

Focal Corneal Decompensation After Filtering Surgery With Mitomycin C

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Purpose: To report a case with focal corneal decompensation after filtering surgery with inadvertent inadequate irrigation of mitomycin C (MMC).

Methods: Case report and review of literature.

Results: A 25-year-old man first referred with the complaint of photophobia. His ocular examinations revealed diffuse keratic precipitates and many iris nodules in both eyes. The primary diagnosis was idiopathic bilateral granulomatous anterior uveitis. The intraocular pressure (IOP) gradually increased in the left eye and was not controlled with a prescription of topical antiglaucoma medications. As the disease progressed, the left eye underwent filtering surgery with MMC 0.02%. The postoperative period was uneventful, and the anterior chamber was deep after surgery. The IOP was controlled without medications; however, the inferior third of the cornea was edematous because of severe endothelial dysfunction.

Conclusions: Inadequate irrigation of MMC during filtering surgery can cause focal corneal decompensation.

Key Words: corneal decompensation, filtering surgery, mitomycin C (*Cornea* 2007;26:1285–1287)

Glaucoma is a major complication of chronic uveitis and is usually recalcitrant to medical therapies. The prognosis of filtering surgery in uveitic glaucoma is guarded; routine application of antimetabolites such as mitomycin C (MMC) is recommended to prevent the fibroproliferative response that may lead to surgical failure, especially in the uveitic eye of a young patient. However, application of antimetabolites may cause early and late complications.^{1–9}

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Corneal decompensation adjacent to the filtering bleb has been reported in uveitic eyes after glaucoma implants and after filtering surgery with MMC in experimental¹⁰ and human¹¹ studies; however, focal corneal decompensation far from the site of filtering surgery has not been previously described.

CASE REPORT

A 25-year-old man first referred with the complaint of photophobia in November 1999. On his first examination, the visual acuity was 20/20 in both eyes. Slit-lamp examinations revealed white fine stellate keratic precipitates, many iris nodules (more prominent in the left eye), mild anterior-chamber reaction (cell and flare), and fleck opacities on the anterior lens capsule. We evaluated the endothelial cell count with $\times 40$ magnification with 0.20-mm² spot size light of the slit lamp and compared the pattern of the cell density with a standard endothelial cell map, which had been labeled with specific cell counts. The estimated endothelial cell counts of both eyes were 2500/mm² with no corneal guttata. The intraocular pressure (IOP) was 10 mm Hg in the right eye and 28 mm Hg in the left eye without any antiglaucoma medication. The cup-to-disc ratio was 0.2 in both eyes. The systemic workup for the possible underlying cause, including HLA-B27, HLA-B5, serum angiotensin-converting enzyme inhibitor, venereal disease research laboratory and purified protein derivative tests were negative and a chest x-ray was normal. The diagnosis was consistent with bilateral idiopathic granulomatous uveitis associated with ocular hypertension in the left eye. Timolol 0.5% was started twice daily in the left eye. The photophobia was relieved, and the IOP decreased to 11 mm Hg initially. Later, dorzolamide eyedrops 3 times a day were needed to control IOP. The patient was lost to follow-up examinations for 4 years. When he returned to the clinic in May 2003, his vision was severely impaired (counting fingers at 2 m), and the eye was painful. The visual acuity of the right eye was 20/20. The IOP was 38 mm Hg in the left eye while taking timolol twice and dorzolamide 3 times a day. The cup-to-disc ratio had progressed to 0.6 \times 0.6 in the left eye, with significant constriction of the visual field. The fundus examination of the left eye revealed macular edema with cystic changes caused by longstanding uveitis. The angle was open (grade IV according to Shaffer classification) in both eyes. There was no sign of endothelial cell dysfunction on slit-lamp biomicroscopy (estimated endothelial cell density was 2500/mm²).

Because of poor medical control, he underwent trabeculectomy augmented by MMC on his left eye in June 2003. MMC 0.02% was applied for 1 minute after dissecting the Tenon capsule before preparing the limbal-based partial-thickness scleral flap. The postoperative period was uneventful, and the patient had a deep anterior chamber postoperatively. The IOP of the left eye decreased to 8 mm Hg without antiglaucoma medications. The bleb remained functional and avascular during the postoperative period, but the inferior third of the cornea gradually became edematous and opaque. On the last

examination, in May 2004, the inferior cornea was completely decompensated with stromal and epithelial edema (Figs. 1, 2), and the corneal thickness, as measured by ultrasound pachymetry, was 720 and 550 μm in the inferior and central cornea, respectively.

DISCUSSION

Glaucoma is one of the well-known morbidities in uveitic eyes.⁶ Patients with glaucoma have borderline endothelial cell function that might be diminished by any additional insult such as adherent keratic precipitates, glaucoma implants,⁶ topical medications,^{7,8} laser iridotomy,⁹ or MMC application.^{10,11}

Corneal decompensation has been reported in patients with glaucoma after multiple glaucoma surgeries and a touch of the glaucoma implants with the cornea⁸; however, to the best of our knowledge, there has been no report of focal corneal decompensation far from the site of surgery after application of MMC.

Meitz et al¹¹ recently reported 2 cases with local decompensation of the corneal endothelium after trabeculectomy with MMC, resulting in a well-demarcated clear zone of the cornea and a second zone with thickening of the cornea and an area of bullous keratopathy adjacent to the filtering bleb. The site of corneal decompensation in this case was far from the filtering bleb.

Corneal decompensation in this patient could be related to the use of topical dorzolamide, which has been reported to cause endothelial cell dysfunction and corneal decompensation in susceptible cases.^{7,8}

Our patient had received dorzolamide for 1 year before surgery. Because topical drugs usually concentrate in the lower third of the cornea in the tear meniscus, one can consider that the inferior third of the cornea in this patient had been already exposed to the toxic effect of dorzolamide, and this medication may have a contributory role in the final endothelial decompensation. However, patients who undergo trabeculectomy have frequently received dorzolamide for several years, yet do not develop corneal decompensation.

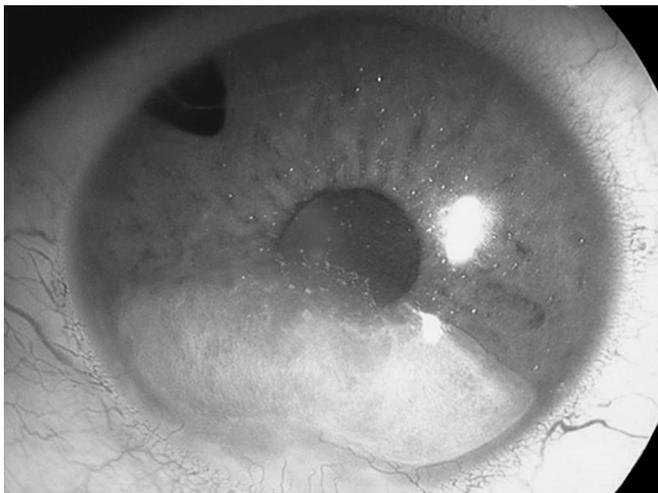


FIGURE 1. Inferior corneal decompensation compatible with area of pooling of MMC.

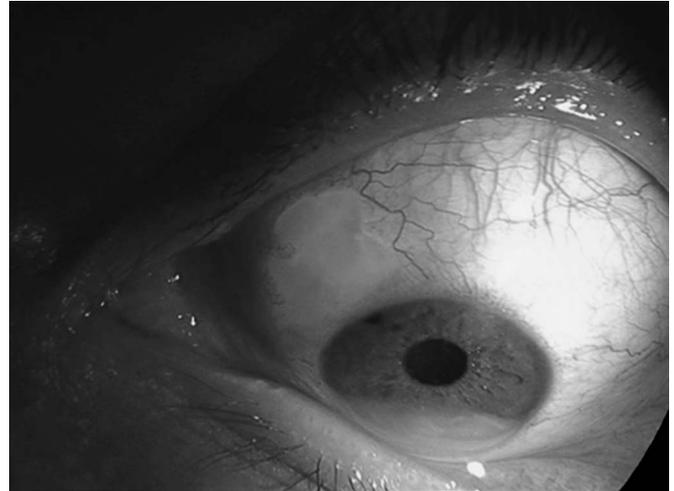


FIGURE 2. Avascular elevated functional bleb.

Corneal decompensation in our case cannot be ascribed to direct exposure of endothelial cells to MMC either; the patient neither had a period of a flat anterior chamber postoperatively nor did an inadvertent entrance of MMC into the anterior chamber occur during the operation. However, MMC has been reported to cause corneal decompensation in an experimental study, in which the transconjunctival route was used as an adjunct to conventional glaucoma filtration surgery in rabbits.¹⁰

Regarding the perfect application time and concentration of MMC, in early protocols, a sponge soaked in MMC 0.5 mg/L was usually applied to the subconjunctival tissues for 5 minutes. Subsequent attempts to reduce the risk of hypotony have included reduced concentrations and exposure times. It has also been suggested that adjustment of exposure time according to each patient's risk of excessive fibrosis may enhance the balance between successful IOP control and incidence of complications. Some retrospective studies suggested that 0.02% MMC applied for 2 minutes may be as effective as higher doses but may be associated with fewer complications.¹²⁻¹⁶

A variety of sponges have been advocated as vehicles for MMC, including Meroce1 and various microsurgical sponges, and it may be possible that manipulating the size or shape of the sponge can influence the effect of the MMC.¹⁶ One study suggested that placing the sponge beneath the scleral flap rather than over intact episclera may improve the success rate without increasing complications.¹⁵ It has also been shown in rabbits that irrigating the ocular tissues with balanced salt solution after removal of the sponge substantially reduced intraocular diffusion of MMC.¹⁶

In an experimental model, irrigation reduced the MMC concentration only in the external half of the sclera, leaving the deep intrascleral concentrations unchanged.^{17,18}

The well-demarcated inferiorly located corneal decompensation prompted the authors to explore the surgical steps. It is hypothesized that MMC was trapped between the lower eyelid and cornea while the eye was rotated inferiorly by the effect of the corneal traction suture. Routinely, we release

the traction suture after MMC application and irrigate the ocular surface with at least 50 mL of balanced salt solution. If one does not release the traction suture during irrigation, MMC may be trapped in the inferior fornix for a considerable time, just enough to cause endothelial cell damage limited to the inferior cornea; the upper limit of the well-demarcated zone coincides with the lower eyelid margin that rests on the lower cornea.

However, it would be more likely that the MMC contributing to the corneal decompensation originated from an excess amount of 0.02% MMC solution in the soaking sponge. Thus, when the sponge was placed underneath the Tenon capsule, excess 0.02% MMC solution was squeezed out of the sponge and accumulated in front of the inferior cornea, which subsequently penetrated into the cornea and led to corneal decompensation in the corresponding area.

Therefore, local corneal decompensation far from the site of surgery can complicate the postoperative course of MMC-augmented filtering procedures. MMC may be trapped between the lower eyelid and the inferior third of anterior corneal surface and in the inferior fornix while the eye is rotated inferiorly under the influence of the traction suture.

In conclusion, meticulous attention should be paid during making the intended concentration and irrigation of MMC off the ocular surface. Traction sutures should be released, and fornices should be irrigated thoroughly. Possible contributory factors to corneal decompensation in our case, including chronic uveitis and keratic precipitates, longstanding poorly controlled glaucoma, surgical manipulations, and topical application of dorzolamide, should also be considered.

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