An Implantable Collamer Lens was implanted in a 24-year-old man with a refractive error of $-5.5 - 0.5 \times 25$ in the right eye. On postoperative day 1, the acuity was hand motions. There was limbus-to-limbus stromal edema with Descemet folds, the anterior chamber was deep, and the pupil was dilated. There was thin fibrin on the collagen copolymer intraocular lens. Vault could not be assessed. The patient was treated with topical steroids, systemic steroids, and topical anti-glaucoma medications. At 3 months, the uncorrected distance visual acuity was 20/20 partial (missing letters). The eye was quiet, and the vault was good. The pupil was 4.0 mm and asymmetrical. There was diffuse iris atrophy and minute anterior subcapsular opacities. Based on the presentation and response to treatment, I believe this was a case of toxic anterior segment syndrome.

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**Complications reported with the use of the Implantable Collamer Lens are cataract and glaucoma.** In our practice, the common causes of glaucoma following implantation of this collagen copolymer intraocular lens (IOL) are retained ophthalmic viscosurgical devices and steroid response. Rarely, nonpupillary block angle closure or pupillary block glaucoma have been reported. Assessment of the vault between the collagen copolymer IOL and the natural lens is very important in the early postoperative follow-up. Long-term inflammation is not found to be associated with this surgery. I present a case in which the patient presented with corneal edema and inflammation on the first postoperative day following insertion of a collagen copolymer IOL.

**CASE REPORT**

A 24-year-old gentleman presented for refractive surgery. His systemic history was unremarkable, and he was not aware of any allergies. The refraction was $-5.5 - 0.5 \times 25$ in the right eye and $-5.75 - 0.75 \times 180$ in the left eye. The anterior segment and fundus evaluations were unremarkable. The Schirmer test showed a wetting of 30 mm in the right eye and 20 mm in the left eye without anesthesia at the end of 5 minutes. Scanning-slit topography (Orbscan, Bausch & Lomb) showed a central thickness of 420 μm and 444 μm, respectively, and keratometry readings were 43.3 @ 96/42.1 @ 6 and 43.8 @ 95/42.3 @ 3, respectively. The white-to-white diameter was 11.6 mm in the right eye and 11.8 mm in the left eye. The anterior chamber depth from the endothelium was 3.13 mm and 3.21 mm, respectively.

Two neodymium:YAG laser iridotomies were performed in the right eye at the 10 o’clock and 2 o’clock positions. A $-8.5$ diopter collagen copolymer IOL with an overall diameter of 12.0 mm and an optic diameter of 4.6 to 5.5 mm was implanted under topical anesthesia. The surgery was uneventful. Diluted pilocarpine was injected at the end of surgery to constrict the pupil.

On postoperative day 1, the uncorrected distance visual acuity (UDVA) in the right eye was hand motions. There was limbus-to-limbus stromal edema with Descemet folds. The anterior chamber was deep, and the pupil was dilated. There was thin fibrin on the collagen copolymer IOL. Vault could not be assessed. The intraocular pressure (IOP) was 28 mm Hg. Two tablets of acetazolamide 250 mg were given orally, and timolol maleate 0.5% was instilled in the eye. The IOP was 12 mm Hg. The patient was continued on prednisolone (Wysolone) eyedrops every 2 hours along with moxifloxacin (Vigamox) eyedrops 4 times a day and timolol maleate 0.5% eyedrops twice a day.

On day 4, the UDVA was counting fingers and the stromal edema remained the same. The patient was started on prednisolone 50 mgm once daily. On day 7, the edema started decreasing. Fundus details were visualized. On day 11, no corneal edema was seen. The pupil remained well dilated. Vault between the anterior surface
of the natural lens and the collagen copolymer IOL was good. Anterior subcapsular lenticular opacities were seen. The oral prednisolone was stopped. On day 14, the UDVA was 20/30 and the IOP was 11 mm Hg. The topical steroid was tapered. On day 21, pilocarpine 2% drops were given 3 times a day. The pupil became horizontally oval and measured 4.0 mm vertically and 6.0 mm horizontally. The pupil became larger after the pilocarpine was stopped.

At 5 weeks, the eye was quiet and the pupil was dilated (Figure 1). The IOP was 11 mm Hg. White localized subcapsular lenticular opacities were seen. The fundus examination was normal. The patient was happy with the vision and did not complain of glare; however, he did complain that the pupil in his right eye was larger.

At 3 months, the UDVA was 20/20 partial (missing letters). The eye was quiet, and the vault was good. The pupil was 4.0 mm and asymmetrical, larger temporally (Figure 2). There was diffuse iris atrophy, and minute anterior subcapsular opacities were seen (Figure 3).

**DISCUSSION**

Toxic anterior segment syndrome (TASS) is an acute postoperative sterile inflammatory reaction of the anterior segment tissues to a toxic substance. It can occur after any anterior segment surgery. It can result from several causes, some of which may be avoidable. Extremes of pH or osmolarity and/or the presence of preservative or detergent residue can lead to endothelial damage, cystoid macular edema, an unreactive pupil, or even glaucoma.

Patients typically present within 12 to 48 hours postoperatively, and the most common finding is a diffuse limbus-to-limbus corneal edema secondary to damage of the endothelial cell layer. Widespread breakdown of the blood-aqueous barrier is also characteristic, with fibrin formation in the anterior chamber and increased anterior chamber inflammation, often resulting in a hypopyon. In severe cases, damage to the iris and trabecular meshwork might occur, resulting in glaucoma that can be medically refractory. With intense topical corticosteroid treatment, most cases resolve over a period of weeks to months, with the cornea eventually clearing and the inflammation subsiding. However, severe cases may result in permanent sequelae, such as persistent corneal edema, iris thinning, a permanently dilated or irregular pupil, peripheral anterior synechia, and glaucoma.

I believe the patient I present was a case of TASS, as he presented with limbus-to-limbus corneal edema and inflammation in the anterior chamber on the first postoperative day. Corneal edema and anterior segment inflammation responded to steroids. The IOP remained normal in the postoperative examinations, and the vault between the ICL and the natural lens was normal. This complication of collagen copolymer IOL surgery has not been
reported in the literature, and a PubMed search revealed no references to it.

Toxic anterior segment syndrome should be suspected in any patient who presents with limbus-to-limbus corneal edema and normal vault following collagen copolymer IOL surgery. In the patient presented, intensive topical and systemic steroid therapy helped in early resolution with no serious sequelae. The pupil constricted slowly over months. Because of the clear lens, anterior lenticular opacities can happen after TASS following collagen copolymer IOL surgery.

REFERENCES