ECRONICON OPEN ACCESS

EC OPHTHALMOLOGY
Case Report

Microsporidial Keratoconjunctivitis Recalcitrant to Topical Ofloxacin in an Immunocompetent Pediatric Patient

Kelly McCormack and Ryan Mercer*

Ophthalmology, The Eye Center, P.A, USA

***Corresponding Author:** Ryan Mercer, The Eye Center, P.A, Columbia, SC, USA.

Received: April 07, 2026; **Published:** July 24, 2026

Abstract

Introduction: The phylum of microsporidia consists of several different genera of resilient, obligate, eukaryotic, spore-forming, intracellular eukaryotes. Microsporidia often affects the cornea and conjunctiva and results in microsporidial keratoconjunctivitis. Risk factors for microsporidial keratoconjunctivitis include ocular trauma, contact lens wear, immunodeficiency, and exposure to contaminated soil or water. We present a case of severe, unilateral microsporidial keratoconjunctivitis in an immunocompetent eight-year-old male with no known risk factors that was successfully treated with topical voriconazole and moxifloxacin after failed response to topical ofloxacin.

Case Presentation: An eight-year-old male presented to an outside eyecare provider with a one-week history of worsening redness, tearing, mucus discharge, and blurred vision of the left eye without an identifiable inciting event. The patient progressed over the following week and developed subepithelial infiltrates, superficial punctate keratitis, and worsening mucus discharge despite being treated with neomycin-polymyxin-dexamethasone (neo-poly-dex) drops. Neo-poly-dex was stopped and he was transitioned to topical ofloxacin ophthalmic drops before being referred to our eye center. No clinical response was observed with ofloxacin drops, so the patient was transitioned to a regimen of topical voriconazole drops, topical moxifloxacin drops, and erythromycin ointment in the left eye. This combination of treatment resolved the patient's signs and symptoms and returned his best-corrected visual acuity (BCVA) back to baseline.

Conclusion: First-line therapy for microsporidial keratoconjunctivitis is topical fumagillin, which is currently unavailable in the United States of America. We present a case of successfully treated microsporidial keratoconjunctivitis in an immunocompetent pediatric patient without known risk factors who failed monotherapy with topical ofloxacin. This case report highlights the effectiveness of combination therapy with topical voriconazole and moxifloxacin for microsporidial keratoconjunctivitis that has failed monotherapy with topical ofloxacin. Furthermore, clinicians should be aware that stromal inflammation following microsporidial keratoconjunctivitis may not represent persistent infection, but instead, may represent an immune stromal keratitis (ISK) that rapidly responds to steroids. Accurate differentiation between persistent infection and ISK is essential to guide appropriate management and optimize recovery.

Keywords: *Microsporidia; Immunocompetent; Pediatrics; Infectious Keratitis; Microsporidial Keratitis; Microsporidial Keratoconjunctivitis*

Abbreviations

BCVA: Best-Corrected Visual Acuity; ISK: Immune Stromal Keratitis; IOP: Intraocular Pressure; PCR: Polymerase Chain Reaction; neo-poly-dex: Neomycin-Polymyxin-Dexamethasone

Introduction

Microsporidia are eukaryotic, obligate, spore-forming intracellular parasites [1]. These organisms are known to cause pathology in multiple organ systems, most commonly gastrointestinal infections, but can also infect the ocular surface [2]. Microsporidial keratoconjunctivitis is a rare ocular infection that can be difficult to treat because of the recalcitrant nature of microsporidia and the limited number of available therapies [1-3]. Historically, microsporidial keratitis was thought to predominate in immunosuppressed individuals, however, an increasing number of cases have been reported in immunocompetent patients since the turn of the millennium with many cases originating in Asia [3]. For example, Chan, Cordelia M.L., *et al*. reports a series of six cases of microsporidial keratoconjunctivitis in Singapore in non-immunocompromised individuals [4]. Joseph, Joveeta., *et al*. reports on 19 cases of microsporidial keratitis in southern India in immunocompetent individuals [5].

Ocular microsporidial infections typically present as either a stromal keratitis or superficial keratoconjunctivitis, with the former being more common in immunocompetent individuals and the latter being more common in immunosuppressed patients [2,3]. The most common genera causing microsporidial keratitis include *Encephalitozoon* and *Nosema* [2]. *Encephalitozoon* species are often confined to the epithelium leading to a superficial keratoconjunctivitis [2]. On the contrary, *Nosema* species are associated with deeper tissue involvement resulting in microsporidial stromal keratitis [2,3]. Risk factors for microsporidial keratitis include contact lens use, trauma, surgery, topical corticosteroids, and exposure to contaminated water/mud/soil [3]. Topical fumagillin has been proposed as first line therapy to treat ocular microsporidiosis but is unavailable in the United States of America [2]. Oral albendazole has also been proposed, but there are associated systemic side effects and can be difficult to obtain due to cost. For these reasons, case reports highlighting effective alternatives against microsporidia are useful in guiding management.

In this study we report a case of unilateral, acute, severe, microsporidial keratoconjunctivitis in an immunocompetent eight-year-old boy who was treated with a regimen of topical voriconazole and moxifloxacin. He proceeded to develop an immune stromal keratitis (ISK), which led to the addition of topical loteprednol etabonate. Pediatric cases of microsporidial keratoconjunctivitis in immunocompetent patients remain rare, and this case highlights a therapeutic approach in cases where standard treatments fail or are unavailable.

Case Report

An immunocompetent eight-year-old boy presented to a local eye care provider with redness, tearing, mucus discharge, and blurred vision of the left eye without an identifiable inciting event. The patient had no history of systemic illness, contact lens use, ocular trauma, or recent travel. His right eye had no clinically significant findings.

Initial evaluation by the outside eye care provider revealed moderate conjunctival injection and mucus discharge after which he was started on neomycin-polymyxin-dexamethasone (neo-poly-dex) ophthalmic solution four times a day to the affected eye for five days then twice a day for two days. According to his mother, symptoms improved after two days but worsened on the third day. Due to a suspected adverse reaction, neo-poly-dex was changed to 0.3% topical ofloxacin every two hours to the affected eye.

The patient's condition continued to decline after day 10 of treatment. At this point, the left eye developed subepithelial infiltrates and superficial punctate keratitis, which were new examination findings. Best-corrected visual acuity (BCVA) of the left eye at the outside eye care providers office was 20/25 and intraocular pressure (IOP) was 18 mm Hg.

The patient was first seen in our clinic, The Eye Center, P.A. in Columbia SC, on day 13 after his initial treatment. During his initial presentation in our clinic, his BCVA was 20/40-2 and his intraocular pressure (IOP) was 28 mmHg. Due to increased IOP, poor response to antibiotic therapy, and clinical appearance of the cornea, the patient was presumed to have herpetic keratitis (Figure 1). Ofloxacin was discontinued and treatment with oral acyclovir suspension (300 mg four times a day) with preservative-free artificial tears was initiated. Also at this time, polymerase chain reaction (PCR) testing was obtained to evaluate for other possible infectious etiologies.

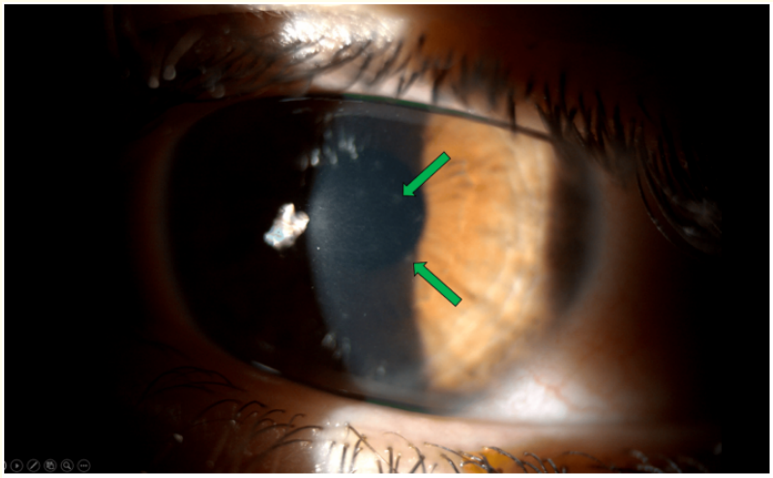

Figure 1: *An original diagnostic slit lamp photograph of the left eye of an eight-year-old boy initially presumed to have herpetic keratitis and later confirmed to have microsporidial keratitis. The green arrows demonstrate the location of subepithelial infiltrates. Informed consent to use this photograph was obtained from the patient's guardian prior to publication. Topcon's DC-4 Digital Camera Attachment was used to capture the image shown in figure 1. The image was stored on IMAGEnet 6 ophthalmic data system.*

By day 15 of treatment, BCVA had further declined to 20/100 in the left eye. The patient denied ocular pain, raising concern for developing neurotrophic keratoconjunctivitis. Examination revealed diffuse coarse punctate keratitis, diffuse moderate conjunctival injection, heavy mucus in the tear film, and a new central abrasion (presumably due to eye rubbing). The patient was started on erythromycin ophthalmic ointment with nighttime patching.

On day 16 of treatment, BCVA decreased to 20/200 in the left eye and IOP measurement was deferred due to patient tolerance and eyelid squeezing. Examination demonstrated less mucus in tear film, persistent moderate diffuse conjunctival injection, meibomian gland dysfunction, hazy corneal epithelium, and healing abrasion. At this point, PCR testing returned positive for Microsporidia (*Encephalitozoon* (*hellem*, *cuniculi*) or *Nosema* (*vittaforma*) *ocularum*) and a diagnosis of microsporidial keratoconjunctivitis was confirmed.

Although topical fumagillin and oral albendazole were considered preferred therapies, fumagillin is unavailable in the United States and oral albendazole has systemic side effects and is expensive. Given the patient's poor response to ofloxacin dosed every two hours, treatment was transitioned to compounded topical voriconazole 1% drops four times a day, and topical moxifloxacin drops every two hours. The patient was encouraged to continue patching the affected eye with erythromycin ophthalmic ointment at night.

By day 18 of treatment, BCVA had improved to 20/25-1 and IOP was stable at 16 mm Hg. Conjunctival injection and mucus continued to improve, and the corneal abrasion had completely resolved. Due to the excellent clinical response, the patient remained on topical

voriconazole 1% drops four times a day, topical moxifloxacin drops every two hours, and erythromycin 0.5% ophthalmic ointment at night.

On day 23 of treatment, BCVA declined to 20/40-2 in the left eye despite medication compliance. This decrease was attributed to mild stromal inflammation, which was presumed to be an immune-mediated response to the microsporidial infection. At this point, the corneal epithelium appeared normal. Loteprednol etabonate ophthalmic suspension 0.25% four times a day was added to the treatment regimen to address stromal inflammation.

By day 30 of treatment, BCVA had improved to 20/25 in the left eye. The patient's mother reported that ofloxacin had been inadvertently discontinued three days earlier and was subsequently restarted. Examination revealed a clear central cornea with a residual, annular stromal inflammatory ring adjacent to the limbus. All medications were tapered to twice a day for one week, then once a day for one week, and then were discontinued. At the final follow-up visit, the patient had a complete recovery and was instructed to return to his normal eyecare provider for routine follow up (Figure 2 and table 1).

Figure 2: *Flow chart summarizing clinical course and management decisions. Microsoft PowerPoint 2023 was used to create the flow chart shown in figure 2.*

Time point	Clinical findings	Management
Initial Presentation	Left eye redness, tearing, blurred vision, moderate conjunctival injection, and mucus discharge; BCVA 20/25; IOP 18 mmHg	Started on neomycin-polymyxin-dexamethasone ophthalmic solution QID
Day 4	Symptoms persisted	Discontinued neo-poly-dex; Started 0.3% topical ofloxacin q2hrs
Day 10	Left eye developed subepithelial infiltrates and superficial punctate keratitis	Continued topical therapy
Day 13	BCVA 20/40-; IOP 28 mmHg PCR testing obtained	Discontinued ofloxacin; Started oral acyclovir suspension 300 mg QID
Day 15	BCVA 20/100; Concern for developing neurotrophic keratoconjunctivitis; New central abrasion, diffuse coarse punctate keratitis, diffuse moderate conjunctival injection, heavy mucus in the tear film	Continued oral acyclovir suspension 300 mg QID; Started erythromycin ophthalmic ointment with nighttime patching.
Day 16	BCVA 20/200; IOP measurement deferred; less mucus in tear film, persistent moderate diffuse conjunctival injection, meibomian gland dysfunction, hazy corneal epithelium, and healing abrasion; PCR testing returned positive for Microsporidia (*Encephalitozoon* (*hellem*, *cuniculi*) or *Nosema* (*vittaforma*) *ocularum*)	Started topical voriconazole 1% drops four times a day and topical moxifloxacin drops every two hours; Continued nighttime patching with erythromycin ophthalmic ointment
Day 18	BCVA 20/25-1; IOP 16 mmHg; Conjunctival injection and mucus continued to improve and the corneal abrasion had completely resolved.	Continued topical voriconazole 1% drops QID, topical moxifloxacin drops q2hr, and erythromycin 0.5% ophthalmic ointment at night.
Day 23	BCVA declined to 20/40-2 with mild stromal inflammation despite medication compliance; corneal epithelium appeared normal.	Loteprednol etabonate ophthalmic suspension 0.25% QID was added
Day 30	BCVA improved to 20/25; central cornea clear with a residual, stromal annular inflammatory ring adjacent to the limbus.	All medications were tapered to twice a day for one week, then once a day for one week, and then were discontinued.

Table 1: *Chronological summary of clinical findings and management.*

Results and Discussion

This case of microsporidial keratoconjunctivitis is notable due to the absence of recognizable risk factors including: contact lens use, trauma, immunosuppression, or clear exposure history. Given that he was a young active boy, exposure to mud, water, or dirt was possible, although there was no known direct exposure.

Microsporidial keratoconjunctivitis has most frequently been treated successfully with systemic albendazole and topical fumagillin [4,6,7]. However, both medications were unavailable to our patient due to access, cost, and potential side effects. Topical fluoroquinolone monotherapy has also been reported as an effective treatment [4,8]. However, this approach was ineffective in our patient, perhaps due

to poor efficacy of fluoroquinolone monotherapy against certain microsporidia species [9]. As mentioned previously, microsporidia are obligate intracellular organisms. This means any form of treatment that targets the organism must transverse the host cell membrane. In addition, the medication must pass the triple-wall structure of the microorganism. This triple-wall structure consists of an exospore, endospore, and plasma membrane. These multiple layers of protection reduce bioavailability of therapeutics that exert their effect by inhibiting DNA replication.

In addition to reduced bioavailability of therapeutics targeting microsporidia, fluoroquinolones are known to have various levels of effect against microsporidia depending on the specific species. For example, the effects of various fluoroquinolones were tested *in vitro* and *in vivo* against *Vittaforma corneae* and *Encephalitozoon intestinalis*. Some fluoroquinolones such as moxifloxacin and ciprofloxacin failed to show an effect *in vivo* against *V. corneae*-infected athymic mice while others such as gatifloxacin, lomefloxacin, and ofloxacin were partially effective. The authors in this study suggested that the efficacy of fluoroquinolones against microsporidia may be unique to each specific species of microsporidia due to various types of topoisomerases expressed during DNA replication. Furthermore, the authors of the study highlighted that none of the fluoroquinolones that were tested completely eradicated *V. corneae*, but some fluoroquinolones did prolong survival of the infected athymic mice. Taking all of this together, fluoroquinolone monotherapy may have various efficacy against microsporidia depending on three factors: 1) the host immune response against the organism, 2), the specific fluoroquinolone used to treat microsporidia, and 3) the type of topoisomerases expressed by the organism [10].

Ultimately, the infection and its sequelae were successfully treated with a combination of compounded topical voriconazole 1%, topical moxifloxacin 0.5%, erythromycin ophthalmic ointment 0.5%, and loteprednol etabonate ophthalmic suspension 0.25%. Other reports have described successful outcomes using corneal debridement and/or pharmacologic therapy, including azithromycin, polyhexamethylene biguanide, fluconazole, albendazole, bicyclohexylammonium fumagillin, voriconazole, tacrolimus, cyclosporine, corticosteroids, and propamidine isethionate [4,5,7,8,11-14]. As microsporidial keratitis is increasingly recognized in the United States, documentation of effective alternative treatment strategies remains clinically important.

Microsporidial keratitis remains rare and may be misdiagnosed in the absence of confirmation with diagnostic testing. The differential diagnosis of microsporidial keratitis includes: dry eye disease, herpetic keratoconjunctivitis, adenoviral keratoconjunctivitis, fungal keratitis, protozoal keratitis, bacterial keratitis, Thygeson's superficial punctate keratopathy, and filamentary keratopathy [3,6,12,15]. In this case, real-time polymerase chain reaction (PCR) played a key role in early diagnosis by allowing for quantitative rapid detection of the organism. Microsporidia are difficult to grow in culture because they are strictly obligate intracellular organisms [1]. Since microsporidia require viable host cells to reproduce, identifying the organism with differential media is not effective. Furthermore, other techniques have limitations due to access to technology (i.e. *in vivo* confocal microscopy), interobserver variability (i.e. *in vivo* confocal microscopy), specificity of findings (i.e. anterior segment optical coherence tomography), and/or sensitivity of findings (i.e. impression cytology). PCR testing kits have a long shelf-life and can be kept in the clinic when diagnostic confirmation of microsporidia is required. For these reasons, PCR has become an indispensable tool to detect microsporidia.

Another distinguishing feature in this case was the immune-mediated stromal inflammation that developed as the patient began responding to therapy. A limited number of reports have described immune stromal inflammation following microsporidial keratoconjunctivitis [16,17]. Our patient experienced a decline in BCVA that was associated with mild, diffuse anterior stromal inflammation on day 23 of treatment. After one week of treatment with topical loteprednol etabonate ophthalmic suspension 0.25%, the patient experienced improved vision, resolved central stromal inflammation, and a new-onset perilimbal ring of stromal inflammation sparing the central cornea. This ring of inflammation resolved with a few more weeks of treatment.

In cases of microsporidial keratitis, ISK was well-described by Mohanty., *et al.* [16]. However, the pathogenesis of ISK remained poorly understood. Proposed mechanisms include: direct invasion of the pathogen into the stroma or an immunological response in the form of

antigen-antibody deposition. Characteristic clinical features include larger nummular lesions > 2 millimeters, disciform or ring-shaped infiltrates, a relatively short history, rapid response to topical corticosteroids, and absence of deep vascularization, anterior chamber inflammation, and elevated IOP [16].

It is important to note that stromal keratitis associated with microsporidia may represent an infectious keratitis or a post-infectious keratitis (i.e. ISK). Differentiating between the two is important to optimizing visual recovery. In our case, the patient was responding well to antimicrobial therapy when he developed stromal inflammation that caused a mild reduction in BCVA. Furthermore, upon initiation of topical corticosteroids he experienced rapid clearing of his stromal inflammation. These two observations supported an immune-mediated cause rather than persistent infectious keratitis.

Limitation of the Study

This case report has several limitations. First, the single-patient nature of this report restricts the generalizability of the findings to all patients. Furthermore, the exact species of microsporidia could not be differentiated based on PCR testing. PCR testing for our patient returned positive for Microsporidia (*Encephalitozoon* (*hellem*, *cuniculi*) or *Nosema* (*vittaforma*) *ocularum*) but did not identify the specific species. This means the therapeutics that helped eradicate the infection in our patient may not have the same level of effect against all microsporidia species.

Conclusion

In conclusion, our patient with PCR-confirmed microsporidial keratoconjunctivitis was an immunocompetent pediatric patient with no known risk factors. This case highlights the effectiveness of combination therapy with topical voriconazole and moxifloxacin for microsporidial keratoconjunctivitis that has failed monotherapy with topical ofloxacin. Furthermore, clinicians should be aware that stromal inflammation following microsporidial keratoconjunctivitis may not represent persistent infection, but instead, may represent an immune-mediated response that rapidly responds to steroids. Although several treatments for microsporidial keratitis have been proposed in the past, it would be helpful in the future for the ophthalmic community to work towards a standardized treatment algorithm to help guide management of this condition.

Conflict of Interest

There are no conflicts of interest to disclose.

Bibliography

1. Fadhilah Araniy., *et al*. "Microsporidium". StatPearls, StatPearls (2023).
2. Weber R., *et al*. "Human microsporidial infections". *Clinical Microbiology Reviews* 7.4 (1994): 426-461.
3. Singh Prabhakar and Koushik Tripathy. "Microsporidial keratitis". StatPearls, StatPearls Publishing (2024).
4. Chan Cordelia M L., *et al*. "Microsporidial keratoconjunctivitis in healthy individuals: a case series". *Ophthalmology* 110.7 (2003): 1420-1425.
5. Joseph Joveeta., *et al*. "Clinical and microbiological profile of microsporidial keratoconjunctivitis in southern India". *Ophthalmology* 113.4 (2006): 531-537.
6. Theng J., *et al*. "Microsporidial keratoconjunctivitis in a healthy contact lens wearer without human immunodeficiency virus infection". *Ophthalmology* 108.5 (2001): 976-978.

7. Lewis Noni L., *et al*. "Bilateral microsporidial keratoconjunctivitis in an immunocompetent non-contact lens wearer". *Cornea* 22.4 (2003): 374-376.

8. Ramatchandirane Balamurugan., *et al*. "Successful treatment of microsporidial keratoconjunctivitis (MKC) With a combination of topical voriconazole 1% and gatifloxacin 0.5%: A large case series of 29 patients". *Cureus* 15.11 (2023): e49247.

9. Hooper David C and George A Jacoby. "Topoisomerase inhibitors: fluoroquinolone mechanisms of action and resistance". *Cold Spring Harbor Perspectives in Medicine* 6.9 (2016): a025320.

10. Didier Elizabeth S., *et al*. "Antimicrosporidial activity of (fluoro)quinolones *in vitro* and *in vivo*". *Folia Parasitologica* 52.1-2 (2005): 173-181.

11. Zhang Yang., *et al*. "Clinical analyses of 4 cases of microsporidial keratoconjunctivitis". *Acta Parasitologica* 70.2 (2025): 71.

12. Mohanty Amrita., *et al*. "Past, present, and prospects in microsporidial keratoconjunctivitis- A review". *The Ocular Surface* 28 (2023): 364-377.

13. Ma Kevin K., *et al*. "Dual molecular diagnosis of microsporidia (*Encephalitozoon hellem*) keratoconjunctivitis in an immunocompetent adult". *Cornea* 40.2 (2021): 242-244.

14. Mohanty Amrita., *et al*. "Sequelae of microsporidial keratoconjunctivitis and its management". *Indian Journal of Ophthalmology* 69.6 (2021): 1537-1543.

15. Moshirfar Majid., *et al*. "A narrative review of microsporidial infections of the cornea". *Ophthalmology and Therapy* 9.2 (2020): 265-278.

16. Mohanty Amrita., *et al*. "Microsporidia-induced stromal keratitis: a new cause of presumed immune stromal (interstitial) keratitis". *The British Journal of Ophthalmology* 107.5 (2023): 607-613.

17. Mohanty Amrita., *et al*. "Management and outcome of microsporidia-induced stromal inflammatory keratitis - A long-term follow-up study". *Indian Journal of Ophthalmology* 73.7 (2025): 1038-1043.

Volume 17 Issue 5 July 2026