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FUNGAL INFECTIONS IN TRANSPLANTATION (S SHOHAM, SECTION EDITOR)

Dark Mold Infections in Solid Organ Transplant Recipients

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Abstract

Purpose of Review Solid organ transplant (SOT) recipients are a high-risk population for invasive fungal infections. While infections with black molds are rare in SOT recipients, they are important to consider since morbidity and mortality are high, and the treatment may differ substantially from other more common invasive fungal infections.

Recent Findings The incidence of black molds tends to be increasing. While the backbone of diagnosis remain traditional tools like histopathology and culture, the rapid evolution of non-culture-based methods for molecular detection promises improved identification of these rare fungi. While for many of those rare fungal infections liposomal amphotericin B remains the treatment of choice, mold active triazoles are the backbone for treatment of scedosporiosis and lomentosporiosis. New antifungal agents like ibrexafungerp, olorofim, and fosmanogepix may provide additional treatment options for the future.

Summary In this paper, we review infections caused by black molds in SOT recipients. The focus lies on epidemiology, diagnostic work-up, and antifungal treatment.

Keywords Transplantation · Rare molds · Mucormycosis · Phaeohyphomycosis · Black molds · Invasive fungal disease

Introduction

In solid organ transplant (SOT) recipients, infection control remains a cornerstone to optimize outcomes. Beside bacterial and viral infection, invasive fungal infections (IFIs) play an important role in this population as well. While infections by *Candida* spp. and *Aspergillus* spp. are responsible for about 70–80% of IFIs in SOT recipients [1, 2, 3, 4], there are other emerging fungal pathogens that may cause infections in these vulnerable populations, including black molds.

This review will focus on the role of black molds as rare fungal pathogens causing infections in SOT recipients, while the review will exclude black *Aspergillus* spp., which are discussed elsewhere [5, 6]. Black molds are characterized by their dark appearance caused by melanin in the cell walls. Melanin classically prevents solar damage through UV radiation in the fungus but is believed to play an important role in enhancing ability for human infection. Since melanized fungi are overrepresented in human infections, they can cause infections in immunocompromised as well as in immunocompetent individuals [7]. For example, it was demonstrated that melanin in cell walls of *Aspergillus* spp. and *Rhizopus* spp. leads to a complete phagosome maturation arrest [8].

Melanized non-*Aspergillus* molds include a wide range of different fungi species, most importantly *Mucormycetes*, followed by *Fusarium* spp. (which may sometimes present as black mold) and *Scedosporium/Lomentospora* spp. but this may vary among geographic regions and transplant types [1, 9, 10]. Others include dematiaceous fungi from which the most important pathogen is *Alternaria* spp. in SOT recipients [11] and other black molds like *Paecilomyces* [12]. While black molds are rarely the cause of invasive infection even in the immunocompromised host, they are important to consider as individual morbidity and mortality are high

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and therapy may substantially diverge in relation to more frequent IFIs [9, 12, 13]. Here we will review epidemiology, diagnosis, and management of black mold infections in SOT recipients.

Epidemiology

IFIs from black molds are rare diseases, and only few larger multicentric cohort studies are available for reliable estimation of prevalence rates [12]. Additionally, the data should be interpreted with caution since the studies were performed two decades ago and awareness of these pathogens as well as diagnostic possibilities has since improved. Furthermore, analysis of collected case series may include a significant selection bias [14].

Overall, prevalence of rare invasive mold infections (IMIs) tends to be increasing [14, 15•, 16, 17]. Whether this is due to an increased recognition of these infections, better diagnostic tools, or a larger at-risk population with concordant increased infection rates remains speculative but all of these factors may contribute to this observation. Importantly, prevalence rates of rare IMIs diverge widely among different geographic areas and even between centers within the same countries.

Fungi from the order Mucorales are responsible for mucormycosis. From this large group, 11 genera and about 27 species have been identified to cause human infection, most importantly *Rhizopus* spp. which account for about 50–70% of mucormycosis [1, 18, 19]. SOT recipients constitute for 3–14% of all mucor infections [15•, 16, 19, 20, 21]. Mucormycosis make up about 2% of all IFIs in SOT recipients while the distribution according to transplant type varies widely among reports [1, 2, 4, 19, 20, 21]. Common risk factors seen in SOT recipients are the use of corticosteroid and immunosuppressive agents, diabetes mellitus, malnourishment, renal failure, and prior therapy/prophylaxis with voriconazole/echinocandins [22].

In fusariosis, only a small proportion of the more than 300 species cause invasive infection. While *Fusarium* spp. are not classic "black molds," there are reports of melanin-producing spp. with positivity in Fontana-Masson stain of up to 30% and will therefore be covered here [23, 24, 25]. *Fusarium solani* spp. and *F. oxysporum* spp. are the most commonly identified in SOT recipients as well as in other at-risk populations [10]. The main pathway of infection is usually the inhalation of airborne microconidia or direct inoculation due to trauma, which leads to infection of the airways (sinuses, lung) and the soft tissue as most infected sites [9, 12]. Invasive fusariosis account for < 1% of all IFIs in SOT recipients [1, 10]. Lung transplant recipients tend to be at higher relative risk, most likely due to the main portal of entry through the respiratory tract [1, 10, 26].

Scedosporium and *Lomentospora* make up about 1% of all IFIs in SOT recipients, primarily infecting lung transplant recipients [1]. The most identified species come from the *S. apiospermum* complex followed by *Lomentospora prolificans*, which is now distinguished from *Scedosporium* [1, 9]. Recent single-center studies report an incidence of 2–3% in lung transplant recipients [27, 28, 29]. Identified risk factors for scedosporiosis include prior colonization (many centers consider pre-transplant colonization as contraindication to lung transplant) and prior use of amphotericin B, in addition to general risk factors for IFI [12]. The main site of infection is the lower respiratory tract, but SOT recipients are also at risk for developing disseminated disease [27, 29].

Infections due to dematiaceous fungi make up to 2.5% of all IFIs in SOT recipients [11]. This heterogenous class of fungi summarizes more than hundred species; *Alternaria* spp. is the most common pathogen in SOT recipients. Most frequently infected sites include the skin and the underlying soft tissue, although the proportion of these cases may be overestimated as other manifestations of the disease are harder to diagnose. Dematiaceous fungi can also infect the lung, sinuses, cerebrum, and bones/joints [11]. While an analysis of the Transplant-Associated Infection Surveillance Network (TRANSNET) population showed disseminated disease in 63%, controversially a recent systematic review of phaeohyphomycosis cases in SOT recipients reported a much lower rate of dissemination (11%) [11, 30•].

Case reports have also reported on black mold infections in SOT recipients caused by *Rasamsonia*, *Paecilomyces*, and *Penicillium* spp. [31, 32, 33, 34, 35].

Diagnosis

IFIs and particularly those caused by rare molds are difficult to diagnose. First, diagnosis requires clinical suspicion while symptoms are mostly nonspecific and range widely from a simple cough or even asymptomatic patients to septic shock [36]. After that, finding the right diagnostic approach is crucial and includes imaging, biomarkers, and obtaining samples for microbiological and molecular testing and histopathological processing. The diagnosis of rare mold infections becomes even more challenging in light of a low pre-test probability and the fact that invasive aspergillosis shares some risk factors and clinical appearance as well as radiological findings with the discussed pathogens [37]. Therefore, a diagnosis can only be made with identification of these molds from optimally otherwise sterile samples, which can also be used to distinguish between colonization and infection [9, 12].

Mucormycosis in SOT recipients usually involves the lung; however, it can also manifest as infection of the sinuses (with cerebral involvement), skin, and gastrointestinal tract

(mostly the stomach in SOT recipients), as well as infection of any other organ (e.g., endocarditis, pyelonephritis) or as disseminated disease [38]. Computer tomography (CT) is considered the first-line imaging of pulmonary infection or sinusitis, while intracranial involvement can be examined better through magnetic resonance imaging (MRI) [13, 39]. Along with features of pulmonary mold infection in CT (e.g., masses, cavities, halo sign, or air-crescent sign in aspergillosis), ≥ 10 nodules, pleural effusion, and concomitant sinusitis have been suggested as characteristics that differentiate invasive pulmonary aspergillosis (IPA) from pulmonary mucormycosis (PM) [40]. A radiological sign that might suggest PM is the reversed halo sign, but similar to the typical radiological signs for aspergillosis these findings were mostly from neutropenic patients with underlying hematological malignancies [40, 41, 42], while radiological presentation may differ in non-neutropenic patients.

Once a suspicion of mucormycosis has been established based on host susceptibility and clinical and radiological findings, all effort should be made to obtain samples to confirm the diagnosis (e.g., BAL or CT-guided biopsy in PM, skin biopsy in cutaneous infection, gastroscopy in gastrointestinal involvement). Direct microscopy preferably using a fluorescent brightener might support a presumptive diagnosis [13, 22]. Mucorales show hyphae that are at least 6–16-µm wide, ribbon-like, and pauci-septate, and branch irregularly, and better visualized with special stains like Grocott methenamine silver (GMS) or periodic acid-Schiff (PAS) [12]. Additionally, culture of specimen can confirm the diagnosis and enable susceptibility testing, with a sensitivity of about 50% [13]. Currently PCR-based techniques are intensively evolving in this field and also show promising results when tested directly from blood or BAL, while still lacking standardization [43]. Fresh material is preferred over formalin-fixed or paraffin-embedded tissue (FFPE) [44]. In a recent study evaluating PCR of serum samples from patients with probable or proven mucormycosis, specificity and sensitivity were about 85 and 90%, respectively [45••].

Fusariosis in SOT recipients usually manifests as infection of the respiratory tract (pneumonia, sinuses), or the skin/soft tissue, as well as disseminated disease with the ability to infect any other organ [12, 46]. In a recent Spanish study, non-neutropenic patients were more likely to have localized infections with pneumonia as the most common manifestation (64.3%), while cutaneous manifestations (21.4%) were less common when compared to neutropenic patients [17]. Blood culture might be positive in about 40% of the cases [47] with higher positivity rates observed in disseminated disease [48]. *Fusarium* spp. show cross-reactivity with *Aspergillus* galactomannan assays and appear quite similar morphologically in histopathologic specimen; therefore, the distinction might be additionally challenging [9, 12]. Also, BDG is usually positive in invasive infection [9]. In addition to culture, matrix-assisted laser desorption/ionization-time of flight mass spectrometry (MALDI-TOF MS) and PCR-based techniques may be used to investigate the fungus to species level [49, 50, 51].

In a retrospective observational study from France, *Scedosporium* spp. and *Lomentospora* mainly infected the lungs, the skin, the bones and joints, and the cerebrum, with the proportion of disseminated disease reaching nearly 50% (with more cerebral, cardiovascular, or osteoarticular locations involved when compared to hematological malignancies) [52]. The diagnostic approach is similar to previously mentioned molds with focus on obtaining specimen for histopathological processing and microbiological testing [9]. If scedosporiosis/lomentosporiosis is suspected, culture requires an additional medium like the Scedosporium Selective Agar (SceSel +) to prevent the overgrowth from faster growing fungi (e.g., *Aspergillus*) [9, 53]. Again, tools to compliment culture and histopathology are MALDI-TOF MS and molecular-based methods [53, 54, 55].

In contrast to the other discussed molds, dematiaceous fungi primarily cause local infection of the skin/soft tissue, clinically manifesting as a variety of papules, plaques, nodules, and subcutaneous masses [14, 56]. Others include infections of the central nervous system (mostly seen in liver transplant), the lungs (predominantly in lung transplant), and disseminated disease [14]. Reported activities before medical care are consulted and distribution of cutaneous lesions suggests direct inoculation as the most common infection pathway [14, 30•]. Direct microscopy may show melanized hyphae in Fontana-Masson staining, but this finding should be interpreted with caution since many other molds (e.g., *Fusarium* spp., *Aspergillus* spp.) can lead to positive results [23]. Confirmation of suspected phaeohyphomycosis requires confirmation by histopathology along with culture and may be assisted by molecular identification techniques mentioned above, especially to aid species identification [14]. It should be noted that culture may show a slow growth [57, 58].

Treatment

Treatment of these rare molds is difficult and often requires susceptibility testing for systemic antifungal treatment. When clinically feasible, surgical therapy of localized infections is an important adjunct [9, 12]. For mucomycosis, high-dose LAmB (5–10 mg/kg/day) is currently considered the first-line therapy and shows overall response rates of about 40% [13, 59]. The European Confederation of Medical Mycology (ECMM) guideline on mucormycosis currently recommends an initial dose of LAmB of 10 mg/kg/day and to avoid slow escalation of doses [13]. Additionally, early surgical resection with clean margins plays a key role, since

the angioinvasive nature of the disease hinders the penetration of antifungal pharmaceutics in infected tissue [60, 61]. If LAmB cannot be given, the alternatives isavuconazole and posaconazole can be considered [13]. In a case–control analysis, patients from the VITAL study receiving isavuconazole were matched with patients from the FungiScope Trial which received LAmB. No significant difference was found in all-cause mortality at day 42 (33 to 39%) [62]. If progressive disease is noticed, salvage therapy may be attempted by switching between the mentioned drugs [13]. Additionally, combination therapy between LAmB and either isavuconazole or posaconazole might be an option for escalation. While combination therapy showed synergistic effects in animal model, improved outcomes in the clinical setting could not be proven [63] Future treatment options may include fosmanogepix and ibrexafungerp which have both shown synergism with LAmB in animal studies [64••]. Optimal therapy duration has not been established yet. Decision about duration should be made on an individual basis according to radiological resolution and clinical response [13]. Reported mortality rates are high ranging from 30 to 50% [16, 20].

Fusarium spp. show variable resistance to polyenes and extended spectrum triazoles. Since delayed treatment has been associated with worse outcomes, an empiric combination therapy with LAmB and voriconazole seems reasonable until susceptibility data is available. Alternatively, monotherapy with one of the mentioned antifungals might be given [9, 12, 65]. Additionally, surgical debridement of localized infections is a cornerstone in the management of fusariosis [66]. For second-line therapy, posaconazole and isavuconazole are options, which both showed success in treating fusariosis with response rates of about 50% (comparable to those of LAmB and voriconazole) [65, 67, 68, 69], as salvage therapy posaconazole is recommended by most guidelines [9, 12]. Future treatment options may include fosmanogepix and olorofim, which are promising novel classes of antifungals in clinical development for treatment of not only fusariosis but also scedosporiosis and lomentosporiosis [64••].

Lomentospora prolificans show intrinsic resistance to most antifungals with voriconazole and posaconazole showing the highest in vitro susceptibility [70]. Terbinafine was found to have synergistic effects in combination with voriconazole [71]. In fact, this combination therapy showed improved outcomes in many case series against therapy with other antifungals [9, 72].Therefore, combination therapy currently is preferred over monotherapy with voriconazole [9, 12]. While a combination of a triple therapy of polyene with voriconazole and echinocandin has shown synergism in vitro, there are limited data in the clinical setting [65].

In scedosporiosis, monotherapy with voriconazole is the antifungal of choice [9, 12, 73]. In a relatively large case series of 107 patients with scedosporiosis treated with voriconazole, SOT recipients represented the largest subgroup (22%) with response rates of 63%. Duration of therapy varied widely from 1 to 802 days (median 103 days), underlining the heterogeneity of this disease [74]. Other azoles like posaconazole or isavuconazole may be considered second-line therapy, but evidence is exceedingly scarce. The role of early surgical debulking has to be stressed once again, as it has shown increased survival in SOT recipients [75].

Located superficial phaeohyphomycosis may be cured with surgical debridement alone, but can be augmented with triazoles, while disseminated disease and deep foci require systemic antifungal treatment [12]. Again, treatment can be difficult due to the variable susceptibility of this heterogenous group [76]. Among the azoles, the best experience is with itraconazole for cutaneous/subcutaneous phaeohyphomycosis with voriconazole considered alternative first-line therapy [9, 76, 77]. Successful treatment with isavuconazole or posaconazole has been described, but optimal treatment remains unclear [14, 78, 79]. In case of disseminated infection, generally combination therapy is initially warranted with either voriconazole or posaconazole plus echinocandin or voriconazole plus terbinafine. LAmB might be an alternative option especially when considering that *Alternaria* spp. (which make up for approximately a third of the cases in SOT recipients) usually show susceptibility to this antifungal [76]. Outcome depends on the infected site. While local superficial infection shows good prognosis with adequate therapy and response rates of 84%, in disseminated disease mortality rates reached 32–69% [14, 30•].

A tabular summary of the discussed rare mold infections with respect to risks as well as diagnostic and treatment considerations is presented in Table 1.

Conclusions

The diagnosis of an IFI is not easy to establish and is based on the clinical suspicion in a patient at risk together with radiological and microbiological findings, such as biomarkers, fungal culture, or PCR-based techniques [9]. Uncommon black molds are even more difficult to diagnose, since they might have a similar clinical presentation to invasive aspergillosis (IA) [10]. Additionally, some of these emerging fungi might show cross-reactivity for the galactomannan assay (e.g., *Fusarium* spp.) used primarily for the diagnosis of IA [47, 80]. Since the therapeutic regimen varies between the different pathogens and delayed therapy is associated with worse morbidity and mortality, the distinction is crucial for patient's outcome.

Another point to make is the lack of diagnostic and therapeutic standard procedure in reality, while clinical manifestations and patient population are also very heterogenous

Table 1 Overview of the discussed black mold infections in SOT recipients

	Mucormycosis	Fusariosis	Scedosporiosis/Lomentosporiosis	Phaeohyphomycosis
Epidemiology (% of IFI in SOT) [1, 2, 4, 10, 11]	≈ 2%	< 1%	≈ 1%	≈ 2.5%
Risk according to transplant type [1, 2, 11, 22, 30•]	Risk tends to be higher in liver, lung, and heart transplant	Risk tends to be higher in lung transplant	Lung transplant recipients are at higher risk	Risk tends to be higher in lung and kidney transplant
Diagnostic considerations [13, 14, 43, 45••]	1,3-β-D-glucan usually negative Mucorales qPCR from serum - 85.2% sensitivity - 89.8% specificity	1,3-β-D-glucan usually positive Serum and BAL-GM might be positive	1,3-β-D-glucan usually positive Selective culture media (e.g., SceSel +) increases isolation rates	1,3-β-D-glucan poorly studied Culture may show slow growth
First-line medical therapy [9, 12, 13]	High-dose LAmB (5–10 mg/kg/day)	Voriconazole + LAmB Until susceptibility testing is available	Lomentosporiosis: -Voriconazole + terbinafine Scedosporiosis: -Voriconazole	Localized infection: -Itraconazole, voriconazole, and posaconazole Disseminated infection: -Posaconazole/voriconazole + echinocandins -Voriconazole + terbinafine (consider LAmB in alternariosis)

SOT solid organ transplant, *IFI* invasive fungal infection, *qPCR* quantitative polymerase chain reaction, *BAL* bronchoalveolar lavage, *GM* galactomannan, *LAmB* liposomal amphotericin B

[56]. The ECMM more recently addressed this issue by creating a score to help physicians measure guideline adherence in this matter, hence a form to measure standardization [81, 82]. Since randomized controlled trials seem out of reach, this together with rigorously reporting all cases in a central register may help to draw valid conclusions and drive scientific effort to improve outcomes of these infections in different populations.

Antifungal therapy of black mold infections currently consists mostly of mold active azoles and LAmB, although there are significant differences between the pathogens, with some showing intrinsic resistance to mold active azoles, while others show frequently high MICs against LAmB [9]. In the SOT population with a significant proportion of organ dysfunction, toxicity (hepatotoxicity for triazoles; nephrotoxicity for LAmB) needs always to be taken under consideration.

Additionally, in the presence of common immunosuppressive agents used in SOT, potential drug-drug interactions have to be considered [83, 84]. Since LAmB is not metabolized by cytochrome P-450 (CYP450) enzymes, drug-drug interactions are generally less an issue, but coadministration of nephrotoxic agents which also accounts for common immunosuppressants like tacrolimus or cyclosporin may sometimes pose a problem [85]. In contrast, azoles are known for their countless drug-drug interactions due to their interference with hepatic and intestinal CYP450, which can increase the levels of common immunosuppressants. As a consequence, measurements of plasma concentrations not only of those systemic antifungals that frequently interact (i.e., voriconazole) but also immunosuppressants with subsequent dose modifications are highly recommended. Compared to voriconazole, isavuconazole may be an attractive option in SOT recipients, since the effect on CYP450 is lower and pharmacokinetics are more predictable [86, 87]. Also, novel antifungal pharmaceutics like fosmanogepix, ibrexafungerp, and olorofim may be of great use in this matter [64••]. Particularly, olorofim and fosmanogepix show promising results in the treatment of fusariosis and scedosporiosis/lomentosporiosis which are resistant to nearly all currently used antifungals. It should be noted that olorofim is a weak inhibitor of CYP 3A4 and ibrexafungerp is a reversible inhibitor of CYP2C8 and CYP3A4. Nevertheless, the current data suggests that these antifungals are not likely to have a clinically relevant impact on the drug levels of current immunosuppressive agents. The evaluation of fosmanogepix in this matter has not been published yet (NCT04166669) [64••].

In conclusion, rare mold infections continue to emerge in SOT recipients and pose significant challenges to diagnosis and clinical management. Knowledge of local epidemiology and a high level of awareness are necessary for early diagnosis and a successful outcome.

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Declarations

Conflict of Interest MH received research funding from Astellas, MSD, Gilead, Scynexis, Pfizer, Euroimmun, and Pulmocide. All other authors declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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