• Letter to the Editor •

Diagnosis and treatment of pupillary block glaucoma following implantable collamer lens surgery: a case report

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Dear Editor,

The latest Visian implantable collamer lens (ICL) with a central port design (Aquaport; model V4c) obviates the necessity for preoperative laser iridotomy or intraoperative iridectomy by allowing the free flow of the aqueous humor between the sides of the intraocular lens. The design of Aquaport can also eliminate the complications associated with iridotomy and iridectomy. Therefore, the V4c ICL has the potential to significantly reduce the risk of pupillary block compared to its predecessors. In this report, we present a case of acute pupillary block glaucoma secondary to obstruction of the central port of a V4c ICL by a membrane, and discuss both the etiology of pupillary block and successful management of this unforeseen complication. This report follows the tenets of the Declaration of Helsinki. The patient was informed about the purposes of the report and signed a consent form.

Case Presentation

A 28-year-old woman presented to our hospital for surgical correction of myopia. The preoperative uncorrected distance visual acuity (UDVA) was 20/500 in the right eye and 20/400 in the left eye. The corrected distance visual acuity (CDVA) was 20/20 in both eyes, with a refraction of -6.25/0.50×16° in the right eye and -5.50/0.50×166° in the left eye. Intraocular pressure (IOP) measured by non-contact tonometer (NT-510, NIDEK Co., Ltd., Japan) was 10 mm Hg (1 mm Hg=0.133 kPa) in both eyes, and the findings in the anterior segment and fundus examination were unremarkable. The depth of the anterior chamber (IOL Master 700, Carl Zeiss MeditecAG, Germany) was 4.27 and 4.23 mm in the right and left eyes respectively, and the axial length was 26.66 mm and 26.15 mm individually. The white-to-white measurement of Pentacam (Oculus Optikgerate GmbH, Wetzlar, Germany) was 12.1 mm in both eyes. The endothelial cell count measured by noncontact specular microscopy (CEM-530, NIDEK Co., Ltd., Japan) was normal, with 2931 cells/mm^2 in the right eye and 2855 cells/mm^2 in the left eye. Ultrasound biomicroscope (MD-300L, MEDA Co., Ltd. Tianjin, China) revealed open angles in both eyes.

The patient had no prior history of systemic or ocular disease and underwent thorough exclusion of surgical contraindications prior to the surgery. Preoperative topical therapy consisted of 0.5% levofloxacin and 0.1% diclofenac sodium eye drops administered four times daily for two days. The patient underwent a clear corneal V4c ICL implantation under topical anesthesia in both eyes (right eye size: -7.0 D, 13.2 mm; left eye size: -7.5 D, 13.2 mm). At the end of the surgery, 0.2 mL of 0.1% carbachol was injected into the anterior chamber. The surgery was uneventful and, during irrigation and aspiration, it appeared that all the viscoelastic was removed. Postoperatively, 20% intravenous mannitol (500 mL) and 500 mg oral acetazolamide were prescribed as routine measures to prevent high IOP caused by residual viscoelastic intraoperative fluid accumulation. Four hours after the surgery, the patient underwent a routine dressing change and eye examination. Her UDVA in both eyes was 20/25 (drug-induced small pupils), and the IOP was 13 mm Hg in the right and 15 mm Hg in

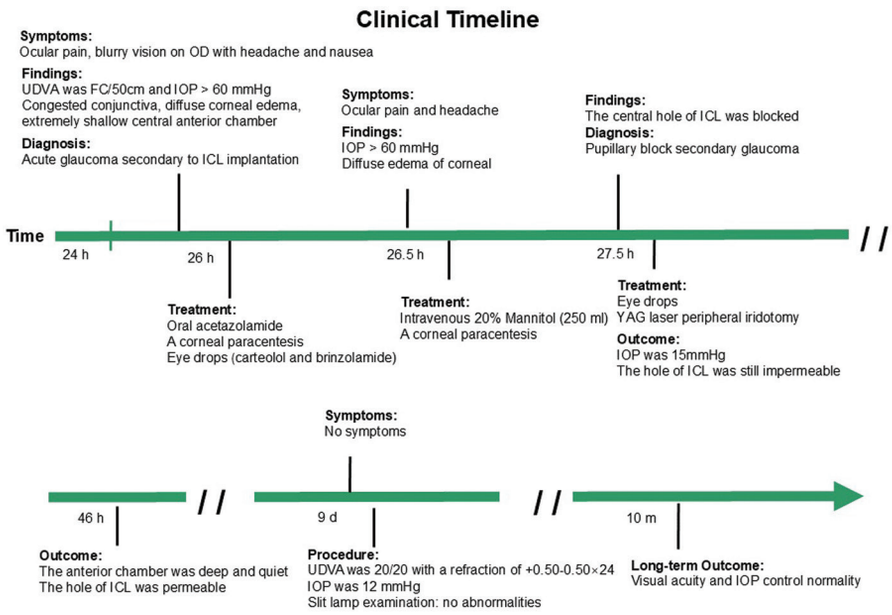

Figure 1 Clinical timeline A 28-year-old female diagnosed and treated of pupillary block glaucoma following a V4c implantable collamer lens (ICL) surgery. OD: Right eye; UDVA: Uncorrected distance visual acuity; IOP: Intraocular pressure; ICL: Implantable collamer lens; YAG: Yttrium-aluminum-garnet.

the left eye. Both eyes were prescribed topical levofloxacin 0.5%, diclofenac sodium 0.1%, and prednisolone acetate 1% four times daily. On the following day, the UDVA improved to 20/20 in both eyes, while the IOP was 12 and 13 mm Hg in the right and left eye, respectively. Both eyes showed a clear cornea, a moderately deep and quiet anterior chamber, and a round pupil (about 3.0 mm in diameter, sensitive to light reflex). The V4c ICL remained well positioned in the posterior chamber, and the lenses were clear. Therefore, the patient was allowed to be discharged with medication.

Two hours later, the patient suddenly presented with ocular pain and, blurred vision in her right eye along with headache and nausea, and so she went to the emergency department of our hospital. A clinical timeline that indicates key events is depicted in Figure 1. On examination, the UDVA was fingers counting (FC)/50 cm in her right eye and the IOP exceeded 60 mm Hg. A slit lamp examination showed congested conjunctiva, diffuse corneal edema, an extremely shallow central anterior chamber, and a narrow peripheral iridocorneal angle in the right eye. The pupil was barely visible as dilated about 6 mm in diameter. No additional eye structures were visible. As an eye emergency, the patient was immediately treated to reduce intraocular hypertension, including corneal paracentesis, acetazolamide, carteolol 2% and brinzolamide 1% eye drops. Although the symptoms relieved slightly after treatment, the patient experienced recurring ocular pain and headache half an hour later with elevated IOP exceeding 60 mm Hg. She was then administered 250 mL 20% intravenous mannitol, along with another corneal paracentesis to reduce IOP. After one hour, the IOP decreased to 28 mm Hg. With the gradual resolution of corneal edema, it was observed that the Aquaport of the ICL was blocked by a pigment membrane (Figure 2). Meanwhile, the anterior chamber was observed to be shallow, with a 2+ flare and 1+ cells in the right eye.

When asked about the history that led to her current symptoms, the patient reported accidental exposure of her right eye to bath water and delayed application of eye drops for two hours after discharge. Combined with the patient's eye examination and medical history, the possibility of infection could not be ruled out. Fortunately, on further examination, the patient's corneal incision was airtight and intact. Stoppage of levofloxacin and prednisolone acetate eye drops were ordered, while more potent moxifloxacin hydrochloride and tobramycin dexamethasone eye drops were used for anti-inflammatory and anti-infection treatment. Moreover, to prevent the IOP from rising again, the patient underwent a peripheral iridotomy of an yttrium-aluminum-garnet (YAG) laser. The right eye was anesthetized with topical proparacaine and two peripheral iridotomies were created in the temporal iris at 1 o'clock and 7 o'clock. The clinical situation improved almost immediately, showing a clear cornea, a deep anterior chamber, and a well-positioned ICL. However, the Aquaport was still impermeable (Figure 3A). The IOP had decreased to 15 mm Hg. Hence, antiglaucoma medications were discontinued. On the following day, slit lamp examination showed the Aquaport of the ICL was permeable (Figure 3B). The glaucoma resolved completely with a deep and quiet anterior chamber.

One week later, the UDVA was 20/20 with a refraction of +0.50/0.50×24° in the right eye and +0.75/0.75×157° in the

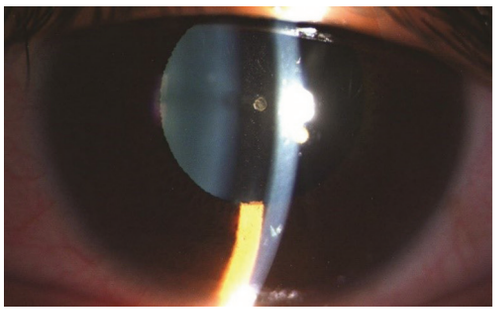

Figure 2 Slit-lamp photograph (×16 magnification) showing a blocked central aperture (Aquaport) in the right eye A pigment membrane dispersed over the anterior surface of the ICL, and flare 2+ and cells 1+ were present in the anterior chamber.

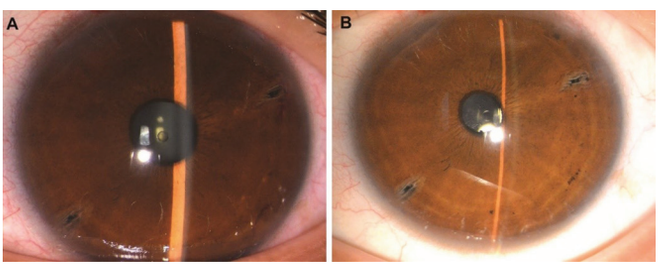

Figure 3 Slit-lamp photographs (×16 magnification) of the right eye immediately (A) and the following day (B) after YAG laser peripheral iridotomy The glaucoma has resolved completely with a deep and quiet anterior chamber. The openings made by both surgical iridectomies (at 1 o'clock and 7 o'clock) are permeable.

left eye. The IOP was 12 mm Hg in the right eye and 11.5 mm Hg in the left eye. The ICLs remained well placed in the posterior chamber, and there were no abnormalities on other examinations. Visual acuity and IOP control normality were maintained thereafter in both eyes at 10mo of follow-up. The endothelial cell count was 2861 cells/mm^2 in the right eye and 2717 cells/mm^2 in the left. The central vault measured by anterior segment optical coherence tomography (AS-OCT RTVUE XR-100, Avanti, Optovue, Fremont, USA) was 722 and 787 μm, respectively.

DISCUSSION

ICL implantation has been a popular and well-accepted refractive surgery for correcting myopia for more than 20y. For the early stage of ICL V4 model, patients should receive preoperative laser iridotomy or intraoperative iridectomy to prevent pupillary block and secondary glaucoma by allowing aqueous humor flow through the iris hole from the posterior to the anterior chamber. However, these procedures may lead to complications such as iris bleeding and glare[1], as well as increase pain and economic burden for patients. The new V4c ICL model has a 0.36 mm Aquaport in the center of the optic, which is designed to allow aqueous flow and therefore reduce the potential complications associated with iridotomy and iridectomy, without the need for prophylactic preoperative laser peripheral iridotomy. Various studies have reported the effectiveness and safety of V4c ICL in the eyes to treat myopia[1-3]. Moreover, it can significantly reduce the probability of high IOP caused by pupillary block, thus the stability of Aquaport in controlling IOP has also been confirmed[4-6].

However, elevated IOP after ICL implantation remains a significant complication that cannot be avoided. It is generally divided into three periods. In the early postoperative period, the temporary increase in IOP is attributed to the retention of viscoelastic material in aqueous humor. The second peak in elevation of IOP is likely secondary to a steroid response. In the late postoperative period, high IOP is the result of multiple factors, such as chronic long-term inflammation or chronic pigment dispersion caused by a poor match between the implanted ICL and the affected eye[7-9]. With the wide application of the V4c ICL, the pupillary block mechanism caused by Aquaport blockage has been reported. In 2017, Grover *et al*[10] first reported a case of acute pupillary block after V4c ICL implantation induced by anterior displacement of the ICL, due to Aquaport blockage with viscoelastic and inflammatory debris. The same day, the anterior chamber was washed helping in IOP control, however, the pupil remained permanently dilated and fixed as a result of acute pupillary block. Gonzalez-Lopez *et al*[11] reported a case of secondary pupil block glaucoma due to iris pigment blocking the central hole of the V4c ICL, due to excessive manipulation of the iris caused by surgical reduction when herniation of the iris occurred six days after intraocular lens implantation. The condition resolved completely after two surgical iridectomies. In 2019, Mansoori and Agraharam[12] showed a case of inflammatory debris blocking the ICL Aquaport after vitreoretinal surgery, the patient was successfully managed with topical steroids and antiglaucoma medications. In 2020, Mimouni *et al*[13] presented a case where following ICL implantation the patient developed toxic anterior segment syndrome with a subsequent pupillary block as a consequence of the occlusion of the Aquaport flow hole. More specifically, six months before ICL implantation, the patient had received two surgeries for progressive keratoconus and lens rotation. Eventually, the patient underwent ICL removal but had a persistent atonic, hypo-reflexive pupil (Urrets-Zavalia syndrome) as a complication. In 2021, Xue *et al*[14] reported a case of acute angle-closure glaucoma that occurred 4y after ICL implantation. The cause was previous uveitis and the original history of angle-open glaucoma, which caused the inflammatory membrane to block the central hole of ICL. After the treatment of YAG laser cutting the membrane combined with antiglaucoma medication, the IOP was controlled stably. However, due to the long-term high IOP, the patient had irreversible visual field defects. All of these indicate that an Aquaport hole may not be sufficient to prevent pupillary

blockage in cases with increased postoperative inflammation.

In our case, the patient had a normal IOP, clear corneal, and anterior chamber within 24h after ICL implantation. Given the sudden appearance of diffuse corneal edema, an extremely shallow anterior chamber, a dilated, fixed pupil, and elevated IOP after 24h, acute glaucoma secondary to an ICL was first diagnosed. As an ophthalmological emergency, a series of intraocular and systemic antihypertensive methods were administered immediately. Upon alleviation of corneal edema, a pigment membrane was observed on the anterior surface of the V4c ICL, blocking the Aquaport opening and causing acute pupillary block due to disruption of normal aqueous humor circulation. The causes could be excluded from improper ICL sizing since we did not find excessive anterior vaulting of the lens on the slit lamp examination, this was also confirmed by the central vault measurement at the 10-month follow-up. For the early elevation of the postoperative IOP, we first attributed the retention of viscoelastic in the anterior chamber. But it was not resolved with multiple paracenteses or various of antiglaucoma medications, which contradicts most studies[8-9]. Given the medical history, acute inflammatory infection was suspected caused by bathwater splash or omission of postoperative drops. However, there was no prolonged inflammatory response that would be typical of infection to confirm this. Given such a short postoperative time frame of the crisis and relatively mild anterior chamber reaction, it was more likely that small iris fragments released during surgical maneuvers, along with increased inflammatory factors and fibrin present in the aqueous humor caused by sterile postoperative inflammation, were responsible. Although we could not identify the exact risk factors, adhesion between them over the ICL causing the blockage of Aquaport might provide a convincing explanation. In such cases, treatment with topical eye drops might take a long time to show effectiveness, and increased the risk of recurrence of elevated IOP, potentially resulting in significant compromise to the optic nerves. Therefore, more effective methods for lowering IOP should be implemented.

Considering that a direct YAG laser over occluded Aquaport could increase the risk of damage to the ICL and crystalline lens, while surgical clearance could increase postoperative inflammation, it was prudent to constrict the pupil with pilocarpine and perform a YAG laser iridotomy, which could ensure dynamic regular aqueous flow between the posterior and anterior chambers and accelerate the absorption of the inflammatory membrane and particles of the iris. Fortunately, the patient was successfully managed with this treatment.

In summary, the complication of pupillary block glaucoma secondary to central hole blockage after implantation of the V4c ICL is extremely rare, and should be considered the complexity of ophthalmopathy as well as excessive procedures. Without prompt diagnosis and treatment of acute elevated IOP, glaucomatous optic nerve damage and irreversible vision loss can occur. Ophthalmologists should pay great attention to similar clinical manifestations, which should be identified from other mechanisms of elevated IOP. The purpose of this report is to illustrate that prompt anti-inflammation and antiglaucoma treatment combined with YAG laser iridotomy may be a simple and feasible method to resolve acute pupillary block glaucoma secondary to obstruction of the Aquaport of the V4c ICL.

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