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Fusarium solani Species Complex as an Opportunistic Pathogen: A Case Series among Individuals with Underlying Conditions in a Tertiary Care Center

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Case Series

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ABSTRACT

Introduction: Fungal infections are an emerging threat in immunocompromised individuals. Among the causative agents, members of the *Fusarium solani* species complex (FSSC) are notable opportunistic pathogens. FSSC infections are associated with significant morbidity and mortality, largely because of the organism's aggressive nature and broad intrinsic resistance to several antifungal agents, particularly fluconazole, itraconazole, and echinocandins. **Case presentations:** This report describes three distinct cases of FSSC infection or isolation with varied clinical manifestations. The first case involved a 67-year-old male farmer who developed fungal keratitis following ocular trauma; corneal scraping cultures yielded an FSSC isolate. The second case involved a 55-year-old patient with a history of chronic obstructive pulmonary disease (COPD) who presented with an acute exacerbation. A computed tomography (CT) scan of the chest revealed a small area of consolidation, and sputum culture yielded an FSSC isolate, although airway colonization could not be excluded. The third case involved a 57-year-old patient with a history of alcoholic liver disease who developed progressive respiratory failure requiring mechanical ventilation. There was no clinical response to broad-spectrum antibiotic therapy. Subsequent blood cultures yielded an FSSC isolate, establishing fungemia. **Conclusion:** These cases underscore the diverse clinical presentations of FSSC-associated disease, particularly in patients with varying degrees of immune impairment, and demonstrate that favorable outcomes may be achievable with timely recognition and appropriate antifungal therapy. Early clinical suspicion and prompt mycological confirmation are essential, especially when patients fail to respond to empirical antibacterial treatment.

INTRODUCTION

Fusarium species are ubiquitous filamentous fungi found in soil, plants, water, and other environmental niches. Fungal keratitis and infections of the skin and nails are common diseases caused by *Fusarium* species [1]. Infections caused by non-*Aspergillus* molds have been reported with increasing frequency in recent years [2], with *Fusarium* species emerging as prominent opportunistic pathogens. Members of the *F. solani* species complex (FSSC) are among the most important causes of human fusariosis and account for approximately 50% of invasive fusariosis cases [3]. The immunological status of the host and the route of infection are key determinants of the clinical manifestations of fusariosis. In immunocompromised patients, fusariosis typically presents as invasive or disseminated disease, whereas in immunocompetent hosts, it generally manifests as localized, superficial infections [4, 5]. However, a spectrum of intermediate immune impairment such as that conferred by poorly controlled diabetes mellitus, chronic corticosteroid use, or advanced hepatic dysfunction may predispose to both localized and invasive disease [3, 5]. We present three distinct cases that illustrate the broad clinical spectrum of FSSC-associated infection, ranging from localized keratitis to bloodstream infection, and underscore the critical management challenges associated with this pathogen.

CASE 1

A 67-year-old male farmer presented with a gritty sensation, redness, itching, and lacrimation in the left eye, along with blurred vision of five days' duration. The symptoms began a few hours after his left eye sustained trauma from a sugarcane leaf. He had a known history of

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type 2 diabetes mellitus (T2DM), for which he was on regular medication, with a recent HbA1c of 8.5%. On examination of the left eye, the cornea revealed a paracentral ulcer measuring 4–4.5 mm with feathery margins, a dry grey-white appearance, and a hypopyon (Figure 1). Visual acuity (VA) in the left eye was limited to hand motion (HM). At presentation, his fasting blood glucose was 140 mg/dL. Based on a clinical diagnosis of a bacterial corneal ulcer, the patient was started on oral ciprofloxacin 500 mg twice daily, topical moxifloxacin 0.5% eye drops, and atropine sulfate 1% ointment as a cycloplegic. After five days of treatment, no significant clinical improvement was observed. This prompted suspicion of a fungal etiology. A potassium hydroxide (KOH) mount of the corneal scraping revealed thin, septate hyphae. Based on the finding of fungal hyphae, empirical systemic antifungal therapy with oral fluconazole 200 mg once daily was initiated while awaiting culture results. Because fluconazole has poor activity against *Fusarium* species, it was subsequently discontinued after the fungal isolate was characterized. Additionally, the sample was processed for fungal culture on Sabouraud dextrose agar (SDA). Duplicate SDA tubes were incubated, one at 25°C and one at 37°C. Fungal growth on SDA appeared within four days of inoculation. The surface of the colony appeared white and cottony, while the reverse was cream-colored. A lactophenol cotton blue (LPCB) mount from the colony revealed septate hyphae, abundant large, sickle-shaped macroconidia with three to five septa, and a few oval microconidia. Based on the macroscopic and microscopic findings, the organism was identified as a member of the FSSC; species-level identification via molecular methods (such as β-tubulin, TEF1, or RPB2 gene sequencing) was unavailable at our institution [6]. Thus, the isolate could not be confirmed as *F. solani* sensu stricto. Subsequently, oral fluconazole was discontinued because of its lack of reliable activity against *Fusarium* species, and the patient was started on targeted therapy with topical 1% voriconazole eye drops, administered hourly for the first 48 hours and continued on a tapering regimen over a total course of 21 days. Topical natamycin 5% is generally considered the preferred first-line treatment for filamentous fungal keratitis; topical voriconazole may be used as an alternative or adjunct depending on clinical circumstances [7]. Upon completion of therapy without surgical intervention, marked clinical improvement was noted; the corneal ulcer had regressed to 2 mm in diameter, the hypopyon had resolved, a residual grade 1 corneal opacity was noted, and visual acuity had improved to 20/30.

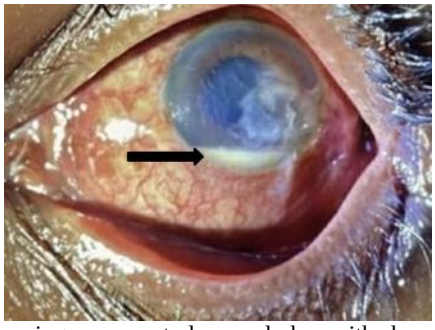

Fig. 1. Clinical photograph of the left eye showing a paracentral corneal ulcer with characteristic feathery margins and a dry greyish-white appearance, along with a hypopyon (black arrow), features consistent with fungal keratitis caused by an FSSC isolate.

CASE 2

A 55-year-old male farmer with a known history of chronic obstructive pulmonary disease (COPD) presented with a two-week history of dyspnea on exertion, fever, and a productive cough. The patient had no history of tobacco or alcohol use and was hemodynamically stable at presentation. He had recently been hospitalized for an acute COPD exacerbation and was completing a 14-day course of prednisolone 40 mg daily at the time of presentation. A chest CT scan revealed a small area of consolidation in the right lower lobe. The initial diagnosis was bacterial pneumonia, and sputum was sent for both bacterial and fungal culture. He was started empirically on IV piperacillin-tazobactam 3.375 g every 8 hours. After five days, the patient failed to show clinical improvement, raising suspicion of a fungal etiology. A KOH mount of the sputum showed fungal hyphae, while the bacterial culture remained negative. Fungal culture yielded white, cottony colonies with a cream-colored reverse on day 4 (Figure 2). The LPCB mount revealed septate hyphae bearing numerous characteristic sickle-shaped macroconidia with three to five septa, along with a few oval microconidia. Based on these findings, the isolate was identified as a member of the FSSC [6]. A repeat sputum culture demonstrated growth of the same organism. Because recovery of FSSC from sputum does not by itself establish tissue invasion, the isolate was interpreted in the context of the patient's clinical and radiological findings, while airway colonization remained a possible explanation. The patient was subsequently treated with intravenous voriconazole, initiated with a loading dose of 6 mg/kg every 12 hours for the first 24 hours and continued at 4 mg/kg every 12 hours for a total of five days, followed by a switch to oral voriconazole 200 mg every 12 hours

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for three months. This regimen led to marked improvement in his respiratory symptoms, with chest CT demonstrating resolution of the consolidation. The patient was discharged in stable condition and remained relapse-free during a six-month follow-up period. However, the distinction between true invasive pulmonary fusariosis and airway colonization in the setting of COPD and recent corticosteroid exposure remains challenging. Because the available clinical and microbiological data did not establish tissue invasion, the case should not be assigned a definitive EORTC/MSG 2020 (The European Organization for Research and Treatment of Cancer/Mycoses Study Group 2020) category without demonstrating all required host, clinical, and mycological criteria [8].

Fig. 2. Colony morphology of an FSSC isolate on Sabouraud dextrose agar (SDA) after four days of incubation, demonstrating characteristic white, cottony colonies with a cream-colored reverse.

CASE 3

A 57-year-old male with a history of alcoholic liver disease was admitted with jaundice and dyspnea. Initial investigations revealed a pH of 7.2, PCO_2 of 45.5 mmHg, PO_2 of 72 mmHg, and HCO_3^- of 14 mEq/L on arterial blood gas (ABG) analysis, along with a total bilirubin of 13.8 mg/dL, lactate of 4.2 mmol/L, hemoglobin of 5.4 g/dL, a white blood cell (WBC) count of 17.6 × 10^9/L, and a Model for End-Stage Liver Disease (MELD) score of 29, indicating advanced liver disease. The patient was classified as Child-Pugh Class C. On hospital day 1, he developed progressive respiratory failure (respiratory rate >30 breaths/min, SpO_2 <90%), requiring endotracheal intubation and mechanical ventilation. Despite broad-spectrum antibiotic therapy with intravenous meropenem 1 g every 8 hours, his condition worsened, raising suspicion of fungal sepsis. Blood cultures (two sets from peripheral sites) grew a member of the FSSC after five days. Culture on SDA yielded white, cottony colonies, and an LPCB mount revealed septate hyphae with characteristic sickle-shaped macroconidia (Figure 3). The source of fungemia could not be definitively established; intestinal translocation was considered a possible mechanism given the absence of a central venous catheter and the patient's advanced liver disease. The patient was managed with combination antifungal therapy consisting of intravenous liposomal amphotericin B at 5 mg/kg once daily and intravenous voriconazole, initiated with a loading dose of 6 mg/kg every 12 hours for 24 hours, followed by a reduced maintenance dose of 100 mg twice daily; this reduced dose was selected because severe hepatic impairment (Child-Pugh C) significantly impairs voriconazole metabolism, and population pharmacokinetic data suggest that substantial dose reduction may be necessary in this population, with therapeutic drug monitoring being particularly important [9, 10]. Isavuconazole, which has a more favorable pharmacokinetic profile in hepatic impairment, could have been considered as an alternative, but was unavailable at our institution. Combination therapy was selected because of the severity of the illness, although the clinical evidence supporting combination therapy for fusariosis remains limited. This regimen resulted in progressive stabilization, with improvement in SpO_2, respiratory rate, and arterial blood gas parameters. Intravenous antifungals were continued for seven days, after which the patient was transitioned to oral voriconazole 100 mg twice daily for three months. Follow-up blood cultures remained sterile, consistent with clearance of fungemia. The patient was discharged in stable condition and remained asymptomatic on subsequent outpatient follow-up.

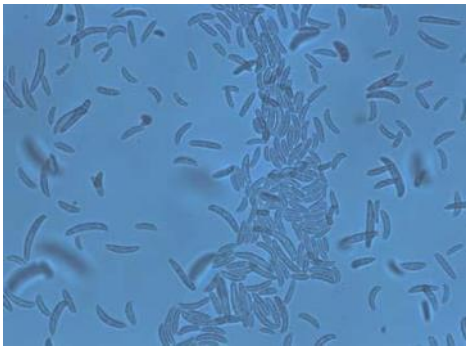

Fig. 3. Lactophenol cotton blue (LPCB) mount of *F. solani* demonstrating septate hyphae, characteristic sickle-shaped macroconidia with three to five septa, and oval microconidia.

DISCUSSION

Fusarium infections are predominantly observed in immunocompromised patients and are often associated with rapid clinical deterioration [4, 11]. In immunocompetent hosts, the infection typically manifests as localized keratitis or onychomycosis. Previous studies from India, including a study by Khurana *et al*. [12], have reported that *Fusarium* species account for approximately 24% to 47% of fungal keratitis cases in different Indian series. Less commonly, clinical presentations can include sinusitis, pneumonia, endophthalmitis, cellulitis, fungemia, or disseminated disease [3]. Disseminated fusariosis is an emerging and often fatal manifestation in severely immunocompromised hosts, in whom adventitious

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sporulation facilitates hematogenous spread [13].

Treatment of fusariosis is complicated by the organism's intrinsic antifungal resistance profile. *Fusarium* species exhibit intrinsic resistance to echinocandins, and many FSSC isolates demonstrate poor susceptibility to fluconazole and itraconazole [14]. Consequently, voriconazole and amphotericin B are among the systemic agents with activity against *Fusarium* species and are commonly used for treatment of invasive disease [15], with voriconazole therapeutic drug monitoring advised to optimize efficacy and safety [16]. Combination antifungal therapy has been used in severe or refractory cases, but evidence supporting its superiority over appropriate monotherapy remains limited [14, 15]. The reported mortality rate for invasive fusariosis remains high, particularly among patients with profound immunosuppression and disseminated disease [17]. In contrast, in immunocompetent hosts, the prognosis is generally more favorable because infections more often remain localized and superficial.

Clinical examination alone is not sufficient for diagnosing *Fusarium* infections, and laboratory confirmation through microbiological testing is critical for selecting the appropriate therapy. However, morphological identification alone cannot reliably distinguish among the multiple phylogenetic species within the FSSC, and molecular methods (β-tubulin, Translation elongation factor 1-alpha (TEF1), or RNA polymerase II (RPB2) gene sequencing) are required for definitive species-level identification [6].

Accordingly, the isolates in the present case series should be reported as FSSC rather than *F. solani* sensu stricto. The first case demonstrates that selection of an antifungal agent without activity against *Fusarium* may result in inadequate initial therapy, underscoring the importance of timely mycological diagnosis and appropriate empirical treatment of suspected filamentous fungal keratitis. For filamentous fungal keratitis, topical natamycin 5% is generally preferred as first-line therapy, with voriconazole used as an alternative or adjunct in selected cases [7]. The second case highlights the diagnostic challenge of interpreting positive sputum cultures in patients with structural lung disease, where *Fusarium* may represent airway colonization rather than true invasive infection. Clinical and radiological improvement after antifungal treatment supports, but does not prove, invasive disease in this case. Without histopathological confirmation or fulfillment of standardized diagnostic criteria, the diagnosis of invasive pulmonary fusariosis remains uncertain [8]. The third case reinforces the importance of prompt antifungal treatment in bloodstream infection caused by FSSC. Together, these cases highlight that timely diagnosis, accurate complex-level identification, and initiation of an appropriate antifungal regimen are pivotal in improving outcomes.

This case series has several key limitations. First, definitive species-level identification via molecular methods [18] and *in vitro* antifungal susceptibility testing [19] were not performed, owing to the unavailability of these resources at our institution. Consequently, identification relied solely on morphological characteristics, and the choice of antifungal therapy was empirical rather than guided by specific susceptibility data. Second, therapeutic drug monitoring was not performed for any patient receiving voriconazole, particularly the patient with severe hepatic impairment in Case 3, where drug accumulation and toxicity are significant concerns. Third, in the second case, distinguishing true invasive pulmonary fusariosis from airway colonization in the setting of COPD and recent corticosteroid use remains inherently challenging. Although the clinical and radiological improvement following antifungal therapy supports invasive disease, the possibility of colonization cannot be definitively excluded without histopathological confirmation. Fourth, the EORTC/MSG 2020 criteria for invasive fungal disease were not formally applied to classify these cases, limiting their comparability with other published series. Fifth, the small number of cases and retrospective case-series design preclude generalization of the clinical outcomes or treatment responses. Finally, no comparator group was available, and causality between respiratory FSSC isolation and pulmonary disease in Case 2 could not be established.

In conclusion, this case series underscores members of the FSSC as opportunistic pathogens with a broad clinical spectrum. Fusariosis should be considered in the differential diagnosis of progressive or atypical infections, particularly in immunocompromised individuals, agricultural workers with relevant environmental exposure, and patients who fail to respond to empiric antibacterial therapy. Prompt diagnosis through direct microscopy and culture is essential for recognizing FSSC-associated infection and enabling the timely initiation of appropriate antifungal therapy, such as voriconazole or amphotericin B, based on known intrinsic resistance patterns, thereby reducing the risk of morbidity and mortality associated with this infection. However, definitive species-level identification via molecular methods and antifungal susceptibility testing are strongly recommended to guide optimal therapy, and standardized diagnostic criteria (EORTC/MSG 2020) should be applied when appropriate to ensure valid case classification in future reports.

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CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest associated with this manuscript.

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DATA AVAILABILITY

The clinical data supporting the findings of this study are presented within the manuscript. Due to patient privacy and ethical restrictions, raw patient data are not publicly available. De-identified data may be made available from the corresponding author upon reasonable request, subject to institutional approval.

AI DISCLOSURE

The authors declare that no generative artificial intelligence (AI) tools were used in the preparation of this manuscript.

AUTHORS' CONTRIBUTIONS

AT: Conceptualization, Methodology, Investigation, Data curation, Writing – original draft. NM: Formal analysis, Supervision, Validation, Resources. AAS: Writing – review & editing, Visualization. All authors have reviewed and approved the final manuscript.

ETHICS STATEMENT

This study was conducted in accordance with the Declaration of Helsinki. The requirement for ethical approval was waived by the Institutional Ethics Committee of Chengalpattu Medical College, as this was a retrospective case series with minimal risk to participants. Written informed consent was obtained from all three patients or their legally authorized representatives for the publication of their clinical details and any accompanying images.

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