CLINICAL VIGNETTE

Cardiac Tamponade in Coccidioidomycosis

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Case

A 30-year-old male with a history of alcoholic pancreatitis and pneumonia presented to urgent care with 1 month of progressively worsening dyspnea on exertion and constant chest discomfort. A chest x-ray demonstrated marked enlargement of the cardiomediastinal silhouette. He was subsequently referred to the emergency department (ED) for further evaluation. On presentation, he reported that four months prior he was diagnosed with pneumonia. He was treated as an outpatient with antibiotics. He reported improvement in pneumonia symptoms but states his breathing did not fully recover. Chest discomfort was worse when supine and with deep inspiration. He also reported experiencing night sweats, myalgia, and fatigue. He denied a history of tobacco use, significant alcohol consumption or drug use.

On physical examination he struggled to speak in full sentences. Lungs were clear to auscultation. Heart sounds were distant and rapid. Jugular venous pulsation estimated at 8cm with negative Kussmaul's sign. No lower extremity edema noted. Laboratory findings were significant for mild normocytic anemia, low albumin, elevated erythrocyte sedimentation rate (ESR) and elevated C-reactive protein (CRP). Cardiac biomarkers were unremarkable. ECG demonstrated sinus rhythm without deviation in ST segments. Echocardiography demonstrated a large circumferential pericardial effusion with equivocal echocardiographic evidence of tamponade (exaggerated respiratory variation across mitral valve, plethora of the IVC and ventricular interdependence). Chest CTA was notable for lobulated consolidation in the basal right lower lobe that may represent atypical mycobacterial or fungal infection. No evidence of pulmonary embolism on CT imaging. He was referred for pericardiocentesis. Pericardiocentesis was performed under echocardiographic guidance with 1150 mL of hemorrhagic drainage. Coccidioides EIA IgM/IgG antibody studies were positive, and he was started on fluconazole. Pericardial fungal culture grew Coccidioides immitis/posadasii. Remaining pericardial studies were unremarkable. Post-pericardiocentesis he reported improvement in dyspnea but had persistence of mild residual pleuritic chest pain. He was treated with high-dose NSAIDs for pericarditis. Colchicine was withheld given severe drug interaction with fluconazole.

Discussion

Coccidioidomycosis, commonly known as Valley fever, is the infection caused by the fungi Coccidioides (Coccidioides immitis and Coccidioides posadasii). The fungus infects via inhalation of arthroconidia in an infected area of the southwestern US or the San Joaquin Valley of California.[1] According to CDC data, 60% of cases come from six counties in Arizona and California, with Kern, Tulare, and San Luis Obispo counties being those in California with the highest rates of infection.[2] Infection rates are also seasonal, with late spring to late fall with the highest incidence of infections in California.[3]

Patients with Coccidioidomycosis can range significantly in severity of illness at initial presentation, with 60% of individuals being asymptomatic or with minimal symptoms, and others suffering from systemic infection.[4] A common presentation is of a community acquired pneumonia, with fever, cough, and chest pain being chief complaints. Imaging may be unremarkable, or may show unilateral dense infiltrates, often with hilar adenopathy in the upper lobes.[5,6]

Extrapulmonary manifestations may also be present, including fever, night sweats, weight loss, arthralgia, or cutaneous manifestations, such as erythema nodosum.[5,6] These extrapulmonary manifestations are only present in <1% of patients presenting with coccidiomycosis.[7]

Pericardial involvement is exceedingly rare, with less than 25 cases reported in literature.[7,8] When the pericardium is involved the presentation may vary from acute pericarditis, pericardial tamponade, effusive-constrictive pericarditis, or chronic pericardial constriction. Cases with pericardial involvement have about 50% mortality.[9] Treatment with amphotericin B or fluconazole was associated with a much higher survivability score (87% vs 20%).[7] Some patients eventually required pericardiectomy.[10]

Given the wide range of presentation of coccidioidomycosis, clinical suspicion is key to diagnosis. Understanding the role of location in illness acquisition is an important factor in social history. Patients with persistent community-acquired pneumonia despite empiric treatment, should raise suspicion for further evaluation, especially if there are new rashes or arthralgias. Upper lobe involvement and hilar or mediastinal adenopathy on imaging should increase suspicion. Serologic testing by enzyme-linked immunoassays for IgM and IgG is used in diagnosis, as well as cultures, though culturing fungi may take several days before growth is seen.[11]

Treatment is dependent on disease severity. Those who are asymptomatic, or with mild or moderate disease may not need treatment, as infection may resolve on its own, though they should still be followed and monitored. Those with severe disease, are pregnant, or are immunocompromised treated with an antifungal. Fluconazole and itraconazole are both routinely used, often for 12-week courses, though treatment may be extended or shortened based on patient response.[6]

Coccidioidomycosis is a rare condition. Increased awareness in areas where the fungus is endemic, as well as high suspicion and imaging could improve treatment.

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