CASE REPORT / OLGU SUNUMU

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Invasive Fusariosis with Disseminated Skin Lesions in an Allogeneic Hematopoietic Stem Cell Transplant Recipient: A Case Report and Literature Review

Allojenik Hematopoietik Kök Hücre Nakli Alıcısında Dissemine Deri Lezyonlarıyla Seyreden İnvaziv Fusariozis: Olgu Sunumu ve Literatür Derlemesi

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Abstract

Infections are a major cause of mortality in immunocompromised patients. Invasive fusariosis is a rare opportunistic fungal infection associated with a poor prognosis. We present a rare and challenging case of a 46-year-old man who underwent allogeneic hematopoietic stem cell transplantation for acute myeloid leukemia (M4) and subsequently developed disseminated skin lesions. This case highlights the importance of maintaining a high index of clinical suspicion for invasive fusariosis in immunocompromised patients, particularly because empirical treatment can be challenging owing to the high minimum inhibitory concentration values for amphotericin B. Early microbiological diagnosis and prompt initiation of appropriate antifungal therapy are essential for improving treatment outcomes. Despite receiving antifungal therapy, the patient died of thrombotic complications. Further research is needed to develop effective strategies for the prevention and treatment of invasive fusariosis.

Keywords: Allogeneic hematopoietic stem cell transplantation, antifungal susceptibility, fungal infection, opportunistic infections

Öz

Enfeksiyonlar, immünkompromize hastalarda önemli bir mortalite nedeni olmaya devam etmektedir. İnvaziv fusariozis, nadir görülen, kötü prognostik fırsatçı bir mantar enfeksiyonudur. Akut miyeloid lösemi (M4) nedeniyle allojenik hematopoietik kök hücre nakli uygulanan ve daha sonra dissemine deri lezyonları gelişen 46 yaşındaki erkek hastaya ait nadir ve zorlu bir olguyu sunuyoruz. Bu olgu, immünkompromize hastalarda, özellikle amfoterisin B'ye karşı yüksek minimum inhibitör konsantrasyon değerleri göstermesi nedeniyle ampirik tedavinin zor olduğu durumlarda, invaziv fusariozis için klinik şüphe duyulmasının önemini vurgulamaktadır. Erken mikrobiyolojik tanı ve antifungal tedavi çok önemlidir. İnvaziv fusariozis için önleme ve tedavi stratejileri konusunda daha fazla araştırma yapılması gerekmektedir.

Anahtar Kelimeler: Allojenik hematopoietik kök hücre nakli, antifungal duyarlılık, fungal enfeksiyon, fırsatçı enfeksiyonlar

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Introduction

Fusarium spp. are recognized as soil saprophytes and important plant pathogens. Certain species can also cause infections in humans. The most common infections caused by *Fusarium* spp. are onychomycosis and keratitis. However, in immunocompromised patients, *Fusarium* spp. can also cause disseminated, life-threatening systemic infections[1].

With the increasing number of solid organ and hematopoietic stem cell transplant (HSCT) recipients, as well as the widespread use of immunomodulatory therapies such as tumor necrosis factor antagonists, the incidence of invasive fungal infections has been increasing worldwide[2]. Although *Fusarium* spp. are relatively rare fungal pathogens, they have been increasingly reported in patients with hematologic malignancies and HSCT recipients, particularly those with profound and prolonged neutropenia[3]. Skin lesions associated with invasive fusariosis typically occur as part of disseminated disease and commonly present as papules or nodules, with or without necrosis. Disseminated fusariosis may also be accompanied by fungemia and characteristic skin lesions, and pulmonary or sinus involvement has been reported. Despite appropriate treatment, the mortality rate remains greater than 50%[4].

In this report, we present a case of invasive fusariosis in a severely immunosuppressed patient who developed the infection after undergoing HSCT for acute myeloid leukemia (AML) and subsequently received chemotherapy for disease relapse.

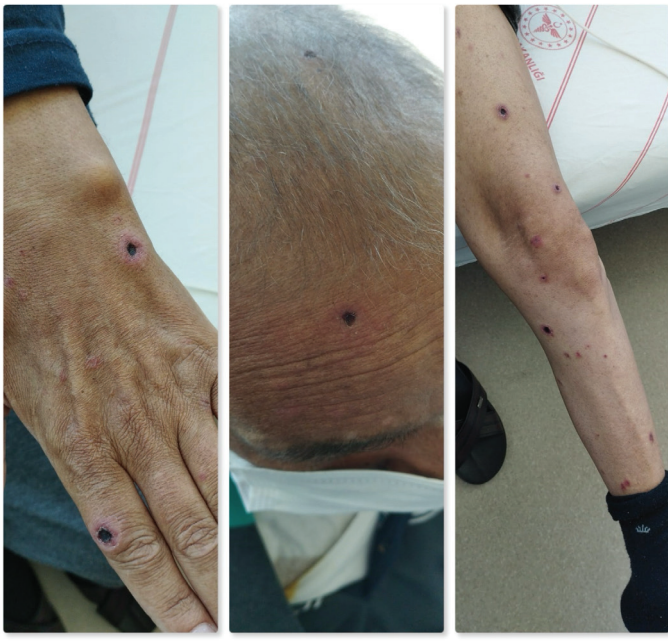

Figure 1. Erythematous, slightly raised nodular lesions with central necrosis on the extremities and head.

Case Report

A 46-year-old man who had undergone allogeneic HSCT 1 year earlier for AML was hospitalized after a bone marrow smear performed because of abnormal blood counts revealed blastic infiltration. Following the diagnosis of post-HSCT relapse of AML, intensive induction chemotherapy was reinitiated. After receiving the FLAG-IDA (fludarabine, cytarabine, idarubicin, and granulocyte colony-stimulating factor) regimen, the patient achieved complete remission, and a second transplant was planned. Following consolidation chemotherapy, a second allogeneic HSCT was performed using a 9/10 HLA-matched unrelated donor. On post-transplant day 18, while profoundly neutropenic and receiving posaconazole prophylaxis, the patient developed fever and painless, non-pruritic, erythematous, slightly raised nodular lesions measuring less than 1 cm on the extremities and head (Figure 1).

On physical examination, the patient's Glasgow Coma Scale score was 15. Vital signs were as follows: temperature, 38.8 °C; pulse rate, 110 beats/min; respiratory rate, 22 breaths/min; and blood pressure, 110/60 mmHg. Laboratory findings showed normal liver and kidney function, a C-reactive protein level of 95 mg/L (reference range, 0–5 mg/L), a leukocyte count of $0.04 \times 10^3/\mu L$, a neutrophil count of $0 \times 10^3/\mu L$, a hemoglobin level of 8.2 g/dL, and a platelet count of $13 \times 10^3/\mu L$. Blood cultures were obtained, and chest computed tomography and a skin biopsy were performed. Antifungal therapy was changed to liposomal amphotericin B (10 mg/kg/day). Chest computed tomography revealed a nodular lesion in the left lung (Figure 2).

Pathological examination of the skin biopsy revealed concurrent leukemic infiltration and fungal hyphae (Figure 3), and *Fusarium* spp. was isolated from blood cultures (Figure 4).

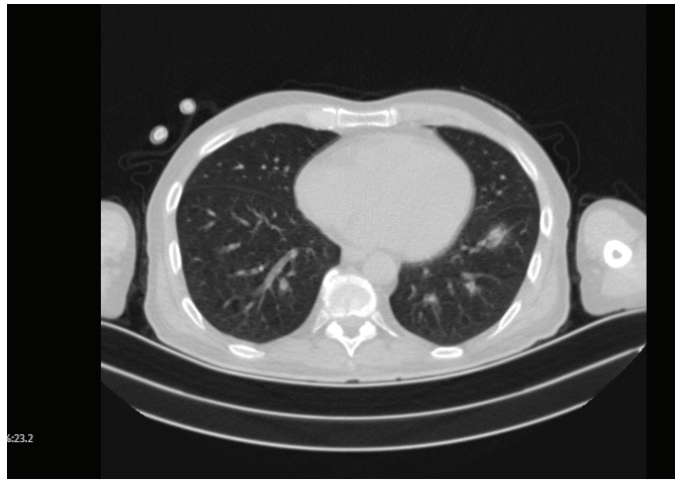

Figure 2. Chest computed tomography showing a nodular infiltrate in the left lung.

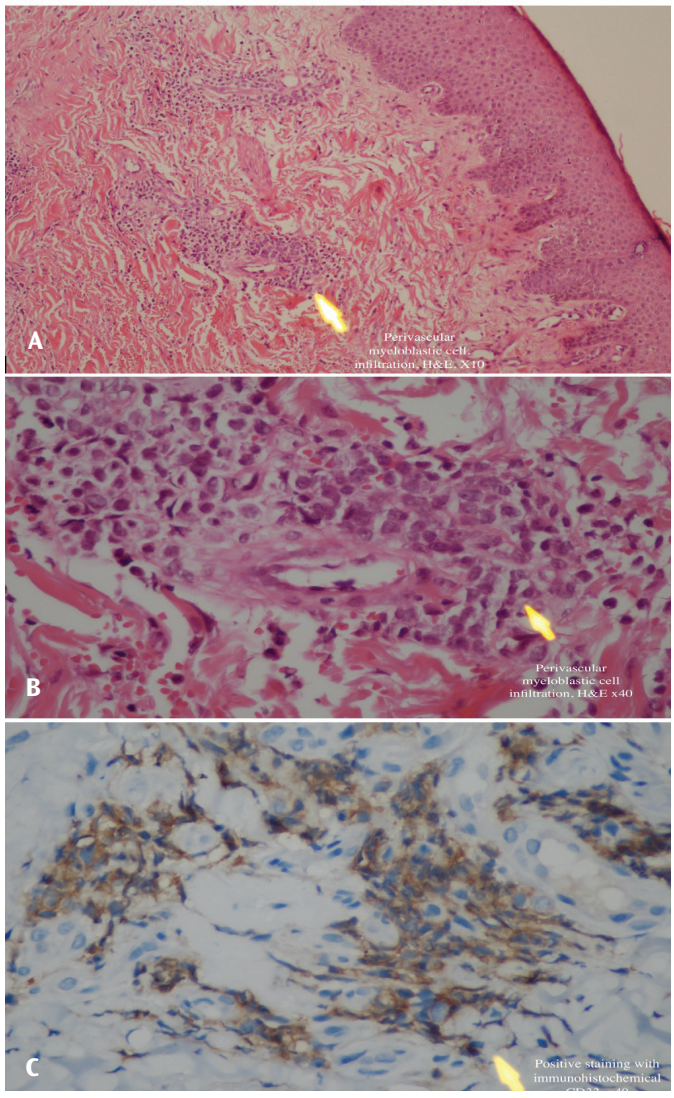

Figure 3. Pathological examination of the skin biopsy. (A) Perivascular myeloblastic cell infiltration (H&E, ×10). (B) Higher-magnification view of myeloblastic infiltration (H&E, ×40). (C) Positive CD33 immunohistochemical staining (×40). H&E, hematoxylin and eosin.

Antifungal susceptibility testing (AFST), performed according to the Clinical and Laboratory Standards Institute M38 guideline, demonstrated a minimum inhibitory concentration (MIC) of >32 µg/mL for amphotericin B, 4 µg/mL for voriconazole, and 32 µg/mL for echinocandins. Voriconazole was subsequently added to the treatment regimen. Although the patient's fever subsided, the central venous catheter was removed because blood cultures remained positive after 72 hours of treatment. Following catheter removal, the patient became afebrile, and subsequent blood cultures showed no microbial growth. Despite antifungal therapy, the patient died on day 18 of treatment due to thrombotic complications. Written informed consent for publication of the clinical information and accompanying images was obtained from the patient.

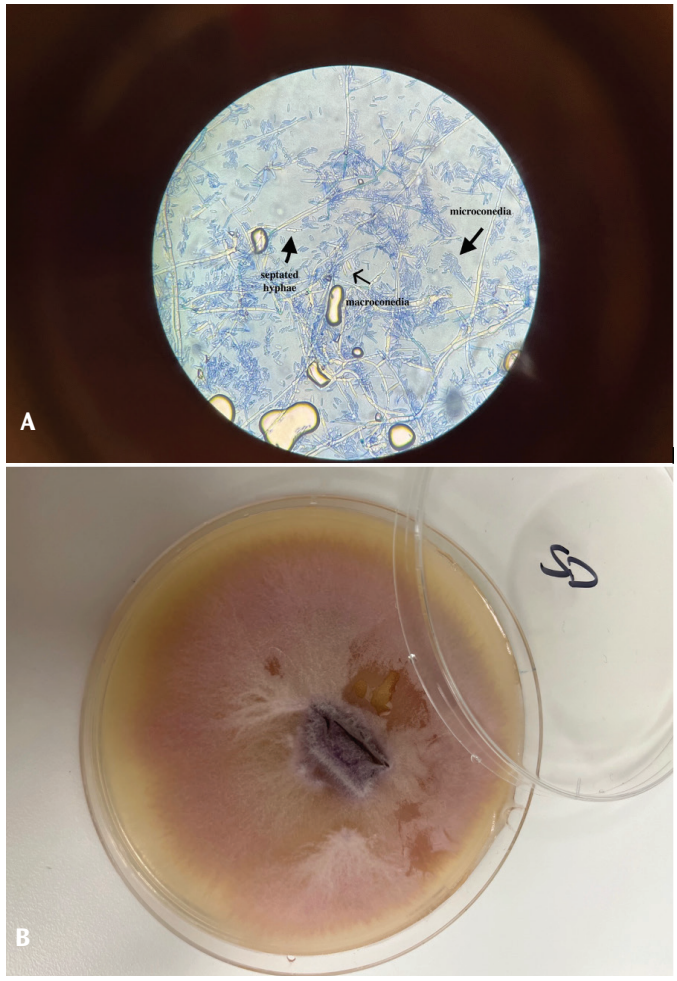

Figure 4. Microbiological identification of *Fusarium* spp. (A) Macroconidia, microconidia, and septate hyphae observed in a lactophenol cotton blue preparation. (B) Pinkish-purple, cottony colony growth on Sabouraud dextrose agar.

Discussion

We report a case of invasive fusariosis in a severely immunocompromised patient who underwent two allogeneic HSCT procedures. Advances in the treatment of hematologic malignancies have improved patient outcomes but have also increased the risk of opportunistic infections[5]. Invasive infections caused by rare fungal pathogens are being reported with increasing frequency in patients with profound immunosuppression. Among these, invasive fusariosis has become increasingly recognized in patients with hematologic malignancies. Individuals with impaired immune function, particularly those with prolonged and profound neutropenia or severe T-cell immunodeficiency, as well as those receiving immunosuppressive therapies such as systemic corticosteroids or chemotherapy, are at particularly high risk[6]. Cases of invasive fusariosis in patients with hematologic malignancies reported during the past 3 years are summarized in Table 1[3,7-17].

Table 1. Reported cases of *Fusarium* infection in immunosuppressed patients.

References	Year	Symptom	Organ involvement	Underlying disease	Treatment	Etiological agent	MIC amphotericin B	Voriconazole
Samborska et al.[3]	2023	Nodular lesion on skin		Relapse ALL	Liposomal amphotericin B + voriconazole	*Fusarium oxysporum*	-	-
Furaijat et al.[7]	2023	Defect of vision	Keratitis + aortic valve involvement	T-cell leukemia and allogeneic bone marrow transplantation	Liposomal amphotericin B + natamycin	*Fusarium petroliphilum*	1 µg/mL	
Kao et al.[8]	2023	Visual impairment + hypoxia	Keratitis + pulmonary nodule	Myelodysplasia + allogeneic bone marrow transplantation	Liposomal amphotericin B + voriconazole	*Fusarium* spp.	>8 µg/mL	>16 µg/mL
Marino et al.[9]	2023	Ulcerated necrotic lesion on the nose		AML + COVID-19	Liposomal amphotericin B + posaconazole	*Fusarium solani* + *Mucorales*		>8 µg/mL
Hannemann et al.[10]	2022	Nodular lesion on skin + fever +	Hepatic renal splenic nodules + left ventricular thrombus	Aplastic anemia Allogeneic stem cell transplantation	Liposomal amphotericin B + voriconazole	*Fusarium solani*	R.	S
Zhong et al.[11]	2022	Necrotic nodular lesion on the skin		ALL	Voriconazole + terbinafine	*Fusarium fujikuroi*	>32 µg/mL	0.25 µg/mL
Fei et al.[12]	2022	Nodular lesion on the skin	Liver-spleen hypodense lesions	ALL + CAR-T + allogeneic bone marrow transplantation	Liposomal amphotericin B + posaconazole-voriconazole	*Fusarium* spp.	-	-
De la Espriella et al.[13]	2022	Necrotic nodular lesion on the skin		Relapse ALL	Liposomal amphotericin B + voriconazole	*Fusarium solani*	8 µg/mL	3 µg/mL
De la Espriella et al. [13]	2022	Generalized erythematous maculopapular skin lesions with a violaceous center + fever	Disseminated osteomyelitis, myositis, and septic arthritis	AML	Liposomal amphotericin B + voriconazole + caspofungin	*Fusarium solani*	3 µg/mL	>32 µg/mL
Brent et al.[14]	2022	Necrotic nodular lesion on the skin + hypoxia	Pulmonary nodule	B-ALL	Liposomal amphotericin B + voriconazole-flucytosine	*Bisifusarium delphinoides*	1 µg/mL	4 µg/mL
Asiimwe et al.[15]	2022	Nodular lesion on the skin + headache sinus pain	Pulmonary nodule Sphenoidal occlusion	Relapsed AML	Liposomal amphotericin B + voriconazole-terbinafine	-	1 µg/mL	8 µg/mL
Al-Farsi et al.[16]	2022	Nodular lesion on the skin		ALL	Liposomal amphotericin B + voriconazole	*Fusarium solani*	1 µg/mL	16 µg/mL
Risum et al.[17]	2022	Headache pain in sinuses	Maxillary cranial involvement	AML	Liposomal amphotericin B + voriconazole-terbinafine	*Fusarium dimerum*	0.5 µg/mL	8 µg/mL

ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; B-ALL, B-cell acute lymphoblastic leukemia; CAR-T, chimeric antigen receptor T-cell therapy; COVID-19, coronavirus disease 2019; MIC, minimum inhibitory concentration.

Our case highlights that prolonged and profound neutropenia during both the AML treatment and HSCT periods was the predominant risk factor for the development of invasive fusariosis.

Fusarium spp. typically cause superficial or localized infections in immunocompetent individuals, whereas in immunocompromised patients, they can cause life-threatening invasive systemic infections. Skin lesions are reported in approximately 70%–75% of cases of invasive fusariosis. In our patient, extensive skin involvement was accompanied by pulmonary involvement and severe immunosuppression[18]. The thrombotic complications observed in our patient may have been associated with the angioinvasive nature of *Fusarium* spp. Angioinvasion can lead to endothelial damage, thrombosis, and possible fungal embolization, all of which may contribute to poor clinical outcomes in patients with invasive fungal disease.

Although the mortality rate exceeds 50%, no gold standard treatment for invasive fusariosis has been established. The limited number of clinical studies on *Fusarium* spp., together with insufficient data on molecular resistance mechanisms, has hindered the establishment of species-specific MIC breakpoints[19]. Species- or species complex-level identification could not be performed because molecular diagnostic methods were unavailable at our center. This represents an important limitation, as identification of the infecting species or species complex (e.g., *Fusarium solani* species complex or *Fusarium oxysporum* species complex) may significantly influence antifungal susceptibility patterns and treatment decisions. Among the available antifungal agents, amphotericin B generally demonstrates the lowest MIC values against *Fusarium* spp., although susceptibility varies considerably among isolates. Voriconazole, posaconazole, and isavuconazole also exhibit antifungal activity, whereas echinocandins are generally considered intrinsically ineffective against *Fusarium* spp.[20]. In six of the cases with available numerical amphotericin B MIC data, the amphotericin B MIC was <4 µg/mL. In contrast, the MIC observed in our case was markedly higher than those reported in the literature (Table 1). This finding highlights the substantial variability in antifungal susceptibility among *Fusarium* isolates and emphasizes the importance of performing AFST to guide individualized therapy. In our case, invasive fusariosis developed despite posaconazole prophylaxis, and the isolate demonstrated voriconazole and amphotericin B MIC values of 4 µg/mL and >32 µg/mL, respectively. The markedly elevated amphotericin B MIC suggests intrinsic or potentially acquired resistance. *Fusarium* species are known to exhibit variable, and often elevated, MIC values for commonly used antifungal agents, which can substantially limit treatment options. Reduced susceptibility to amphotericin B has been associated with differences in cell membrane composition, biofilm formation, and species-specific resistance mechanisms. The elevated MIC observed in our isolate may explain the poor clinical response despite early initiation of liposomal amphotericin B. These findings underscore the importance of AFST in guiding individualized treatment strategies, particularly in immunocompromised patients with disseminated infection[19,20]. However, not all institutions have access to specialized laboratory testing such as AFST or molecular species identification. In resource-limited settings or remote regions, collaboration with reference laboratories should be considered whenever possible. As illustrated by the cases summarized in Table 1, AFST can provide valuable information for optimizing the management of invasive *Fusarium* infections.

Conclusion

Fusarium spp. can cause invasive fungal infections with high mortality rates in immunocompromised patients and remain challenging to diagnose and treat. Early diagnosis and individualized patient evaluation are essential because well-established, evidence-based treatment recommendations are lacking.

Ethics

Informed Consent: Written informed consent for publication of the clinical information and accompanying images was obtained from the patient.

Footnotes

Authorship Contributions

Surgical and Medical Practices: A.K., H.S., A.E.T., Concept: A.K., E.E.E., S.K., Design: A.K., E.E.E., H.S., A.E.T., S.K., Data Collection or Processing: A.K., E.E.E., H.S., A.E.T., S.K., Analysis or Interpretation: A.K., Literature Search: A.K., E.E.E., Writing: A.K., E.E.E.

Conflict of Interest: The authors declare no conflict of interest.

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