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Cutaneous Infection by *Fusarium oxysporum*

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Abstract

Fusarium is a genus of filamentous fungi that can cause disseminated infections. Fusariosis is the second most common invasive fungal infection in patients with hematological malignancies. Disseminated infections occur specifically in patients with profound neutropenia. *Fusarium* infections are suspected in those who have had a prolonged fever; other clinical forms include cutaneous, sinusitis, pneumonia, endophtalmitis, and fungemia. Cutaneous fusariosis in immunocompromised patients presents in a disseminated form. The most common lesion in patients with cutaneous involvement are erythematous and painful nodules or papules, with or without central necrosis. In the work, we report a case of cutaneous fusariosis in a pediatric patient, which is rare, with condition that leads to immunosuppression. The presentation was solely cutaneous, which is the least common presentation. It is necessary to determine if there is involvement of different organs. Prolonged treatment is required. More large-scale clinical studies are needed to characterize the optimal antifungal regimen and treatment duration.

Keywords: *Fusarium oxysporum*; Neutropenia; Immunosuppression; Cutaneous Fusariosis

Introduction

Fusarium is a genus of filamentous fungi that can cause superficial, locally invasive, or disseminated infections. Superficial infections typically occur in immunocompetent individuals [1-3], with two main types of localization: keratitis and onychomycosis [3,4]. Fusariosis is the second most frequent invasive fungal infection in patients with malignant hematologic conditions (only surpassed by aspergillosis), but it is the most common cause of cutaneous manifestations (70-75% of patients present cutaneous lesions). Invasive and disseminated infections occur almost exclusively in immunocompromised patients, specifically those with profound neutropenia and T-cell deficiencies. Patients with hematologic malignancies and recipients of hematopoietic stem cell transplants represent the most severe reported cases [5]. In immunosuppressed patients, Fusarium infections are suspected in those who have had prolonged fever unresponsive to antibiotics along with profound neutropenia. Other clinical presentations include cutaneous fusariosis, sinusitis, pneumonia, endophthalmitis, and fungemia [6-8]. Cutaneous fusariosis can present as localized or disseminated, with the latter being more common in immunocompromised patients (occurring in 88% of cases). In cases of invasive infection (fungemia), cutaneous involvement occurs in 70% of cases; however, isolated cutaneous involvement is the least frequent presentation [1-8]. The most reported lesions in patients with

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cutaneous involvement include multiple erythematous, painful nodules or papules, with or without central necrosis. Less commonly, other lesions such as onychomycosis, foot infections (indistinguishable from tinea pedis), and periungual cellulitis are reported[1]. The usual point of entry for Fusarium into the individual is via the respiratory tract, through inhalation of conidia from the fungus; however, the skin can also serve as an entry point if there are lesions or breaks in the skin's continuity [9]. In a review of cases in immunocompromised patients, cutaneous lesions accounted for 11% of the reasons for admission [9,10]. In this report, we describe a case of disseminated cutaneous infection caused by *Fusarium oxysporum* in a pediatric patient with a hematologic malignancy undergoing treatment.

Clinical Case

This is a 15-year-old male patient diagnosed with high-risk acute lymphoblastic leukemia (pre-B and pro-B cells) for his age, with a diagnosis made 4 months prior. His current condition began 5 days after receiving his first consolidation chemotherapy cycle, presenting with generalized dermatosis on the lower and upper limbs, including the soles of both feet. The lesions were characterized by erythematous-violaceous macules, painful to the touch, which later evolved into nodules of the same colour, some of which spontaneously drained non-foul-smelling whitish material. The patient also experienced fever up to 38°C, lasting for 10 days. The cutaneous lesions persisted until the day he was admitted to a tertiary care unit, 2 months after the onset of his illness. During this time, he received empirical treatment with meropenem, vancomycin, fluconazole, and conventional amphotericin B, showing partial improvement, after which the treatment was discontinued. New lesions appeared a few days later, prompting his referral to a tertiary care hospital.

Upon physical examination, generalized dermatosis on the lower (Figure 1) and upper limbs was confirmed, characterized by violaceous spots and subcutaneous nodules, painful to palpation, some with serous (non-foul-smelling) drainage. The rest of the physical exam was unremarkable. A biopsy was performed on a lesion on the left thigh, which revealed the presence of irregular, thick, hyaline septate hyphae and fusiform macroconidia in a Grocott-Gomori stain (Figure 1). A culture was performed on fluid from a lesion using Sabouraud medium, where a woolly colony with pink diffusible pigment was observed. Microscopic examination revealed hyaline septate hyphae organized into coremium structures, with conidiophores bearing fusiform macro- and microconidia (Figures 2 and 3). MALDI-TOF MS identified the pathogen as *Fusarium oxysporum*.

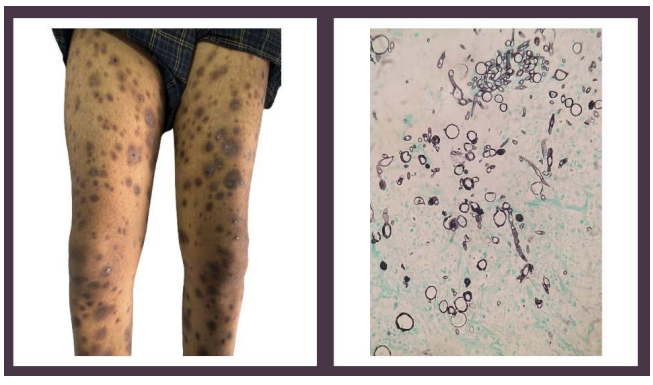

Figure 1: Disseminated fusariosis, with nodular, necrotic lesions, and biopsy showing thick, irregular, hyaline, septate hyphae and fusiform macroconidia (Grocott-Gomori 40X).

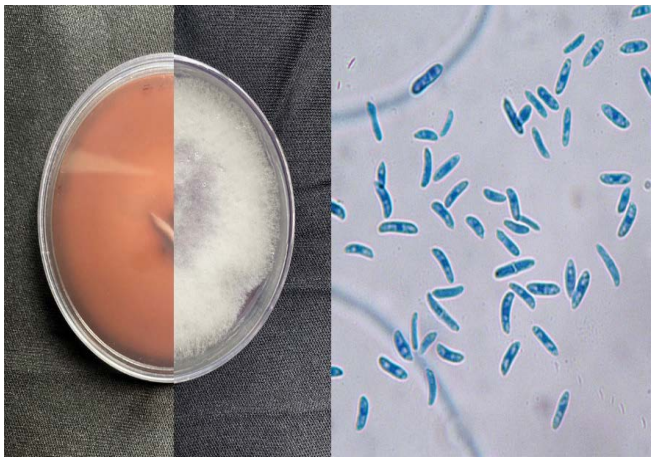

Figure 2: *Fusarium oxysporum* culture (front and reverse) in Sabouraud medium, with microscopic examination revealing fusiform microconidia (40X).

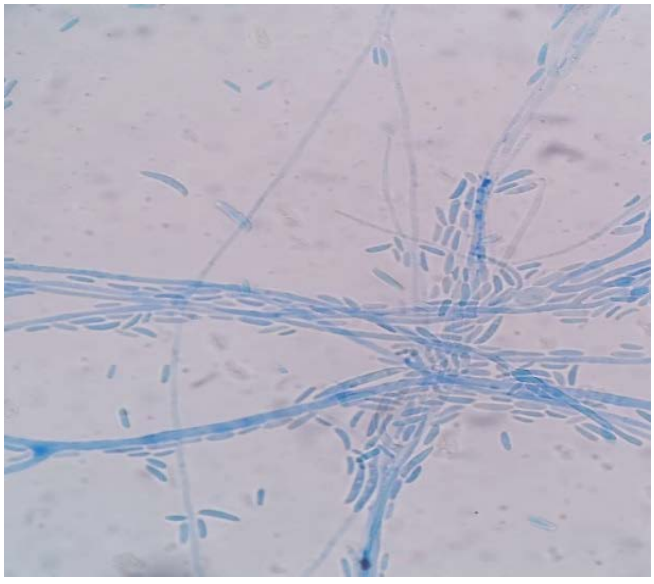

Figure 3: Microscopic examination of *Fusarium oxysporum* culture, showing hyaline, septate hyphae organized in coremium, and conidiophores with fusiform macro- and microconidia.

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An intensive search for involvement of other organs was conducted using imaging studies, blood and cerebrospinal fluid cultures, and an ophthalmologic evaluation. No other tissues were affected, and the diagnosis was concluded to be generalized cutaneous fusariosis. The patient was treated with liposomal amphotericin B at 5 mg/kg/day and voriconazole at 400 mg every 12 hours for 4 weeks. Treatment was continued with voriconazole for an extended period (more than 12 weeks) due to the slow clinical improvement. Currently, the patient still has cutaneous lesions, though fewer in number, and no new lesions have appeared (Figure 4).

Figure 4: Cutaneous lesions on the lower limbs after 12 weeks of treatment.

Discussion

Fusarium species are ubiquitous fungal organisms found in soil. They are considered phytopathogenic, parasitizing living plants, their debris, and aquatic systems worldwide [3]. The most significant species causing fusariosis in humans is the *Fusarium solani* complex (FSSC), followed by the *Fusarium oxysporum* complex (FOSC) and *Fusarium verticillioides* [7,11]. In our case, isolation was performed by culture, identifying F. *oxysporum (sensu stricto)*. Prolonged neutropenia and T-cell dysfunction are particularly critical risk factors among immunocompromised hosts, and *Fusarium* is recognized as the second leading cause of filamentous fungal infections in these patients, after *Aspergillus* spp [10,12]. The innate immune response plays a crucial role in defense against fungal infections. Macrophages and neutrophils damage *Fusarium* spp. hyphae, and this effect is enhanced by the production of interferon-gamma, granulocyte colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, and interleukin-15 (IL-15). The effect of IL-15 is mediated by the release of interleukin-8 and by direct stimulation of hyphal damage. Therefore, patients with reduced levels of these cells are at higher risk of infections by *Fusarium* spp [13]. The most common clinical manifestations of fusariosis include pulmonary infections and cutaneous infections, with the possibility of dissemination to other tissues. A review of 232 patients with fusariosis reported cutaneous involvement in 167 patients, with localized infection in 20 patients and disseminated infection in 147 patients. In more than 80% of cases, no history of skin trauma was reported. The cutaneous lesions were characterized by the presence of erythematous papules or nodules with or without central necrosis and rapid clinical progression. The overall mortality rate of patients with fusariosis was 66%, both in immunocompromised and immunocompetent patients, and 100% among those with persistent neutropenia [10]. Recognizing cutaneous involvement is a very important diagnostic key, as these lesions appear early in the disease and can precede dissemination [14,15]. Cutaneous lesions should be quickly evaluated by biopsy for histopathological analysis and cultures. *Fusarium* spp. can be isolated from blood cultures in 40–60% of cases [5,16]; in our patient, it was isolated from the fluid of one of the lesions, not from blood. Identification through culture is essential, as it is difficult to differentiate fusariosis from other agents of hyalohyphomycosis via histopathological study. The *Fusarium* genus is identified in cultures by the presence of multiple hyaline macroconidia shaped like crescents or bananas [16], which we identified in our patient's sample. The prognosis of cutaneous fusariosis remains poor, both due to the infection itself and the underlying predisposing condition [17]. Management of these patients includes the use of systemic antifungals, surgical debridement of infection sites, removal of colonized central catheters, and improving the patient's immunological status [5,16-18]. Treatment with antifungals is challenging, not only due to the lack of randomized clinical trials but also because of a high estimated rate of antifungal resistance [5,19]. Reports have used voriconazole, posaconazole, and amphotericin B either as monotherapy or in combination [7]. Some reports recommend combined therapy with voriconazole and liposomal amphotericin B, a regimen used in our patient, but there is still limited evidence, and more clinical studies are needed, especially in pediatric patients [10]. A systematic review by Cortés-López et al. [13] concluded that the combination of amphotericin B with a triazole demonstrated a survival rate of over 50% at 12 months. Reported mortality in immunocompromised patients with fusariosis is high, ranging from 50% to 66% in both immunocompromised and immunocompetent patients [10,16,17]. To reduce the mortality of this condition, it is crucial to prevent dissemination in infected patients, ensure early diagnosis, and administer prolonged antifungal therapy [20].

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In this report, we present a case of cutaneous fusariosis in a pediatric patient, which is uncommon according to the literature, but occurred in the context of an underlying condition leading to immunosuppression. *Fusarium* species should be especially considered in the differential diagnosis of local and invasive fungal infections when cutaneous manifestations are present. In our patient's case, the presentation was exclusively disseminated cutaneous fusariosis, which is reported as the least common form and can lead to a delayed diagnosis. The clinical assessment of the patient should determine whether other organs are affected, which is essential for determining severity and initiating antifungal therapy. *Fusarium* infections often require prolonged treatment durations (i.e., months), as was the case with our patient, until neutrophil counts are consistently maintained, as this is the cornerstone of the immune response and ultimately improves patient prognosis. Further large-scale clinical studies are needed to define the optimal antifungal regimen and the duration of treatment, especially for pediatric patients.

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