Case Report: A Case of Coccidiomycosis Confounding a Systemic Lupus Erythematosus Diagnosis

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Introduction: Systemic Lupus Erythematosis is a rare autoimmune disease with prevalence of 20-150 cases per 100,000 in the United States. Despite increased awareness among physicians and improvements in serologic testing, the time to diagnosis from symptom onset is still delayed for many patients, with one study estimating the average time to diagnosis of 6 years. One significant cause of diagnostic delay is due to the heterogeneity in the disease presentation, with symptoms that can present similarly to many other autoimmune, infectious, or hematologic diseases. Many of the symptoms of SLE are nonspecific, such as night sweats, headache, weight loss, fatigue, arthralgias, and rash are very similar to symptoms of Cocciodiodomycosis infection, as in this case. While there are an estimated 150,000 cases of Coccidiodal infections in the U.S annually, 60% of patients remain asymptomatic. Symptomatic patients experience symptoms similar to SLE, which can obfuscate and further delay an already difficult to diagnose rare disease, as in the case of the patient below.

Presentation: A 33-year-old Hispanic male presented with a 3-week history of cough, dyspnea, hemoptysis, fevers, chills, fatigue, myalgias, and loss of appetite. He had recently traveled to Arizona where he initially experienced symptoms and was given a course of antibiotics without improvement. During his admission, the patient tested positive for Coccidioides Ab IgM, Coccidioides titer of <1:2, and Coccidioides immitis Ab <1:2. Respiratory sputum fungal culture grew 2 colonies of Coccidioides immitis/posadasii. The patient also tested positive for anti-dsDNA, anti-RNP, anti-SM, anti-smRNP, and anti-chromatin, but these results were attributed to acute infection rather than SLE. Patient was discharged on a 3-month course of fluconazole and outpatient follow-up with rheumatology.

Patient was subsequently lost to follow-up and presented back to the ED 9 months later with worsening dyspnea, cough, fatigue, rash and weight loss. It was unclear if the patient completed fluconazole treatment, so there was high concern for recurrent or disseminated Coccidioidosis. Patient subsequently underwent a battery tests including CTAP, CTPE, abdominal ultrasound, bloodwork for hepatitis, HIV, legionella, strep pneumoniae, coccidioides, fungitell assay, tuberculosis, and aspergillus, lymph node excision, bone marrow biopsy, skin biopsy, GI biopsy, and renal biopsy. After ruling out infections and malignancy, attention turned to SLE with patient being positive again for SLE markers and positive dsDNA Crithidia (1:160). Patient was started on steroids and hydroxychloroquine with symptom improvement.

Conclusion: In this case of a patient with concomitant Coccidioides and SLE, it is still difficult to delineate if the patient's already existing SLE resulted in an immunocompromised state that allowed the Coccidioides infection to become symptomatic, or if it was the Coccidioides that triggered or unmasked the SLE. Because of the non-specific presentation and inconsistent follow-up, the patient's final diagnosis was not reached until over 1 year after symptom onset and potentially longer given the uncertainty of when symptoms actually began. While increased awareness, improved serological testing, and criteria such as the ACR, EULAR/ACR, and SLICC can guide clinicians, cases such as these emphasize the need for further research to facilitate time to diagnosis in patients with confounding illness.