Meropenem was also initiated on day 7. HAART therapy was put on hold until HIV genotype was confirmed.

Week 2-3

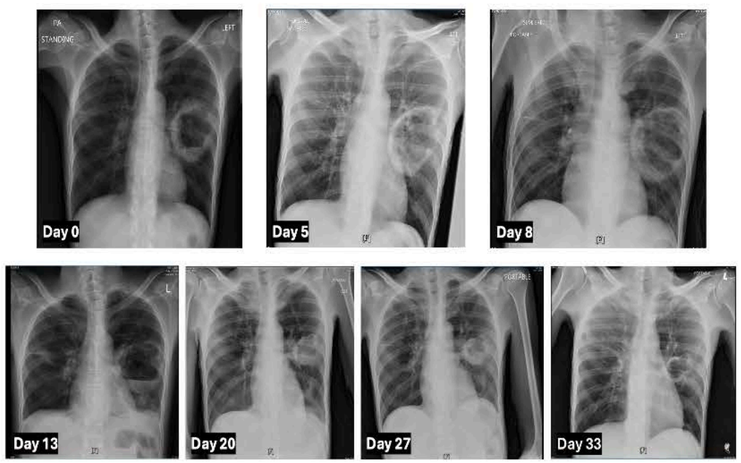
- Due to worsening neutropenia,heme-onc was also consulted at this time and Neupogen 60 mcg/day was administered. A low CD4:CD8 prompted an urgent bone biopsy which ruled out T-cell proliferative disorder.
- In the following days, sputum and urine culture were positive for Histoplasma (in cavitary lesion). A positive beta-D glucan finding was suggestive of systemic invasion.

Week 3-5

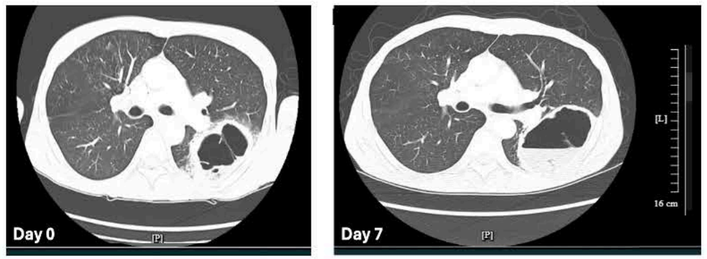
- Patient was started on Liposomal Amphotericin B 175 mg for 2 weeks. After 2 weeks, amphotericin B was transitioned to a loading dose of Itraconazole 200 mg (every 8 hours) for 3 days on an empty stomach. Maintenance dose of Itraconazole 200 mg (every 12 hours) was initiated consecutively.

Week 5

- Itraconazole (CYP3A4 inhibitor) levels were repeatedly measured. QT, LFTs, and overall drug-drug interactions were monitored during this time.
- Repeat chest x-rays were done to monitor cavitary lesion and response to treatment.
- After 5 weeks of hospitalization, the patient's health was optimized for safe discharge with HAART therapy (Biktarvy), Itraconazole, and acetaminophen (as needed).



Serial chest x-ray imaging showing the progression of lung cavitation and its response following treatment over 33 days.



Chest CT of lung cavitation on Day 0 and its progression to lung abscess formation on Day 7.

Discussion

Pulmonary histoplasmosis can progress through several stages based on the severity and duration of the infection. The initial stage often involves asymptomatic or mild symptoms resembling a common cold or flu. This stage is more common in healthy individuals and may resolve spontaneously. However, in immunocompromised patients or those with underlying lung disease, the infection can progress to chronic pulmonary histoplasmosis, (characterized by persistent respiratory symptoms such as cough, chest pain, and shortness of breath). Radiologically, chronic pulmonary histoplasmosis may manifest as lung nodules, cavities, or fibrosis on imaging studies.[2]

Severe pulmonary histoplasmosis can lead to complications such as respiratory failure or disseminated disease. Disseminated histoplasmosis occurs when the fungus spreads from the lungs to other organs, causing systemic symptoms such as fever, weight loss, hepatosplenomegaly, and lymphadenopathy. Diagnosis involves a combination of clinical suspicion, imaging studies (chest X-ray, CT scan), and laboratory tests (serology, fungal culture) to confirm the presence of *Histoplasma* organisms or antigens in biological samples.

According to the National Institutes of Health (NIH) guidelines, management of moderate to severe disseminated histoplasmosis in HIV-infected patients involves induction therapy with intravenous amphotericin B, followed by maintenance therapy with oral itraconazole. Amphotericin B is preferred for initial treatment due to its rapid fungicidal activity and efficacy in controlling severe disease. Lipid formulations of amphotericin B are often used to minimize nephrotoxicity, a common side effect of conventional amphotericin B. Once clinical improvement is observed and the patient is stable, transition to oral itraconazole is recommended for long-term maintenance therapy. Itraconazole is effective in preventing relapse and reducing the risk of recurrence.[1,3-4]

Initiating HAART therapy concurrently with treatment for histoplasmosis, rather than waiting until discharge, is essential to optimize immune function and prevent disease progression in HIV/AIDS patients.[3]

Conclusion

Management of disseminated histoplasmosis in HIV patients requires a multidisciplinary approach involving infectious disease specialists and HIV care providers to optimize outcomes and minimize complications. Obtainig detailed patient history and providing patient education on HAART therapy adherence can be effective measures to prevent disease occurrence.

References

Contact Info