

Histoplasmosis is Mistakenly Diagnosed as Miliary Tuberculosis in Non-Endemic Regions

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Description

The fungus *H. capsulatum*, which is mostly found in the feces and guano of birds and bats, is responsible for the systemic fungal disease known as histoplasmosis. This condition has multiple manifestations and is more severe in its disseminated form and immunosuppressed patients, putting the patient at risk of death if it is not detected early. In endemic regions like South America, invasive histoplasmosis is the most common AIDS-defining condition. There is a high mortality rate when invasive histoplasmosis is mistakenly diagnosed as miliary tuberculosis in non-endemic regions where the diagnosis is less common. The case of an incorrect diagnosis of tuberculosis as invasive histoplasmosis is presented here. In light of the patient's clinical deterioration and positive *Aspergillus galactomannan* antigens, the diagnosis of histoplasmosis was considered later. When confronted with a culture-negative case of miliary tuberculosis that has not improved clinically despite anti-tuberculosis treatment, this case emphasizes the significance of taking into account other opportunistic infections. Additionally, it highlights the diagnostic tools for invasive histoplasmosis that are available in regions where the disease is not endemic. Immunocompromised hosts may present with severe and disseminated histoplasmosis, whereas immunocompetent patients typically present with an asymptomatic or self-limiting infection. This patient, a 26-year-old male on long-term TNF- α inhibitor therapy, presented with six months of diarrhea, fever, and hematochezia. He had previously suffered from ulcerative colitis. He was febrile and hypotensive when he was admitted and his initial diagnosis was pancytopenia. Imaging revealed pulmonary infiltrates, pancolitis, and enlarged mesenteric lymph nodes. After examining the colonic biopsy, it was ultimately determined that the patient had disseminated histoplasmosis.

Neurological Symptoms

Histoplasmosis was confirmed by bone marrow biopsy, which also revealed hemophagocytic lymphohistiocytosis. Amphotericin B, intravenous immunoglobulin, etoposide, and corticosteroids were ultimately administered to the patient. Histoplasmosis usually affects the lungs and is self-limiting and

benign. However, in immunocompromised individuals, it may manifest as a disseminated disease. About 5–10% of cases of disseminated disease involve the Central Nervous System (CNS). Only a few cases of isolated CNS histoplasmosis have been reported in the literature. It typically appears as a single ring-enhancing lesion on imaging studies. It has a wide range of symptoms, from acute, severe infection to progressive, chronic meningitis, which makes it harder to get the right diagnosis, proper testing, and the right treatment. A 57-year-old man from the Midwest of the United States is the subject of our case. In 2019, he was misdiagnosed with gliosarcoma, for which he received appropriate treatment after developing new neurological symptoms, presented for follow-up; on a Magnetic Resonance Image (MRI), a worsening of ring-enhancing brain lesions was observed.

Histoplasmosis of the Central Nervous System (CNS) was identified following a reexamination of surgical pathological cases. If CNS histoplasmosis is not diagnosed promptly, the patient's chances of recovery can be reduced. Histoplasmosis is a fungal infection that is brought on by *Histoplasma capsulatum*. Japan is thought to be free of the disease's endemicity. The majorities of patients who have been diagnosed with histoplasmosis in the past typically have traveled to endemic areas and have been exposed to caves and bat guano. When suspecting histoplasmosis, travel history and risky activities should be thoroughly evaluated because this crucial information may be overlooked. Native American cases have also been suggested, albeit in small numbers. In addition, it is assumed that the recent coronavirus disease 2019 epidemic has reduced the number of travelers and endemic mycoses. However, when treating travelers traveling to endemic areas, clinicians should carefully consider the differential diagnosis of histoplasmosis. An immunocompliant Japanese woman who traveled to an endemic country in the past developed histoplasmosis is the subject of this case report. Even in the absence of risk factors like travel-associated activities or immunodeficiencies, our case report suggests that physicians should still include histoplasmosis in their differential diagnosis. In this case, disseminated histoplasmosis and COVID-19 infection were found in an Argentina renal transplant recipient. A chest Computed Tomography (CT) scan revealed multiple bilateral centrilobular

opacities in a tree-in-bud pattern in both lobes, in addition to the patient's respiratory symptoms. The patient was initially treated for tuberculosis after being diagnosed with bacterial community-acquired pneumonia. Histoplasmosis was identified a month later, and the *Histoplasma capsulatum* LAmB clade was isolated from oral, skin, and sputum lesions. The patient was admitted to the hospital, and intravenous liposomal amphotericin B was administered to begin treatment. During the course of the antifungal treatment, the patient's respiratory symptoms got worse, a new chest CT showed a unilateral lesion that looked like ground glass, and SARS-CoV-2 was found in a new nasopharyngeal sample.

Disseminated Histoplasmosis

Plasma therapy was also given, and the immunosuppressive regimen was changed (everolimus was stopped, mycophenolate mofetil was cut down, and meprednisone was made more powerful). Finally, after five days of oral itraconazole treatment for histoplasmosis, the patient's progress was favorable and he was discharged. Immunocompromised patients, such as those with AIDS, hematologic malignancies, transplant recipients, and those who take corticosteroids for an extended period of time, frequently develop disseminated histoplasmosis. We present the case of a 53-year-old man who underwent renal transplantation in 2013 and had a history of end-stage renal disease caused by uncontrolled hypertension. He brought a 5-day history of dyspnea to the hospital, and blood tests revealed pancytopenia. Peripheral blood smear diagnosis of disseminated histoplasmosis was made quickly. If disseminated histoplasmosis is suspected, we urge doctors to perform a peripheral blood smear. The fungus *Histoplasma capsulatum* is responsible for the fungal infection known as histoplasmosis. It can spread through the droppings of birds or bats and is typically found in a small number of endemic areas in the United States. A serious symptom of the fungal infection, disseminated histoplasmosis is most common in people who already suffer from

immunosuppression. Our patient, a 60-year-old immunocompetent male with a history of significant alcohol abuse that led to end-stage liver failure, presents with an unusual case of disseminated histoplasmosis. The patient began receiving treatment and initially displayed some signs of improvement; however, despite receiving treatment, he continued to deteriorate due to an overwhelming histoplasmosis infection. This case demonstrates the significance of maintaining a high suspicion level even in immune compromised patients with no apparent risk factors. It also demonstrates that an integrated treatment approach necessitates a prompt diagnosis based on a high suspicion index. In immune compromised hosts, histoplasmosis-associated hemophagocytic lymphohistiocytosis is a rate-limiting but fatal disease. The diagnosis of an invasive fungal infection is made even more difficult by unusual clinical presentations. Hemophagocytic lymphohistiocytosis resulting from progressive disseminated histoplasmosis presenting as cellulitis in a patient with systemic lupus erythematosus is the subject of the case described here. To accurately and promptly diagnose opportunistic infections in immune compromised patients, a high suspicion index, histopathology, and molecular diagnostic methods are necessary. Diffuse lymphadenopathy and Cytomegalovirus viremia dominated the initial examinations. A biopsy of an axillary lymph node revealed disseminated histoplasmosis and necrotizing lymphadenitis. He continued to clinically deteriorate despite intensive antimicrobial treatment, raising suspicion of hemophagocytic lymphohistiocytosis. The patient was successfully treated with dexamethasone and etoposide in accordance with the HLH-94 protocol, as she met 5 of the 8 diagnostic criteria from HLH-2004. This case demonstrates that this high-risk patient population can successfully treat and survive acquired hemophagocytic lymphohistiocytosis, despite the high mortality rates and poor clinical outcomes of hemophagocytic lymphohistiocytosis in HIV/AIDS patients. In addition, our case highlights the significance of keeping a broad differential diagnosis for HIV/AIDS patients who present with sepsis.