



Clinical Studies

Recurrence of cutaneous coccidioidomycosis 6 years after valley fever: A case presentation and literature review



Rana Shahab^a, Nasir K. Amra^b, Rola Rabah^b, Justin Saunar^c, Joan Soriano^c, Jaffar A. Al-Tawfiq^{a,d,*}

^a Specialty Internal Medicine, Johns Hopkins Aramco Healthcare, Dhahran, Saudi Arabia

^b Pathology department, Johns Hopkins Aramco Healthcare, Dhahran, Saudi Arabia

^c Microbiology Laboratory, Johns Hopkins Aramco Healthcare, Dhahran, Saudi Arabia

^d Department of Medicine, Division of Infectious Diseases, Indiana University School of Medicine, Indianapolis, Indiana, USA

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ABSTRACT

Coccidioidomycosis is usually acquired by inhalation of spores of *Coccidioides immitis* and *C. posadasii*. The disease ranges from a self-limited acute pneumonia (Valley Fever) to a disseminated disease. We present a 44-year-old healthy male who had patchy hair loss of several months duration resembling discoid lupus. He developed a firm non-scaly red plaque on the right forehead. Initial biopsy showed spongiotic dermatitis, and he was treated with systemic steroids. He then developed forehead and periorbital cellulitis and was treated with systemic antibiotics. A second biopsy showed fungal hyphae, and he was treated with itraconazole 200 mg bid for 4 months beyond clinical resolution. A year later, he presented with intermittent swelling of the right forehead lesion and worsening of the scalp lesions. A forehead biopsy showed interface dermatitis and negative PAS stain for fungi. Scalp biopsy was highly suggestive of discoid lupus and he was started on plaquenil. Many months later, a third biopsy showed fungal infection, and the culture grew *C. immitis*. He was treated with itraconazole. Retrospectively, the patient gave a history of Valley fever 6 years back when he was in Arizona, USA.

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1. Introduction

Coccidioidomycosis is an infection usually acquired by inhalation of spores of a dimorphic fungus of the genus *Coccidioides* (*Coccidioides immitis* and *C. posadasii*) (Welsh et al., 2012). The disease ranges from a self-limited acute pneumonia (Valley Fever) to dissemination in immunosuppressed patients. The most common form of the disease is primary pulmonary coccidioidomycosis and is usually asymptomatic in 60% of patients (Crum et al., 2004; Parish and Blair, 2008). Dermatologic manifestations may occur in 50% of cases during Valley Fever (Garcia Garcia et al., 2015). Immunity to exogenous reinfection occurs in most patients with primary disease (Hector and Laniado-Laborin, 2005). Here, we present a case of cutaneous coccidioidomycosis reoccurring 6 years after the initial disease.

2. Case Presentation

On February 7, 2012, a 44-year-old male was seen at an outside clinic for a two-month history of hair loss and he was treated with doxycycline for presumed scalp folliculitis, resembling discoid lupus. Two of the

patient's siblings had lupus erythematosus. Then, he presented to us with a firm non-scaly red plaque on the right forehead. On examination, there were two atrophic patches of hair loss with hyperpigmented center suggestive of scarring alopecia, discoid lupus, or orlichen planopilaris. He was treated with clobetasol propionate 0.05% lotion once a day.

One week later, he presented with a firm plaque, 3 × 3 cm, on the right forehead with an associated mild swelling of the surrounding area with no scales. The working diagnoses were calcinosis cutis or discoid lupus. Antinuclear antibody (ANA) was negative. A 3-mm skin biopsy was taken and he was started on a tapering dose of oral prednisone. The right forehead biopsy showed spongiotic dermatitis with chronic perivascular and periadnexal inflammation with numerous eosinophils, suggestive of arthropod bite, drug reaction, or urticarial reaction (Fig. 1).

Ten days later, he presented with a swelling of the right side of the face associated with purulent discharge of one-day duration. He had no fever or chills. Examination showed a mass with fluctuation on the forehead. Prednisone was discontinued, and he was started on amoxicillin-clavulanic acid. The following day, the swelling got worse and he was unable to open his right eye. He was admitted as a case of eosinophilic cellulitis. Vancomycin was started in addition to pain medications. WBC was 9500 with 7% eosinophilia and IgE 7420. Next day, the swelling markedly improved and the forehead lesion was crusting

* Corresponding author. Tel.: +966-13-877-3524; fax: +966-13-877-3790.

E-mail addresses: jaffar.tawfiq@jhah.com, jaltawfi@yahoo.com (J.A. Al-Tawfiq).

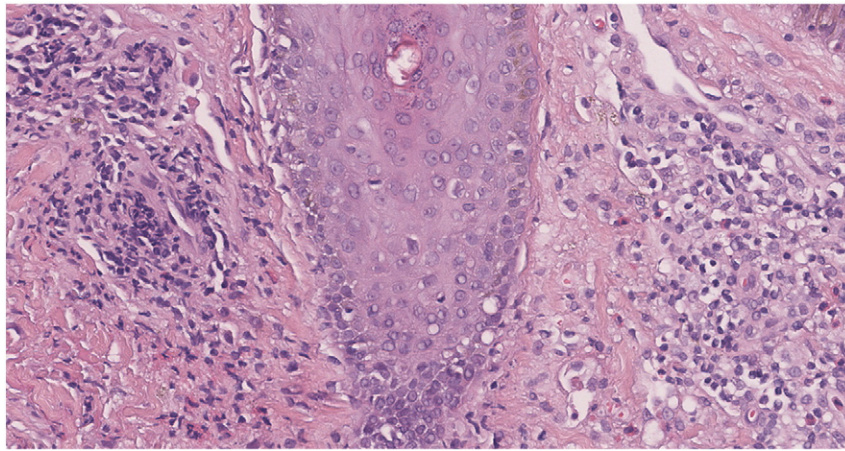


Fig. 1. First biopsy demonstrating superficial spongiotic dermatitis with eosinophils (hematoxylin and eosin, $\times 280$ magnification).

and erythematous. Culture of forehead lesion grew coagulase-negative *Staphylococcus*. He did well and was discharged on clindamycin.

On follow-up 1 week after discharge, there was a 2-cm infiltrated plaque on the right forehead with overlying crust. Two biopsies were taken for hematoxylin and eosin stain and fungal, mycobacteria and bacterial cultures. The patient was treated with itraconazole 200 mg bid and clindamycin was continued as the infectious disease physician felt that the lesion resembled coccidioidomycosis. Histopathology showed fungal granulomatous inflammation (Fig. 2) and the tissue grew a mold. However, no further characterization was done by the microbiology laboratory. The patient was kept on itraconazole for a total of 4 months until clinically the forehead lesion resolved completely.

In April 2013, he presented with pruritic scalp lesions, hair loss, and a mobile cystic mass on the right forehead. Biopsies showed interface dermatitis with a superficial, perivascular interstitial, and perifollicular lymphohistiocytic infiltrate extending to the deep margins suggestive of lupus erythematosus. GMS stain was negative for fungal organism. ANA was negative and the patient was started on plaquenil 200 mg bid and tacrolimus ointment to be applied to the skin lesions. The forehead lesion worsened and a repeat biopsy showed granulomatous dermatitis with fungal elements (Fig. 3) and the culture grew *C. immitis* (Fig. 4). The patient was started on voriconazole 200 mg bid initially then it was changed to itraconazole after a couple of weeks for compliance. In July 2014, Coccidioides antibodies level was 1:8 and then the patient recalled having

a diagnosis of Valley Fever in Arizona, USA in 2006 for which he was treated. *C. immitis* titer was monitored and remained at 1:2 after completing 12 months of therapy. He was seen on a regular follow-up and the forehead lesion healed.

3. Discussion

Coccidioidomycosis is usually acquired through inhalation of the spores of *C. immitis* or *C. posadasii*. *C. immitis* is geographically found in central and southern California including the San Joaquin Valley whereas *C. posadasii* is found in Arizona, New Mexico, Texas, Nevada, and Utah (Brown et al., 2013). The most frequent manifestation of coccidioidomycosis is being asymptomatic or as a mild upper respiratory infection. Other patients may develop an acute respiratory infection 1 to 4 weeks after exposure (Crum et al., 2004; Parish and Blair, 2008). Extrapulmonary dissemination may occur in 22% of patients with racial difference where African Americans develop disseminated bony disease and Filipinos develop cutaneous or central nervous system disease (Crum et al., 2004), with predominance among immunocompromised patients (Adam et al., 2009). Skin involvement presents with papules, nodules, ulcers, warty lesions, or acne like anywhere on the body but most commonly on the face. The current case did not raise the diagnosis of coccidioidomycosis due to the non-availability of a previous history, and thus, the diagnosis was not considered as was stated previously

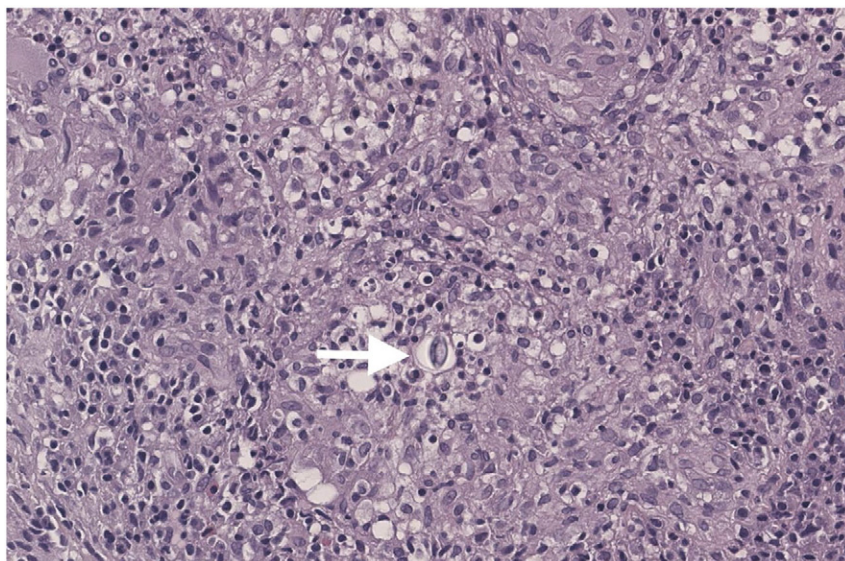


Fig. 2. Second biopsy with fungal granulomatous inflammation with arrow pointing at Coccidioidomycosis endospore (hematoxylin and eosin, $\times 880$ magnification).

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