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Opinion Article

Opportunistic *Lomentospora* and *Scedosporium* Infections in Transplant Patients

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DESCRIPTION

Opportunistic fungal infections have emerged as a major threat to immunocompromised individuals, particularly those undergoing organ or hematopoietic stem cell transplantation. Among the most concerning fungal pathogens are *Lomentospora prolificans* (formerly *Scedosporium prolificans*) and species within the *Scedosporium apiospermum* complex. These fungi are known for their environmental ubiquity, intrinsic resistance to multiple antifungal agents, and their ability to cause disseminated, life-threatening infections in susceptible hosts. The incidence of these infections has been rising, in part due to advances in transplantation procedures, improved survival rates of immunocompromised patients, and increased awareness and diagnostic capabilities.

In transplant recipients, the risk of opportunistic fungal infections is magnified due to prolonged neutropenia, high-dose corticosteroid use, and immunosuppressive regimens designed to prevent graft-versus-host disease or organ rejection. *Lomentospora* and *Scedosporium* species are capable of exploiting such immunological vulnerabilities to cause invasive infections, most frequently affecting the lungs, skin, soft tissues, and central nervous system. Unlike more common fungi like *Candida* or *Aspergillus*, these pathogens are difficult to eradicate due to their robust biofilm formation, intrinsic resistance to most antifungal drugs, and delayed or missed diagnosis.

The pathogenesis of these fungi involves a complex interplay between fungal virulence factors and host immune status. Both *Lomentospora* and *Scedosporium* exhibit traits such as thermotolerance, adhesion to host tissues and medical devices, and resistance to phagocytosis. Their ability to survive and multiply in neutrophil-depleted environments is a critical feature contributing to their pathogenicity in transplant patients. Moreover, these fungi possess a remarkable genetic plasticity that enables them to upregulate drug efflux pumps, modify ergosterol biosynthesis pathways, and produce melanin and other protective factors that impair antifungal efficacy.

Clinical presentation of these infections can vary, but often includes fever unresponsive to antibacterial treatment, cough, dyspnea, hemoptysis, cutaneous nodules, and neurologic signs suggestive of CNS involvement. Imaging studies may reveal multifocal infiltrates or abscesses, but definitive diagnosis requires microbiological and molecular identification. Traditional culture methods are often slow and insensitive, while histopathology can be non-specific. Molecular diagnostic tools such as PCR and MALDI-TOF mass spectrometry have greatly improved the speed and accuracy of species-level identification, although access to these tools remains limited in many clinical settings.

Therapeutic management of *Lomentospora* and *Scedosporium* infections in transplant patients remains a significant challenge. These organisms are notoriously resistant to the majority of available antifungals. *Lomentospora prolificans*, in particular, exhibits near-universal resistance to amphotericin B, triazoles, and echinocandins. Combination therapy, often involving voriconazole and terbinafine, is commonly attempted, but with limited clinical success. Newer triazoles such as isavuconazole and posaconazole offer some promise against *Scedosporium apiospermum*, though susceptibility varies. Due to the multidrug-resistant nature of these fungi, the success of treatment frequently hinges on surgical debridement of infected tissues and immune reconstitution.

One emerging area of interest is host-directed therapy, aiming to enhance immune responses to fungal pathogens through cytokine therapy or adoptive transfer of antifungal T cells. Additionally, advances in fungal genomics and transcriptomics have shed light on the molecular mechanisms underpinning antifungal resistance, opening new avenues for drug target discovery. Genes encoding efflux pumps, such as *cdr1B* and *mdr1*, and mutations in *cyp51A* associated with azole resistance have been documented, albeit less thoroughly than in *Aspergillus*. The lack of robust animal models and difficulty in conducting randomized clinical trials for rare infections remain barriers to progress in this area.

Another critical gap in management is the lack of standardized susceptibility testing protocols for these fungi. While the Clinical and Laboratory Standards Institute (CLSI) and the European Committee on Antimicrobial Susceptibility Testing (EUCAST) provide breakpoints for common fungal pathogens, such guidelines for *Lomentospora* and *Scedosporium* are still evolving.

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This hampers the ability of clinicians to make informed therapeutic decisions and contributes to the empiric use of broad-spectrum antifungals, which may further drive resistance.

Public health and clinical awareness of these rare but deadly infections must be increased. Registry-based surveillance systems can help track incidence, resistance trends, and outcomes, providing valuable data for informing clinical practice and policy. Multidisciplinary approaches involving infectious disease specialists, mycologists, microbiologists, surgeons, and transplant physicians are essential for optimizing outcomes. Education and training in mycology should also be emphasized in medical curricula to prepare future clinicians to recognize and manage these complex infections.

In conclusion, *Lomentospora* and *Scedosporium* infections represent a formidable challenge in the care of transplant recipients. Their intrinsic antifungal resistance, ability to cause severe disseminated disease, and the limited efficacy of current treatment regimens demand a concerted effort toward early detection, improved diagnostics, and the development of novel antifungal agents. With the continued rise in transplantation procedures and immunocompromised populations, addressing the threat posed by these emerging opportunistic molds is of paramount importance for the future of transplant medicine.