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A Case of Disseminated Coccidioidomycosis: When the Treasure Chest Wall Opened

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Abstract

Coccidioidomycosis is an infection caused by soil-dwelling fungi, *Coccidioides*, that are endemic to the southwestern United States, northern Mexico, and scattered areas of Latin America. It typically presents with pulmonary manifestations that resemble symptoms of bronchitis, pneumonia, and the flu. Extrapulmonary manifestations that involve the skin, lymph nodes, bones, and joints have also been well described, but disseminated coccidioidomycosis initially presenting as chest wall infection without pulmonary symptoms is not. In this article, we present a case of a 33-year-old man who presented with chest wall swelling and eventually diagnosed with chest wall abscesses due to disseminated coccidioidomycosis. We propose that consideration of disseminated coccidioidomycosis in nonresolving swelling, mass, lesions, or abscess especially in endemic areas for coccidioidomycosis and in travelers to the endemic area may prevent the progression and further complications of coccidioidomycosis.

Keywords

coccidioidomycosis, valley fever, fungal infection, dissemination, soft tissue infection

Introduction

Coccidioidomycosis, also known as "San Joaquin Valley fever" or "Desert Rheumatism," is one of the most feared fungal infections in its endemic regions, particularly given the variations in its presentation. It usually presents as a primary pulmonary infection through inhalation of aerosolized arthroconidia. However, the diagnosis can be more challenging when disseminated coccidioidomycosis presents as soft tissue infection. Subcutaneous abscesses are rare form of soft tissue dissemination and can occur without the underlying osteomyelitis.[1,2] It is important to mention that the number of reported cases of coccidioidomycosis has risen substantially during the past 2 decades: 2271 in 1998 to 15 611 in 2018, with a peak of 22 641 in 2011.[3] The increased incidence of coccidioidomycosis and the variations in its presentation mark the importance of ranking disseminated coccidioidomycosis as one of the differential diagnoses when the patient presents with soft tissue infection, especially in endemic areas and in travelers to the endemic area.

Case Presentation

A 33-year-old Hispanic male presented complaining of painful chest wall. He was in his usual state of health until 6 months before he presented. He first developed a flu-like illness with unresolved residual generalized weakness and fatigue. He noticed that his chest wall started to become swollen about 6 weeks prior and developed a "painful rash" on the chest. He was seen by several physicians in the community and was diagnosed with "shingles" and "skin infection." He was treated as such with antiviral and antimicrobials. His rash resolved but swelling and pain of the chest wall continued with new-onset mild shortness of breath. He eventually presented to a local hospital emergency room (ER). On evaluation in the ER, he denied fevers, coughs, night sweats, or chills. His initial workup including complete blood count, complete metabolic panel, urinalysis, and brain natriuretic peptide were within normal limits. His chest X-ray did not show any abnormalities as well (Figure 1). However, his chest computed tomography (CT) scan with contrast suggested multiple abscesses in the mid-portion and the right side of his chest as evidenced by the presence of multiple loculated fluid cystic lesions (Figure 2a and b). These

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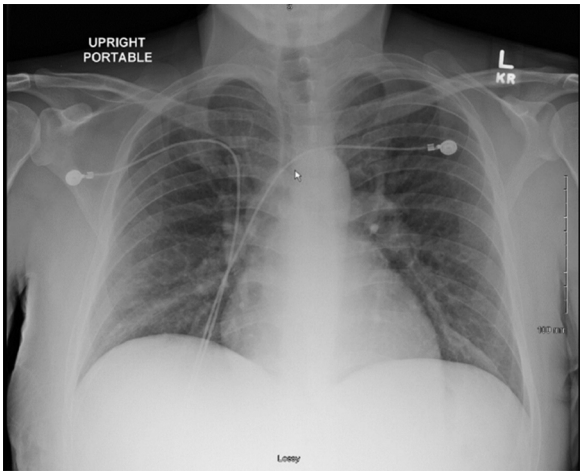

Figure 1. Chest X-ray without evidence of pulmonary coccidioidomycosis.

extended approximately to an area of 14.5 × 9.2 × 3.2 cm. A CT-guided fine needle aspiration was performed, and purulent material was sent for culture.

The patient underwent incision and drainage (I&D), with the placement of negative pressure wound therapy dressing or "wound vac." Repeat chest CT scan revealed decompressed right chest wall abscesses with multiple open wounds (Figure 3). His fine needle aspiration culture grew a "fungus" eventually identified as *Coccidioides immitis.* His coccidioidomycosis serology was positive for immunodiffusion (ID) immunoglobulin (Ig) M and IgG, and complement fixation (CF) of >1:512. He was started on liposomal amphotericin B and continued for 1 week. He was discharged with 400 mg of oral fluconazole to follow-up in the wound care center. His open wounds continued to drain and eventually were closed approximately 6 months later.

After a year of treatment, his CF titers slightly improved to 1:256. He was referred to Valley Fever Institute due to the lack of serological response and persistent fatigue despite complete resolution of soft tissue infection. His physical examination only showed chest wall scars related to his I&D. His fluconazole dose was increased to 800 mg daily. The remaining workup including whole-body bone scan did not reveal any detectable foci of infection. He has been followed-up since then and his CF titers gradually improved to 1:32 after 2 years of high-dose fluconazole. The duration of his treatment remains unknown and is set to be continued until complete serological response (CF of <1:2) is achieved.

Discussion

Coccidioidomycosis is usually a self-limited pulmonary disease, particularly in "immunocompetent individuals." Most of the patients develop mild to moderate respiratory symptoms that resolve within a few weeks. However, in about 1% to 5% of cases, it can disseminate to almost any organ system. Some known risk factors for the extrapulmonary disease include extremes of age, African or Filipino ancestry, pregnancy, and known immunosuppression.[4] However, our patient is a young Hispanic male without any significant medical history, "an immunocompetent host," who has clear evidence of soft tissue dissemination. This indicates that there is little known about the immunogenetics of hosts dealing with this infection.

When this host fungus infects, it activates both innate and adaptive immune systems, which makes its immunopathology interaction enigmatic. The response is chronological, multidimensional, and complex. The fungus activates neutrophils, macrophages, and dendritic cells. Antibodies are not evidently protective and only indirectly indicate the severity of illness like in our case with very high CF titers. In contrast, T-cell responses seem to be important. The responses of T-helper (Th) cells, and the ratios of Th1 and Th2 cells, and Th17 and regulatory T-cells appear to be essential.

There are 2 mechanisms of dissemination in coccidioidomycosis: lymphatic and hematogenous. Pericarditis and supraclavicular lymphadenitis represent the lymphatic dissemination, whereas the central nervous system spread represents the hematogenous dissemination. In this case, perhaps the chest wall soft tissue dissemination is a result of a local spread or an invasion from the adjacent pulmonary foci. This form of local dissemination in our experience is rare.

Furthermore, although the patient is a resident of known coccidioidomycosis endemic region, his diagnosis was significantly delayed. This demonstrates the challenges of making a prompt and appropriate diagnosis of coccidioidomycosis.

Although definitive diagnosis of coccidioidomycosis can be complicated, the diagnosis is typically established by using information from a combination of clinical, radiographic, and laboratory features.[5] The diagnosis becomes more challenging in the absence of pulmonary symptoms, as seen in our case.

A positive culture or histopathological/cytopathological identification of the organism in clinical specimens is considered the gold standard for coccidioidomycosis diagnosis.[5] In our case, a positive culture was established. Nevertheless, serology is most widely used given the relative convenience and rapid availability. Enzyme immunoassay (EIA), ID, and CF are commonly available serologic tests for *Coccidioides* species. CF and ID tests of either tube precipitin antibodies (IgM) or complement-fixing antibodies (IgG) are recommended to be performed in reference laboratories.[6] Similarly, EIA is used to detect IgG and IgM and often performed in local laboratories. When positive, EIA results need to be confirmed by CF or ID.[6] An important point to remember is EIA IgM has a high probability of false-positive rates. As a result, the 2 well-known reference laboratory centers for coccidioidomycosis at the University

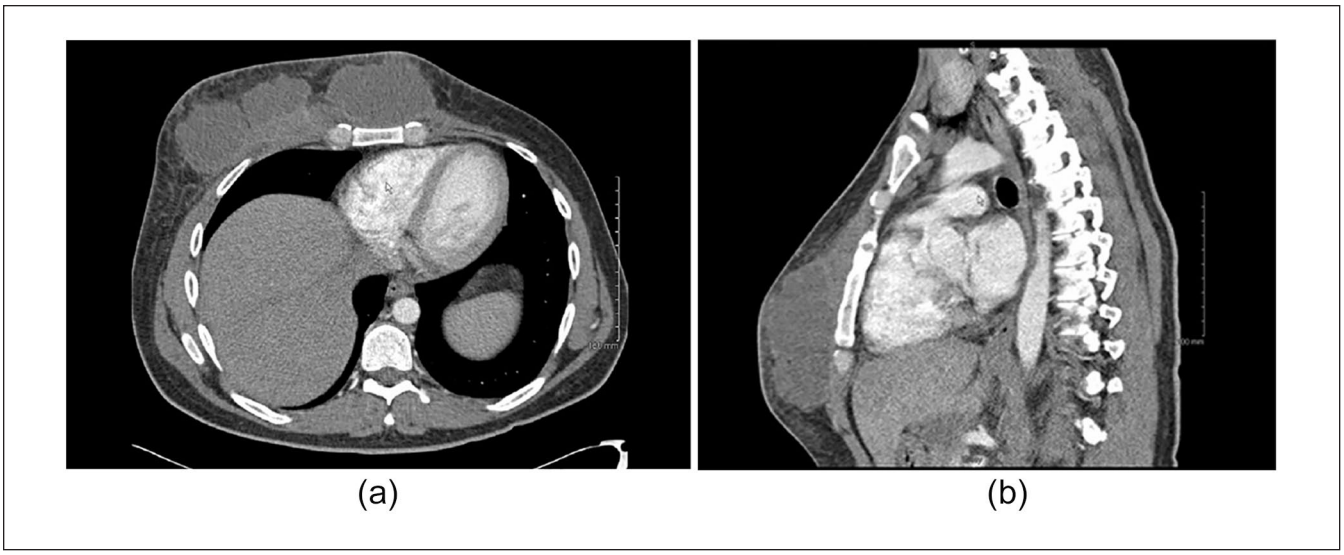

Figure 2. (a) Computed tomography (CT) scan of chest with contrast, axial view revealing right chest wall abscesses. (b) CT scan of chest with contrast, sagittal view revealing right chest wall abscesses.

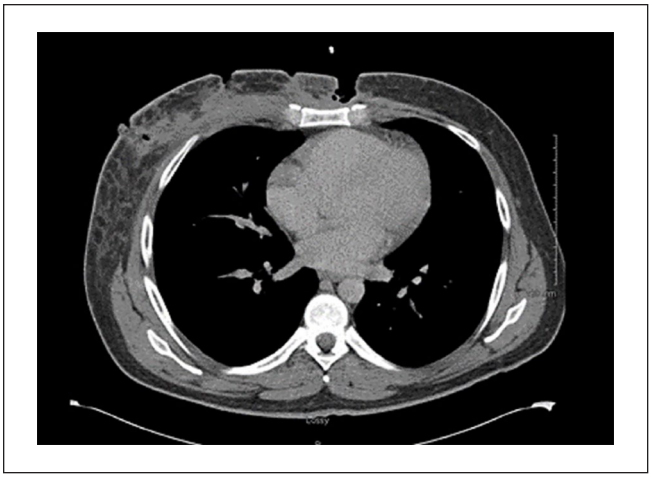

Figure 3. Computed tomography scan of chest with contrast, axial view, revealing decompressed right chest wall abscesses after incision and drainage with several open wounds.

of California, Davis, and Kern County Public Health do not test for EIA in their panel. Overall, serologic testing can be limited and may be insensitive in early infection. Serial serological testing is recommended in cases with high clinical suspicion.[6] A complete blood count with differential for peripheral eosinophilia is seen in 25% to 30% of cases.[6] Radiographic evidence of the pulmonary involvement of the infection may vary from pulmonary infiltrates, pleural effusions, empyema, hydropneumothorax, adenopathy, pulmonary nodules, cavities, tree-in-bud changes, and multifocal ground-glass infiltrates, depending on the severity and the chronicity of the disease.[5]

Subcutaneous tissue infection with development of the abscesses is a well-defined form of disseminated coccidioidomycosis. These foci of infection may be small or quite large as seen in this case. Commonly, these are culture positive like in our case. The immediate temptation is to attempt to drain. However, these fungal abscesses differ from the typical bacterial abscesses in terms of the preferred form of management. While I&D are usually appropriate for bacterial abscesses, if opened, the fungal abscesses will drain for weeks to months and create an unpleasant management problem. Therefore, large volume sequential aspirations are preferred over I&D.[2] The patient in our case underwent I&D of his abscesses after initial fine needle aspiration, while his culture results were still pending. He underwent 6 months of dealing with his chronic open wounds and persistent drainage. In a perfect situation, this could have been avoided with serial needle aspirations and azole therapy if the diagnosis had not been delayed.

Conclusion

Coccidioidomycosis, although typically presents as primary pulmonary infection, could initially manifest with the involvement of any organ system after dissemination. This is usually the case when the primary infection is not diagnosed or was clinically insignificant or self-limiting.

This fact should be considered in a nonresolving soft tissue mass, lesions, or abscess especially in endemic areas for coccidioidomycosis and in travelers to the endemic area. Remote exposures should also be considered, further demonstrating the vital role a thorough patient history plays to ensure the prompt and correct diagnosis. Soft tissue dissemination if diagnosed is often managed by needle aspirations; if any abscess formation is found, with the conjunction of long-term azole therapy.

Authors' Note

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Declaration of Conflicting Interests

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Ethics Approval

Ethical approval to report this case was obtained from IRB Kern Medical with IRB #19083.

Informed Consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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