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Palmar Rash in a Patient With Valley Fever Pneumonia: A Diagnostic Challenge

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Abstract

Coccidioidomycosis, also known as Valley Fever, is a fungal infection endemic to the southwestern United States. While the disease typically presents with limited pulmonary symptoms, extrapulmonary symptoms can also be present. In this case, an 84-year-old female developed palmar erythema following initiation of treatment with fluconazole for management of symptomatic pulmonary Coccidioidomycosis. On exam, the patient had bilateral palmar erythema that began improving a few weeks after initiation of treatment. It was ultimately resolved following a short course of steroids and completion of treatment with fluconazole. This case illustrates a rare, reactive palmar eruption occurring after the initiation of antifungal therapy for pulmonary coccidioidomycosis, underscoring its role as a great mimicker and the diagnostic challenge of paradoxical or reactive phenomena during treatment.

Categories: Family/General Practice, Infectious Disease, Dermatology
Keywords: coccidiomycosis pneumonia, disseminated coccidiomycosis, great mimicker, palmar rash, valley fever

Introduction

Coccidioidomycosis, commonly known as Valley Fever, is a fungal infection caused by the dimorphic fungi Coccidioides immitis and Coccidioides posadasii, which are endemic to arid and semi-arid regions of the southwestern United States, Mexico, and parts of Central and South America [1]. Inhalation of airborne arthroconidia leads to infection, commonly manifesting as pulmonary disease. Following inhalation, arthroconidia transition from barrel-shaped spores to spherules within the lungs [2]. These spherules undergo internal septation to form endospores, which are released into surrounding tissues upon rupture, allowing further propagation of infection and potential dissemination throughout the body [2].

While coccidioidomycosis primarily affects the lungs, extrapulmonary spread can involve the lymphatics, central nervous system, skin, and musculoskeletal system [3,4]. Cutaneous manifestations are broadly categorized as either reactive or organism-specific. Reactive presentations, such as erythema nodosum, erythema multiforme, Sweet's syndrome, or a generalized morbilliform eruption, are most commonly associated with primary pulmonary infection rather than a primary rash [5,6].

In contrast, organism-specific cutaneous lesions typically occur secondary to hematogenous dissemination and rarely result from primary skin inoculation. Disseminated cutaneous lesions may appear as papules, plaques, nodules, ulcerations, abscesses, or lesions mimicking cutaneous T-cell lymphoma (e.g., mycosis fungoides), and they can occur anywhere on the body [7,8].

Primary cutaneous coccidioidomycosis is uncommon and typically self-limited, presenting as ulcerated nodules with regional lymphadenopathy and sporotrichoid spread [9]. Diagnosis is established when characteristic clinical and laboratory features are present. Patients typically lack pulmonary involvement but have clear evidence of traumatic inoculation, followed by an incubation period of one to three weeks. The lesion usually appears as a painless, ulcerated nodule or plaque with a chancriform morphology. Early serologic testing shows a positive precipitin reaction and coccidioidin skin test, while complement fixation remains negative or low. Lymphadenopathy and sporotrichoid nodules may develop, and the lesion often resolves spontaneously after several weeks [10]. An estimated 150,000 new cases of coccidioidomycosis occur annually in the Southwestern U.S., with a notable rise in incidence between 2001 and 2010, particularly in Arizona, where rates increased from 12 to 58.2 per 100,000 population [1,6]. Factors contributing to this trend include population growth, increased numbers of immunocompromised and elderly individuals, climate change, and land development activities that disrupt endemic soil.

This report describes an 84-year-old woman currently residing in Arizona who developed a bilateral palmar eruption following fluconazole initiation for pulmonary coccidioidomycosis, showcasing the diagnostic challenge in distinguishing cutaneous manifestations associated with medications and underlying

infections.

Case Presentation

An 84-year-old woman presented to the emergency department with fever and respiratory distress. On arrival, her vital signs were notable for hypotension (blood pressure 75/47 mmHg), hypoxemia (oxygen saturation 86-98%), and a heart rate of 69 bpm. Initial laboratory results revealed leukocytosis (WBC 11.8 × 10^9/L), elevated creatinine (1.56 mg/dL), and elevated liver enzymes (alanine aminotransferase (ALT) 89 U/L, aspartate aminotransferase (AST) 81 U/L). Additional laboratory findings are provided in Table *1*.

Test	Result	Reference Range
Initial Evaluation in Emergency Department:		
White Blood Cells (WBC)	11.8 X 10^9 /L	3.6-11.1 x 10^9 /L
Hemoglobin	10.2 g/dL	11.5-16.0 g/dL
Creatinine	1.56 mg/dL	0.57-1.25 mg/dL
Estimated Glomerular Filtration Rate (eGFR)	33 ml/min/1.73 m^2	>90 ml/min/1.73 m^2
Alanine Aminotransferase (ALT)	89 U/L	6-55 U/L
Aspartate Aminotransferase (AST)	81 U/L	5-34 U/L
Brain Natriuretic Peptide	54 pg/mL	0-100 pg/mL
Lactic Acid	1.1 mmol/L	0.5-2.0 mmol/L
Procalcitonin	0.74 ng/mL	0.05-0.15 ng/mL
Troponin	<0.04 ng/mL	0-17 ng/mL
COVID-19 Testing	Negative	Negative
Influenza Testing	Negative	Negative
Inpatient Infectious Workup (Hospital Day 1, Hospital Day 2):		
Streptococcus pneumoniae Urinary Antigens	Negative	Negative
Legionella pneumophila Urinary Antigens	Negative	Negative
Blood cultures	unremarkable	-
Respiratory culture	Rare gram-positive cocci	-

TABLE 1: Initial Laboratory and Microbiological Findings During Hospitalization for Pulmonary Coccidioidomycosis

Results demonstrate elevated leukocytes, creatinine, and liver enzymes. Infectious testing was negative for Streptococcus pneumoniae and Legionella pneumophila; blood cultures were unremarkable. Rare gram-positive cocci were present in the respiratory specimen.

COVID-19 and influenza testing were negative. Chest radiography demonstrated extensive right upper lobe consolidation consistent with pneumonia (Figure *1*). Her Pneumonia Severity Index (PSI) score of 104 placed her in Risk Class IV (91-130 points), which is associated with an estimated 8.2-9.3% 30-day mortality [11]. The patient received a 2-liter intravenous fluid bolus for concern of dehydration, supplemental oxygen at 3 L/min via nasal cannula, and was empirically treated with ceftriaxone and azithromycin. Given her high risk of mortality, she was admitted for inpatient management of acute hypoxemic respiratory failure secondary to community-acquired pneumonia.

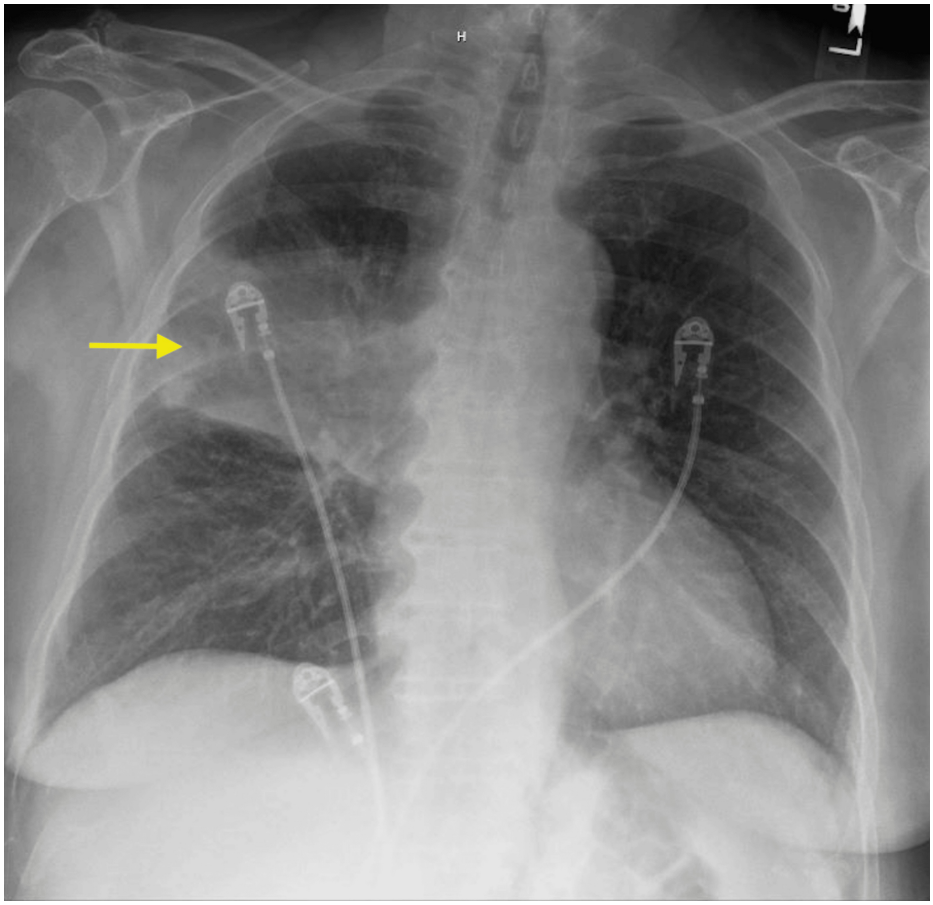


FIGURE 1: Chest Radiograph Demonstrating Right Upper Lobe Consolidation at Initial Presentation

The yellow arrow indicates right upper lobe consolidation.

On hospital day 1, the patient remained on supplemental oxygen. Serologic testing for Coccidioides spp. was obtained, and antimicrobial therapy was broadened to cefepime and doxycycline due to clinical deterioration. By hospital day 2, her respiratory status had improved, and her supplemental oxygen was reduced from 3 L/min to 1 L/min via nasal cannula. A bacterial etiology remained unclear as urinary antigens for Streptococcus pneumoniae and Legionella pneumophila were negative, and blood cultures remained unremarkable. Respiratory cultures showed rare gram-positive cocci, although final results were pending. Cefepime and doxycycline were continued based on her clinical improvement.

On hospital day 3, Coccidioides serologies returned with a positive IgM and negative IgG, suggestive of acute infection. In addition to cefepime and doxycycline, fluconazole 400 mg IV daily was initiated as it is the first-line treatment for Coccidiomycosis. The patient was transitioned to room air from nasal cannula as her clinical course was improving.

However, liver enzymes began to rise modestly, with ALT 115 U/L and AST 97 U/L on day 4. Prior to treatment with fluconazole, transaminases were already elevated at admission and continued to rise throughout hospitalization. At discharge on day 5, ALT and AST had further increased to 248 U/L and 245 U/L, respectively. Fluconazole therapy was discontinued at discharge because of the continued rise of liver enzymes (Table *2*).

Time	ALT (U/L)	AST (U/L)
Admission	89	81
Inpatient Day 4	115	97
Discharge	248	245

TABLE 2: Liver Enzyme Trends During Hospitalization for Pulmonary Coccidioidomycosis

Results demonstrate progressive elevation in liver enzyme levels throughout hospitalization. ALT: alanine aminotransferase, AST: aspartate aminotransferase

Three days post-discharge, at a scheduled transition of care management appointment, the patient presented to her primary care provider with a painful bilateral palmar eruption (Figure *2A*). The patient reported noticing the eruption during hospitalization.

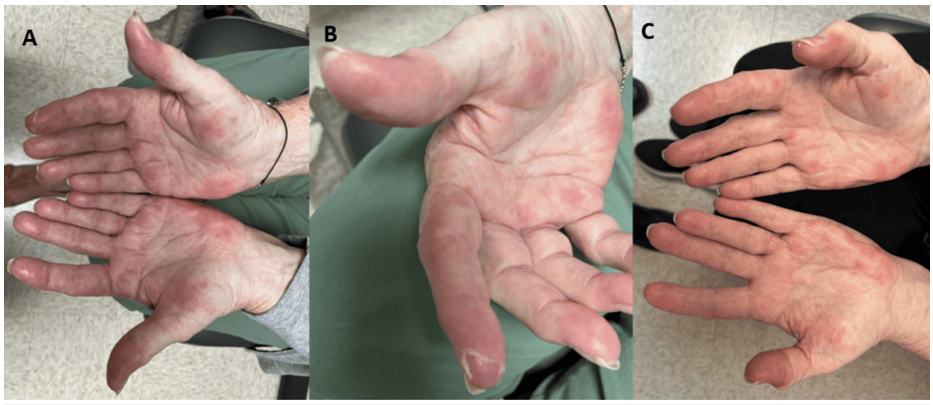


FIGURE 2: Photographs of Both Palms Demonstrating Progression of Palmar Eruption

Panel A: Initial presentation of bilateral palmar erythema with ill-defined patches three days after discharge.

Panel B: Close-up photograph showcasing focal desquamation on right index finger observed two weeks post-discharge.

Panel C: Improvement of the palmar eruption bilaterally after desquamation, approximately four weeks after discharge.

On initial examination, the eruption demonstrated patchy, ill-defined, dusky-red erythema with a symmetric, bilateral distribution predominantly involving the palmar periphery. The eruption was painful and isolated to the palms.

The differential diagnosis for the rash included cutaneous manifestations of liver injury, fluconazole use, or disseminated coccidioidomycosis. Since the eruption remained despite fluconazole discontinuation, a coccidioidomycosis-associated eruption was favored, and fluconazole therapy was reinstated at a reduced oral dose of 200 mg daily to treat the underlying infection. She was also prescribed a 10-day course of prednisone 20 mg daily to help improve persistent respiratory symptoms and palmar erythema.

To address the elevation in liver enzymes, other potentially hepatotoxic medications were held (e.g., allopurinol, non-steroidal anti-inflammatory drugs (NSAIDs), atorvastatin), with acetaminophen limited to a maximum of 1g daily.

At a subsequent visit the following week, liver enzyme levels improved, allowing fluconazole to be increased to a therapeutic oral dose of 400 mg daily. The palmar eruption remained inflamed, with focal desquamation observed on the tip of the right index finger (Figure *2B*). Overall, the eruption was progressing towards resolution as the patient reported reduced finger swelling.

During the next two weeks, both palms underwent extensive desquamation before improvement. At four

weeks post-discharge, the eruption had substantially subsided (Figure 2C). At this visit, fluconazole was increased to 800 mg daily, with a planned treatment duration of three months. The escalation in fluconazole therapy was supported by the resolution of the eruption and the patient's tolerance of fluconazole 400 mg daily without any further increase in liver enzymes. The patient was seen out-patient every two weeks to allow for monitoring of symptoms, including her intense fatigue and residual respiratory symptoms.

A working diagnosis of the rash as a cutaneous manifestation of coccidioidomycosis was favored given the coinciding pulmonary infection, lack of explanatory causes like no prior history of chronic liver disease or alcohol use, and non-progressive lesions with continued fluconazole therapy. The possibility of association with medication or liver injury can not be conclusively excluded based on the available clinical findings and testing.

After three months of 800 mg daily fluconazole, the dosage was reduced to 400 mg daily. As the liver enzymes normalized, allopurinol and atorvastatin were reintroduced to the patient's daily regimen for gout and hyperlipidemia. The patient tolerated the resumption of both medications while continuing fluconazole, without recurrence or development of adverse symptoms. Over the next two months, the patient was closely monitored with serial Coccidioides serologies and liver function tests (LFTs).

During this treatment period, IgM and IgG immunoassay measurements declined (Figure 3). The inflammatory markers C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) also showed an overall downward trend with fluconazole therapy (Figure 4).

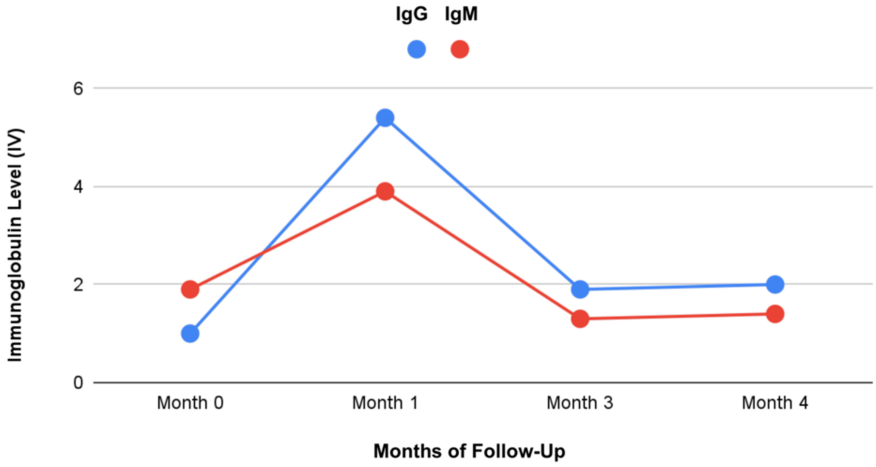


FIGURE 3: Serial Plotting of Coccidioides IgM and IgG Over Time

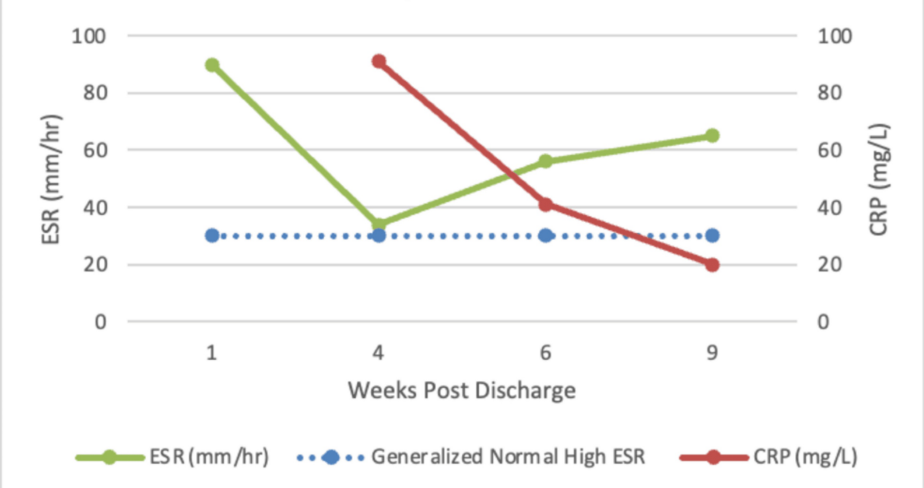


FIGURE 4: Serial Plotting of Inflammatory Markers During Outpatient Care

ESR: erythrocyte sedimentation rate, CRP: C-reactive protein

The patient ultimately completed a six-month course of fluconazole therapy with clinical and serologic improvement, normalization of liver enzymes, and resolution of palmar erythema.

Discussion

Coccidioidomycosis, caused by the dimorphic fungi Coccidioides immitis and Coccidioides posadasii, is a systemic fungal infection endemic to the southwestern United States, parts of Mexico, and Central and South America [1,12]. The most common presentation is a primary pulmonary infection. However, extrapulmonary and cutaneous manifestations are typically associated with disseminated disease occurring in about 1% of cases [13]. Primary cutaneous coccidioidomycosis is rare, typically self-limited and presents as ulcerated nodules with no pulmonary involvement [9]. Alternatively, secondary cutaneous lesions may appear as papules, plaques, nodules, ulcerations, or abscesses, which most frequently occur on the head and neck [7,8].

The development of palmar erythema during fluconazole therapy posed a diagnostic challenge in distinguishing between medication or infectious causes. The palmar location of the rash was atypical and initially raised a broad differential including infectious causes (e.g., syphilis, Rocky Mountain spotted fever, scabies), inflammatory causes (e.g., eczema, psoriasis), drug reactions (e.g.,erythema multiforme), palmar erythema secondary to liver disease/injury, and erythema nodosum which does not typically have palmar involvement.

The onset of the palmar rash during hospitalization for pulmonary coccidioidomycosis supports the possibility of a coccidioidomycosis cutaneous manifestation, though the underlying mechanism remains unclear. However, serial serologic testing during outpatient follow-up raised concern for evolving or persistent infection, potentially due to subtherapeutic dosing or insufficient duration of fluconazole during the initial course of antifungal therapy.

The pathophysiology of the palmar rash remains uncertain, with direct cutaneous dissemination and inflammatory response to the underlying infection as possibilities. While hematogenous dissemination can not be excluded, the timing of the palmar eruption's appearance after initiating antifungal therapy also raises the question of a reactive or paradoxical immune-mediated phenomenon without direct fungal involvement of the skin. Although a phenomenon resembling an immune reconstitution inflammatory syndrome (IRIS) was considered, the patient had no documented immunological deficits, making IRIS an unlikely cause [14].

The patient's recent pulmonary coccidioidomycosis infection guided the physician's suspicion of a coccidioidomycosis-associated cutaneous manifestations. Although serology confirmed a pulmonary coccidioidomycosis infection, direct fungal involvement of the skin could not be confirmed without a palmar biopsy with histopathologic examination or fungal culture showing spherules with endospores. As a result, an inflammatory or medication-induced cause remains possible. This case shows the difficulty in definitively determining the cause of cutaneous eruptions alongside antifungal therapy.

Treatment for coccidioidomycosis typically includes fluconazole, but any oral azole therapy may be utilized

[15]. Duration of treatment varies depending on severity. In this case, IV fluconazole therapy was initiated during hospitalization but was discontinued prior to discharge to address the elevated liver enzymes. At the first outpatient appointment, fluconazole was gradually restarted with 200 mg orally daily. Once liver enzymes improved, dosage was increased to 400 mg daily, which is therapeutic. Following the resolution of the palmar eruption, fluconazole was raised to 800 mg daily. The palmar improvement does not establish a fungal cause because of the simultaneous use of prednisone.

The cause of the patient's elevated liver enzymes remained unclear. AST and ALT were elevated prior to the initiation of fluconazole therapy, suggesting that fluconazole was not responsible for the initial findings. The subsequent rise in liver enzymes could have reflected disseminated coccidioidomycosis, hepatic injury from hypoxic respiratory failure, and medication-associated hepatotoxicity. Transaminases ultimately returned to normal during continued fluconazole therapy.

This case contributes to the growing body of literature on the diverse dermatologic presentations of coccidioidomycosis and highlights the importance of considering this diagnosis when evaluating unusual rashes in endemic regions. Cutaneous findings, such as erythema nodosum, erythema multiform-like eruptions, or nonspecific exanthems may be among the earliest signs of infection and can precede or accompany pulmonary symptoms. Early recognition of these immune-mediated skin lesions, particularly in individuals with known environmental exposure, can prompt timely diagnostic testing and reduce delays in treatment. This case highlights the necessity of considering both inflammatory and medication-induced cutaneous manifestations when eruptions develop during treatment for pulmonary coccidioidomycosis in the absence of histopathologic analysis of dissemination.

Conclusions

Cutaneous manifestations of coccidioidomycosis are a rare but important consideration in patients with a palpable palmar rash and prior pulmonary symptoms, especially in endemic regions. This case highlights the need for clinical suspicion of possible disseminated coccidioidomycosis and appropriate mycological testing in diagnosing atypical dermatologic presentations during fungal infections. Prompt recognition of dissemination and histopathologic confirmation through skin biopsy can establish direct fungal involvement and eliminate other possibilities. This case adds to the literature on unusual dermatologic presentations associated with coccidioidomycosis and highlights the diagnostic challenge of rashes that appear during the treatment course.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Acquisition, analysis, or interpretation of data: Abdul Waheed, Jennifer Blackwood, Benjamin Ihms, Rachel Lowe, Muheema Husain

Drafting of the manuscript: Abdul Waheed, Jennifer Blackwood, Benjamin Ihms, Rachel Lowe, Muheema Husain

Critical review of the manuscript for important intellectual content: Abdul Waheed, Jennifer Blackwood, Benjamin Ihms, Vidhya L. Kumbera, Rachel Lowe, Muheema Husain

Supervision: Abdul Waheed, Benjamin Ihms, Vidhya L. Kumbera

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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