Aspergillus-induced Malignant Glaucoma

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**Purpose:** To report a case of *Aspergillus flavus*-induced keratomycotic malignant glaucoma.

**Methods:** A 48-year-old woman presented with a severe corneal ulcer. She had received topical steroids for 10 days before presentation.

**Results:** Microbiological studies revealed *A. flavus* as the offending pathogen. Nonresponsive high intraocular pressure with associated shallow anterior chamber was present. With a diagnosis of keratomycotic malignant glaucoma, the patient underwent therapeutic keratoplasty with cataract extraction with posterior chamber wash. However, there was a recurrence of malignant glaucoma in the postoperative period. Patient underwent limited pars plana vitrectomy.

**Conclusions:** Keratomycotic malignant glaucoma is a rare complication of severe fungal ulcer. We believe that the use of topical steroids in this case probably led to increased fungal penetration, with the formation of a lens–iris fungal mass and subsequent malignant glaucoma. Nonresponse to medical therapy warrants urgent surgical intervention. To the best of our knowledge, *Aspergillus*-induced keratomycotic glaucoma has never been reported previously. This report also highlights that therapeutic keratoplasty with cataract extraction alone may not be sufficient for management of such cases, and a limited pars plana vitrectomy may be needed.

**Key Words:** keratomycotic malignant glaucoma, keratitis, fungal infection

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Keratomycotic malignant glaucoma is a poorly recognized condition affecting cases with severe fungal corneal ulcer. Fungus penetrates into the anterior chamber and facilitates the formation of a lens–iris–fungal mass at the pupillary area, thereby causing the diversion of aqueous, leading to a high intraocular pressure.\(^1\) Thus far, the reported cases have been seen in association with *Fusarium* keratitis.\(^1\)–\(^3\)

We are reporting keratomycotic malignant glaucoma in a case of *Aspergillus flavus* keratitis. Also, this report highlights that lens removal and a posterior chamber wash, at the time of therapeutic keratoplasty, may not be sufficient in managing this condition, contrary to what has been previously reported.\(^1\) Limited pars plana vitrectomy to break the pathogenetic cycle is necessary, and a failure to do so may lead to the recurrence of the condition in the postoperative period, necessitating repeat surgery for management of this condition.

**CASE REPORT**

A 48-year-old woman presented with a history of pain, redness, and decreased vision of the right eye of 16-day duration. She had previously been treated with topical steroids for a period of 10 days by a local ophthalmologist. There was no history of any previous eye surgery.

On examination, the best-corrected visual acuity (BCVA) was perception of hand movement close to her face in the right eye and 6/6 in the left eye. Left eye examination was within reference limits. In the right eye, the cornea revealed a total, full-thickness infiltrate, with irregular feathery margins. Corneal scrapings were performed, which showed fungal hyphae on KOH mount, later identified as *A. flavus*. The intraocular pressure (IOP), which was digitally measured, was high, with a uniformly shallow anterior chamber. She was started on moxifloxacin 0.5% eye drops 4 times per day, natamycin 5% eye drops every hour, amphotericin 0.15% eye drops every hour, atropine 1% eye drops 3 times per day, and tablet itraconazole 100 mg daily. The high IOP did not respond to intravenous mannitol and oral tablet acetazolamide, which were given under supervision. However, no clinical response was seen, and on the third day, a large corneal central perforation was present (Fig. 1). With the diagnosis of keratomycotic malignant glaucoma, she was advised to have urgent therapeutic keratoplasty (TPK), with an extracapsular cataract extraction (ECCE) and posterior chamber wash. After preoperative mannitol, TPK with ECCE was performed. Graft size of 10.5 mm was sutured by using 16 interrupted 10/0 nylon sutures. The posterior capsule was left intact, but no intraocular lens was placed, and 2 peripheral iridotomies (PIs) were performed at 2 and 10 o’clock positions. Postoperatively, her pressures were controlled on timolol 0.5% eye drops 2 times daily, with a formed anterior chamber for the first 2 days. However, on the third postoperative day, there was a recurrence of extremely high nonresponsive IOP with a flat anterior chamber (Fig. 2). Both PIs were open. The Seidel test was negative. Ultrasound B-scan revealed fluid pockets in the vitreous cavity, suggestive of an aqueous misdirection.

She was operated on by using limited pars plana vitrectomy. The anterior chamber was also reformed with basal salt solution, and a gentle goniosynechiolysis was performed. At the last 3-month
The graft was mildly edematous, IOPs were normal, and visual acuity was 20/400.

DISCUSSION

Malignant glaucoma, secondary to fungal corneal ulcers, can result in late perforation and rupture of the globe. Fungus penetrates into the anterior chamber and facilitates the formation of a lens–iris–fungal mass at the pupillary area, thereby causing the diversion of aqueous, leading to high IOP.1–3 It is important to recognize this syndrome in cases of fungal corneal ulcer, because at this stage, the eye will be lost unless the malignant glaucoma is also managed by surgical intervention. Previously, reported cases had *Fusarium* keratitis, unlike our case, where *A. flavus* was isolated. This may either be caused by a reporting bias or because *Aspergillus* species causes a less severe and not so rapidly progressive form of keratitis, which is more amenable to therapy than *Fusarium* keratitis.3 However, the outcome of mycotic keratitis depends on the interplay of the agent (virulence, resistance to drugs, and toxicity) and host factors (predisposing factors, inflammatory response, and hypersensitivity reactions) in addition to timely diagnosis and appropriate medical treatment.4 In our case, previous steroids probably increased the virulence and progression of the keratitis with formation of lens–iris–fungal mass, thereby causing the diversion of aqueous, leading to a high IOP and resulting keratomycotic glaucoma. This pressure was nonresponsive to the maximal medical therapy including intravenous mannitol. The classic concept of malignant glaucoma has changed over the years and now includes a variety of secondary angle closure glaucomas unresponsive to the antiglaucoma measures.5 With this broader definition, fungal malignant glaucoma was first described by Jones et al2,3 in cases of mycotic keratitis. In these cases, a uniform shallowing of the anterior chamber was present, as opposed to an iris bombe and associated elevation of IOP. The case presented here had the characteristics described by Jones to make diagnoses of keratomycotic malignant glaucoma: raised tension unresponsive to standard antiglaucoma measures, uniform shallowing of the anterior chamber, and recovery of the fungus from the anterior chamber.

TPK, ECCE, and posterior chamber wash may suffice in these cases, without additional vitrectomy.1 This also has the advantage of maintaining the barrier effect by preserving the vitreous face. However, this patient had a recurrence of the malignant glaucoma in the postoperative period, which did not respond to the medical treatment. The Pls were patent, and the ultrasound B-scan revealed fluid pockets in the vitreous cavity. The patient had to be urgently reoperated on to prevent sight-related sequelae because of the high IOP, as well as for symptomatic relief. After the limited vitrectomy, the patient’s IOP was controlled, with no recurrences. This may suggest that, under these situations, TPK with ECCE might not control the underlying mechanisms at work. Limited pars plana vitrectomy may be necessary to break the pathogenic cycle, and a failure to do so may lead to the recurrence of the condition in the postoperative period, necessitating a repeat surgery for the management of this condition.

In summary, we report a unique case of keratomycotic malignant glaucoma to highlight a different fungal etiology and a recurrence of the malignant glaucoma after therapy with previously reported operative techniques.

REFERENCES