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When Trauma Meets *Fusarium*: A Rare but Life-Threatening Invasive Fungal Wound Infection

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Abstract

Post-traumatic invasive fungal infections are rare but associated with high mortality. We report the case of a 58-year-old man with type 2 diabetes mellitus who was admitted to the intensive care unit following a road traffic accident resulting in an unstable pelvic fracture and a closed left ankle fracture complicated by hemorrhagic shock. After surgical fixation, the clinical course was marked by polymicrobial necrotizing skin and soft tissue infection of the left lower limb, requiring broad-spectrum antibiotic therapy, repeated surgical debridements, and hyperbaric oxygen therapy. Owing to progressive tissue destruction and the development of septic shock, a mid-thigh amputation was performed. Mycological examination of intraoperative tissue biopsies revealed *Fusarium spp*., leading to the initiation of voriconazole therapy. Despite optimal medical and surgical treatment, the patient died secondary to multiorgan failure. This case highlights the severity of post-traumatic invasive fusariosis in patients with predisposing factors such as diabetes mellitus and underscores the importance of early clinical suspicion, prompt mycological diagnosis, and multidisciplinary management to improve outcomes.

Keywords: Invasive fusariosis; *Fusarium*; Wound infection; Intensive care unit; Diabetes mellitus

Introduction

Invasive fungal infections (IFI) represent a growing challenge in healthcare settings, with their incidence closely linked to the increasing number of immunocompromised patients [1]. Post-traumatic IFI represent a rare but increasingly recognized clinical entity, occurring in approximately 6.8% of severely injured combat casualties, and are predominantly caused by filamentous fungi, with *Fusarium* spp. emerging as a significant pathogen [2]. Infection may occur through inhalation of airborne conidia, direct inoculation through skin disruption, or contact with contaminated soil or plant material [3]. Regardless of the initial site of infection, hematogenous spread can lead to disseminated disease with multiorgan involvement, which is associated with poor clinical outcomes [3]. Current knowledge of invasive fusariosis is largely based on small retrospective studies and isolated case reports [4]. In Tunisia, epidemiological data on invasive fusariosis remain limited. Reporting additional cases is therefore essential to improve understanding of the local mycological epidemiology, predisposing factors, clinical presentation, and therapeutic approaches. We herein describe a case of post-traumatic invasive fusariosis occurring in a diabetic polytrauma patient admitted to the Intensive Care Unit of the Military Hospital of Tunis.

Case Presentation

A 58-year-old man with a history of type 2 diabetes mellitus was admitted to the Intensive Care Unit following a road traffic accident (day 0) in which he was struck by a bus while walking. Initial assessment revealed an unstable pelvic fracture and a closed fracture of the left ankle, complicated by hemorrhagic shock. Delayed osteosynthesis was performed on day 7. The postoperative course was complicated by the development of a polymicrobial necrotizing skin and soft tissue infection of the left lower limb. Microbiological cultures yielded *Enterobacter cloacae*, *Morganella morganii*, and *Acinetobacter baumannii*. The patient received broad-spectrum antibiotic therapy in combination with repeated surgical debridement and hyperbaric oxygen therapy. Despite these measures, progressive extension of the necrotic lesions and the onset of septic shock on day 22 necessitated a left mid-thigh amputation. Tissue biopsies of the skin lesions were collected for microbiological investigation. Culture on Sabouraud agar grew *Fusarium* spp. on day 25 (Figure 1), prompting initiation of voriconazole therapy. Despite antifungal treatment, repeated surgical excisions, and continued hyperbaric oxygen therapy, the patient's condition progressively deteriorated, and he died on day 60 from multiorgan failure.

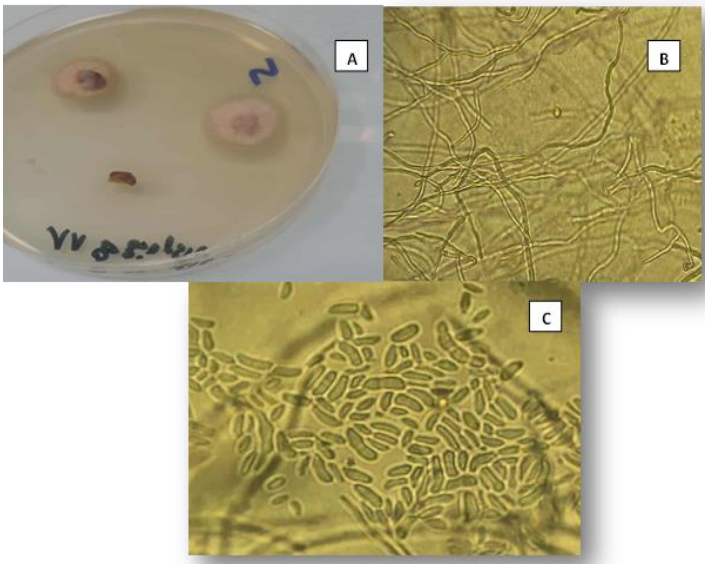

Figure 1: *Fusarium spp. isolated from a tissue biopsy collected from the amputation site. Macroscopic appearance of the culture on Sabouraud agar (A); microscopic examination showing thin, hyaline septate hyphae (B); and characteristic microconidia of the genus Fusarium (C).*

Discussion

Fusarium spp. are ubiquitous saprophytic filamentous fungi widely distributed in soil, water, and air, including seawater and community and hospital water distribution systems [4]. However, they are only rarely isolated from ambient air, where they account for approximately 1 to 2% of fungal species. Their geographic distribution is remarkably broad, ranging from temperate and tropical regions to extreme environments such as deserts and polar areas. Notably, *Fusarium solani* has even been isolated from the highly radioactive environment of the damaged Chernobyl nuclear reactor [5]. More than 300 phylogenetically distinct species have been described, although only a limited number are implicated in human disease, mainly the *F. solani, F. oxysporum*, *and F. fujikuroi* species complexes [6]. In military settings, *Fusarium* spp. account for approximately 21% of post-traumatic IFI, following Mucorales and *Aspergillus* spp. (34% and 31%, respectively) [7]. The U.S. military reported 143 cases of post-traumatic IFI attributable to Mucorales, *Aspergillus*, and *Fusarium* in 55%, 45%, and 24% of cases, respectively [8]. In contrast, epidemiological data from civilian populations remain limited. A systematic review of 75 post-traumatic IFI found that *Fusarium* spp. accounted for only 11% of reported cases, whereas Mucorales were responsible for 75% [9]. The incidence of invasive fusariosis is increasing, with mortality rates ranging from 40% to 80% and approaching 100% in patients with persistent neutropenia [10]. Hematological malignancies, particularly acute leukemias, and hematopoietic stem cell transplantation remain the classical risk factors for disseminated fusariosis [4]. Although any organ may be affected, cutaneous involvement predominates (70–90%), followed by pulmonary disease (25–40%) [10]. However, invasive fusariosis has also been reported in the absence of immunosuppression. A recent French multicenter retrospective study found that 20% of ICU patients with invasive fusariosis were immunocompetent at admission [11]. Such infections have classically been described following severe trauma, burns, or in patients with diabetes mellitus [5,12]. These observations suggest that disruption of cutaneous barriers and metabolic disturbances alone may create a favorable environment for invasive infection [13].

Various types of skin lesions have been described, including subcutaneous nodules, ecthyma-like lesions, and, less frequently, bullous lesions [2]. A target-like lesion characterized by central necrosis surrounded by a peripheral inflammatory halo is considered highly suggestive of fusariosis. These lesions are often multiple and predominantly involve the extremities [5]. The diagnosis of invasive fusariosis relies on a multimodal approach combining clinical, microbiological, and histopathological findings. In a series of 84 cases of invasive fusariosis, culture alone established the diagnosis in 77% of patients, whereas the combination of culture and histopathology allowed diagnosis in 100% of cases [14]. Direct mycological examination remains essential for the diagnosis of *Fusarium* infections. However, although it confirms the presence of fungal elements, its sensitivity and specificity are limited. In particular, it cannot reliably distinguish the fungal genus involved. Therefore, it must

always be complemented by culture. Histopathological examination typically reveals hyaline septate hyphae invading necrotic tissue, best visualized using Gomori–Grocott silver or periodic acid–Schiff (PAS) staining. While these findings support the diagnosis of hyalohyphomycosis, they do not allow differentiation between etiological agents. Confusion with *Aspergillus* spp. is particularly common. Nevertheless, a useful morphological clue is that *Fusarium* hyphae tend to branch at right angles, whereas *Aspergillus* hyphae usually branch at acute angles [6]. Subculture on potato dextrose agar (PDA) is recommended to facilitate species identification. Colonies often exhibit bright pigmentation ranging from violet to orange, sometimes associated with a diffusible pigment that changes from brick red to dark brown [5]. Species identification relies on the morphological characteristics of conidia produced by phialides of variable size and arrangement. Macroconidia, characteristic of the genus, are fusiform, multicellular, and septate, whereas microconidia are more abundant, typically unicellular, and oval to ellipsoidal in shape. Mesoconidia and chlamydospores may also serve as useful morphological features for species differentiation [5].

Despite their diagnostic value, direct microscopy and culture have limited performance. Studies have shown that 35–40% of isolates cannot be accurately identified based solely on cultural and microscopic characteristics [5]. In our patient, the diagnosis of invasive fusariosis was established only on day 25, in a clinical context initially dominated by polymicrobial bacterial infection. This diagnostic delay, frequently reported in trauma and surgical settings, highlights the importance of maintaining a high index of suspicion for fungal infection in progressive necrotic wounds that fail to respond to appropriate antibacterial therapy. Among non-invasive fungal biomarkers, serum 1-3 β-D-glucan (BDG) has demonstrated promising diagnostic performance, with a sensitivity of 76.7% in a series of 73 cases of invasive fusariosis. Notably, BDG positivity preceded diagnosis by conventional methods, such as culture or histopathology, in 73% of patients [15]. In contrast, serum galactomannan has limited sensitivity, reaching only 24% in a series of 101 cases [16]. In addition, galactomannan may exhibit cross-reactivity with *Aspergillus* spp., likely due to similarities in fungal cell wall components [17]. Molecular techniques have emerged as valuable tools for species identification. Although ribosomal DNA sequencing, particularly of the ITS2 region, remains the standard molecular marker for most fungi, cross-reactivity with *Aspergillus* and *Rhizopus* species has been reported [17]. More recently, sequencing of the translation elongation factor 1-alpha (TEF1α) gene has demonstrated superior discriminatory power for *Fusarium* species identification [5]. Matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALDI-TOF MS) also provides rapid species-level identification. In a study including 289 isolates, correct identification of *Fusarium* species complexes was achieved in 82.8% of cases [18]. Voriconazole is currently recommended as the first-line treatment for invasive fusariosis [4]. Liposomal amphotericin B represents an alternative in cases of intolerance or azole resistance [4]. Early surgical management, including repeated debridement of necrotic tissue and, when necessary, amputation, is an essential component of treatment in invasive cutaneous forms. In our case, despite amputation of the affected limb, repeated surgical excisions, hyperbaric oxygen therapy, and voriconazole treatment, the outcome was unfavorable. The patient ultimately died from multiorgan failure, consistent with the high mortality rates reported in the literature [9,11].

Conclusion

This case highlights the severity of post-traumatic invasive fusariosis and the diagnostic challenges associated with this rare infection. Post-traumatic IFI should be considered in patients with progressive necrotic wounds that fail to respond to appropriate antibacterial therapy, particularly in the presence of predisposing factors such as diabetes mellitus. Early recognition, prompt mycological investigation, and multidisciplinary management are crucial to improving patient outcomes.

Declaration of interests

The authors declare no conflicts of interest related to this article.

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