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Opinion Article

Cryptic Keratitis: Paecilomyces as a Rare Trigger for Post-Surgical Corneal Melting

Linda Williams*

Department of Texas Health Science Center at San Antonio, Floyd Curl Drive, San Antonio, TX, USA

DESCRIPTION

Post-operative ocular complications often present in predictable patterns, enabling clinicians to rely on established diagnostic frameworks and therapeutic approaches. However, not all postoperative inflammations follow textbook behavior some are subtle, deceptive, and far more destructive than they initially appear. Among the rare yet clinically significant culprits is paecilomyces, an opportunistic filamentous fungus capable of infiltrating the cornea following even routine ocular procedures. Although uncommon, paecilomyces keratitis is associated with a unique challenge: its cryptic presentation delays diagnosis, its intrinsic resistance to conventional antifungal therapy complicates treatment, and its enzymatic activity accelerates corneal degradation, ultimately leading to a devastating condition known as corneal melt.

Corneal melting characterized by stromal liquefaction, collagen breakdown, and progressive thinning is a feared complication in corneal pathology, particularly when triggered by microbial agents. In the context of paecilomyces infection, the risk becomes even more pronounced due to the organism's capacity for deep stromal penetration and its ability to evade early clinical suspicion. The postoperative eye, particularly one with a clear corneal incision, becomes a vulnerable environment where microbial entry, impaired wound healing, and weakened structural integrity converge to create fertile ground for infection.

This commentary aims to unpack the clinical importance of paecilomyces as an understated yet dangerous postoperative pathogen. It explores how this fungus evades detection, the mechanisms through which it induces corneal melting, the unique diagnostic and therapeutic challenges it presents, and the implications for surgical practice. By examining these factors closely, clinicians can better appreciate the importance of early suspicion and proactive management when encountering atypical keratitis in the postoperative phase. *Paecilomyces* species, primarily *paecilomyces lilacinus* and *paecilomyces variotii*, are saprophytic fungi that thrive in soil, decaying plant matter, and moist environments. Their ubiquity in the natural world does not translate to frequent ophthalmic infections; instead, their pathogenicity emerges in selective conditions involving compromised ocular surfaces or breaches in corneal integrity. What makes paecilomyces particularly concerning in postoperative settings is its resilience this organism demonstrates inherent resistance to several frontline antifungal medications, especially natamycin and amphotericin b.

Though rare, when paecilomyces enters the postoperative corneal environment, it behaves as a highly adaptive invader. The compromised immunological landscape of the surgically manipulated eye allows it to colonize tissues with minimal early inflammation. Its subtle but persistent growth gives rise to deep stromal infiltrates that often mimic noninfectious causes such as sterile melt or immune-mediated inflammation. This masquerading behavior is what makes paecilomyces particularly dangerous: Its pathogenic potential is overlooked until significant structural damage has already occurred. Clear corneal incisions common in cataract surgery and many minimally invasive ocular interventions have significantly advanced modern ophthalmology by enabling rapid recovery and minimal patient discomfort. Yet, precisely because these incisions traverse the corneal layers with fine precision, they create microchannels that may unintentionally facilitate microbial entry in the early postoperative phase. Even a minor wound leak or insufficient sealing can allow environmental fungi to invade deeper corneal layers.

The postoperative environment is further compromised by temporary neurologic and immunologic changes. Desensitization of corneal nerves reduces the eye's natural blinking reflex, while steroid use common after surgery can diminish local immunity and prevent timely recognition of infection. In the presence of paecilomyces, these combined factors create an ideal scenario for colonization. Once inside, the organism can anchor to the stromal collagen matrix and begin releasing destructive enzymes, initiating tissue breakdown long before overt clinical symptoms emerge. One of the defining characteristics of *paecilomyces keratitis* is its cryptic initial presentation. Whereas bacterial keratitis often presents with dramatic symptoms intense pain, dense infiltrates, and copious discharge paecilomyces infections

Correspondence to: Linda Williams. Department of Texas Health Science Center at San Antonio, Floyd Curl Drive, San Antonio, TX, USA, E-mail: **lindawilliam@ck.pi**

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follow a more subdued trajectory. Early signs might include mild foreign body sensation, slight haze, or minimal redness. These subtle manifestations are easily mistaken for normal postoperative irritation or steroid-responsive inflammation.

Moreover, routine smear and culture techniques may fail to detect the organism in the early stages. Paecilomyces grows slowly, does not always appear on common fungal media, and may require extended incubation. Consequently, empirical therapy is frequently misdirected toward bacterial pathogens, leaving the fungal infection unchecked. This delay is clinically significant: by the time paecilomyces is identified, the corneal architecture may already show signs of melting or deep stromal involvement. The lack of pathognomonic features also complicates diagnosis. Stromal infiltrates may appear feathery, granular, or ring-like, but these findings are not exclusive to fungal infections. Advanced diagnostic tools like confocal microscopy, PCR-based fungal DNA analysis, or specialized stains may be required to confirm the organism's presence. The need for such sophisticated diagnostics further underscores the importance of early suspicion, particularly in patients with atypical postoperative presentations.

Corneal melting is the culmination of destructive biochemical processes that compromise the structural integrity of the cornea. In Paecilomyces infections, this process is driven by both fungal and host-derived factors. The organism secretes proteolytic enzymes such as metalloproteinases that degrade stromal collagen. These enzymes not only liquefy tissue but also trigger an inflammatory cascade that recruits neutrophils to the site of infection. Neutrophils, in turn, release collagenolytic enzymes as part of their immune response. The resulting enzymatic overload overwhelms the corneal repair mechanisms, creating a downward spiral of tissue destruction. The cornea becomes progressively thinner, more fragile, and at risk of perforation. In addition, fungal hyphae can penetrate deep stromal layers, creating channels that further destabilize the corneal matrix.

Clinically, this melting process may initially manifest as localized thinning or surface irregularity but can rapidly escalate into extensive stromal loss. In severe cases, descemet's membrane becomes exposed or ruptures, leading to full-thickness perforation. Once melting begins, halting its progression becomes significantly more difficult, requiring aggressive antifungal therapy and sometimes urgent surgical intervention. Managing paecilomyces keratitis is complicated by the organism's intrinsic resistance to several first-line antifungal medications. Natamycin, commonly used for filamentous fungal keratitis, is often ineffective against paecilomyces species. Likewise, amphotericin b shows limited efficacy. This resistance narrows the therapeutic window and increases the urgency for accurate diagnosis. Voriconazole has emerged as the most effective antifungal agent against paecilomyces due to its excellent corneal penetration and strong activity against filamentous fungi. However, the success of treatment depends heavily on early initiation. Delayed therapy or initial misdiagnosis leading to reliance on ineffective medications allows the infection to progress into deeper stromal layers, increasing the risk of irreversible damage. Even with optimal antifungal therapy, adjunctive measures such as debridement, amniotic membrane grafting, or collagen crosslinking may be necessary to stabilize the cornea. In advanced cases, surgical intervention in the form of therapeutic keratoplasty becomes unavoidable. Unfortunately, even after transplantation, recurrence of infection remains a possibility if fungal elements extend beyond the excised tissue.

The occurrence of paecilomyces-related corneal melt in postoperative patients highlights the need for heightened clinical vigilance. Surgeons must remain aware that fungal infections, though rare, can complicate even routine procedures. Environmental contamination, improper medication storage, or insufficient sterilization protocols may increase the risk of fungal entry. Postoperative follow-up should include careful assessment for atypical signs that deviate from expected healing patterns. Any suspicion of fungal involvement warrants prompt culture, specialized diagnostic testing, and early initiation of antifungal therapy. Steroid use must also be carefully monitored, as these medications can mask symptoms while exacerbating fungal proliferation. On the preventive front, maintaining strict aseptic surgical protocols, using sterile balanced salt solutions, and educating patients about medication hygiene can significantly reduce the risk of postoperative fungal contamination. For high-risk patients those with chronic ocular surface disease, immunocompromise, or environmental exposure clinicians should maintain a higher index of suspicion for unusual infectious agents.

CONCLUSION

Paecilomyces keratitis is an uncommon but formidable postoperative complication capable of progressing from subtle inflammation to catastrophic corneal melt if not promptly recognized. Its cryptic presentation, resistance to standard antifungal agents, and destructive enzymatic activity make it a particularly challenging pathogen to manage. In postoperative eyes especially those with clear corneal incisions the risk of microbial invasion and delayed detection is amplified, underscoring the importance of careful monitoring and diagnostic precision.

Ultimately, awareness of paecilomyces as a potential cause of postoperative keratitis can significantly improve clinical outcomes. Early suspicion, timely diagnostic interventions, and rapid initiation of appropriate antifungal therapy, particularly with voriconazole, are critical for preventing severe tissue damage. By understanding the unique behavior of this opportunistic fungus and recognizing its ability to trigger corneal melting, ophthalmologists can adopt more proactive strategies to preserve corneal integrity and protect postoperative vision.