A 59-year-old Caucasian male with uncontrolled type II diabetes mellitus presented with two weeks of subjective fevers, dry cough, and worsening shortness of breath. Upon presentation, vitals were heart rate of 135 beats per min, afebrile, and tachypnea with O2 saturation 85% on room air. His significant laboratory values were 17% eosinophilia with white blood cells count of 10,200/μL, sodium 125mEq/L, glucose 327 mg/dL, and creatinine 1.7 mg/dL. HIV antibody/antigen, blood cultures obtained prior to initiation of broad-spectrum antimicrobials, tests for histoplasma, mycoplasma, and legionella urinary antigen were all negative. A chest radiograph showed diffuse reticulonodular pulmonary infiltrates. A subsequent computed tomography of the chest showed a right upper lobe cavitary lesion with a milary pattern of diffuse pulmonary micronodularity (Fig. 1a, b). He required intubation and mechanical ventilation shortly after admission. Endotracheal aspirate cultures did not grow any bacterial, mycobacterial, or fungal pathogen. Coccidioidomycosis serology revealed positive IgM antibodies to *Coccidioides* by immunodiffusion suggestive of acute coccidioidomycosis infection. The serum was negative by complement fixation. On day 6 of hospitalization, the patient developed septic shock requiring vasopressors and acute kidney injury requiring hemodialysis. The patient’s clinical course continued to deteriorate despite intravenous liposomal amphotericin B and fluconazole and he eventually expired.

Coccidioidomycosis is an endemic fungal infection in the Southwestern regions of the United States caused by dimorphic fungi *Coccidioides immitis* or *Coccidioides posadasii*, with *C. immitis* being more prevalent in California’s Central Valley. Individuals living in endemic areas with diabetes, HIV, on chemotherapy or other immunosuppressive medications, or pregnant in the third trimester are all at risk of disseminated disease [1].

In correlation with clinical symptoms, serum serology, bodily fluid smear, and culture are used to establish the diagnosis of coccidioidomycosis. In our patient, the fungal cultures were negative due to a low sensitivity of coccidioidomycosis cultures in the setting of miliary coccidioidomycosis which is thought to occur through hematogenous or lymphatic spread and not due to a primary parenchymal lung disease. Sputum cultures are positive in less than 40% of cases and lung biopsy is often required for diagnosis of miliary nodules [2]. Enzyme-linked immunoassays (EIA) and immunodiffusion are the most common tests used to make a diagnosis of coccidioidomycosis. In our patient, the serum was negative by complement fixation which may have been due to the timing of the test. The optimal detection of complement fixation peaks at approximately 1–2 months from the onset of symptomatic infection [3]. Polymerase chain reaction (PCR) assay has been used in our central California hospital laboratory to identify *C. immitis* in specimens including sputum, and bronchoalveolar lavage (BAL) with a more rapid result time of 4 h, and similar specificity and sensitivity when comparing other
serological testing and fungal cultures [4]. PCR was not yet available during our patient’s hospitalization.

In immunosuppressed individuals, disseminated disease such as miliary coccidioidomycosis may occur through hematogenous or lymphatic spread, and lead to respiratory failure [5].

A miliary pattern on chest imaging may be seen with metastatic cancer, tuberculosis, coccidioidomycosis, histoplasmosis, or candidiasis [6].

Amphotericin B and azole in combination or alone are used for the treatment of miliary coccidioidomycosis. Medical providers should optimize testing and management for immunocompromised patients in coccidioidomycosis endemic areas to reduce the risk of infection and the rate of dissemination.

**Declaration of Competing Interest**

No conflicts of interest. Nothing to Declare

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**Consent**

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