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CASE REPORT

Suspected Cefuroxime-Induced Cystoid Macular Edema Following Non-Toric Implantable Collamer Lens Implantation: A Case Report

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Purpose: We present a rare case of suspected cefuroxime-induced cystoid macular edema (CME) subsequent to non-toric implantable collamer lens (ICL) surgery.

Case Presentation: A 23-year-old lady with high myopia underwent refractive ICL implantation in both eyes. On the first day, the right eye was performed ICL implantation first, and postoperative uncorrected distance visual acuity (UDVA) was improved to 20/20. On the following day, the left eye underwent ICL implantation. Surprisingly, postoperative UDVA dropped to 20/63 with no improvement upon correction. At the end of each surgery, 0.1 mL of cefuroxime (1.5 mg/0.1 mL) will be injected into the anterior chamber. Scanning laser ophthalmoscopy (SLO) revealed macular edema in the left eye, followed by optical coherence tomography (OCT) confirming CME and serous retinal detachment. With the treatment of pranoprofen and prednisone, the CME decreased significantly and UDVA was increasing on the fifth postoperative day. At six-month follow up, the CME had completely subsided and UDVA reached to 20/20.

Conclusion: Acute CME after ICL implantation, anatomically and temporally aligned with intracameral cefuroxime injection and lacking other inflammatory or vascular triggers, strongly points to intracameral cefuroxime ocular toxic syndrome (ICOTS). Ophthalmologists should be aware that suboptimal visual recovery after ICL surgery may indicate CME and must attend to the dose safety of intracameral cefuroxime.

Keywords: implantable collamer lens, cystoid macular edema, intracameral cefuroxime ocular toxic syndrome

Introduction

Cystoid macular edema (CME) is more commonly described following cataract surgery, with a reported incidence of 0.1%–2.35%.[1] However, CME after implantable collamer lens (ICL) implantation are infrequent. Notably, one of the few medical reports complications was about CME occurring after the implantation of a toric ICL,[2] whereas in our case, it occurred after the implantation of a non-toric ICL.

Case Presentation

A 23-year-old lady with high myopia presented for evaluation of refractive surgery. She was in good heath and with no history of hypertension, diabetes, or any other ocular history, except for ptosis surgery three years ago and allergic to penicillin. Additionally, she had been wearing frame glasses for 14 years and contact soft lenses for 5 years. Her best-corrected visual acuity (BCVA) in the right eye was 20/20 with –8.75–0.75 × 40, and 20/20 with –9.75–0.75 × 135 in the left eye. Compared with the previous data, her refractive error and ocular examination were stable over the past two years. Due to her large myopia and relatively thin corneal thickness, the current popular corneal refractive surgeries such as SMILE and FS-LASIK were not suitable for her. Moreover, she accepted no additional astigmatism, so we considered implanting ICL (EVO Visian ICL; STAAR Surgical, Switzerland) for her.

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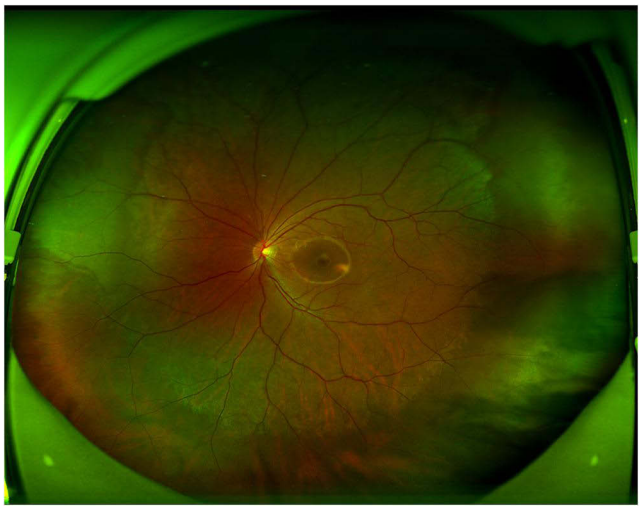

Figure 1 The SLO photography of the left eye revealed macular edema.

Under topical anesthesia, the patient first underwent implantation of an ICL (crystal labeling data −10.00D VICMO12.6 SN:S868865) in the right eye. Intraoperatively, 0.1 mL of cefuroxime (1.5 mg/0.1 mL) was injected into the anterior chamber for endophthalmitis prophylaxis. UDVA in the right eye was 20/20 on the first post-operative day. One day later, an ICL (crystal labeling data −11.00D VICMO12.6 SN:S858308) was implanted in the left eye under identical anesthesia and antibiotic prophylaxis. Post-operative UDVA in the left eye was 20/63 and did not improve with manifest refraction. The intraocular pressure is within the normal range, with 16 mmHg in the right eye and 17 mmHg in the left eye. Slit-lamp biomicroscopy revealed no wound leakage, a quiet anterior chamber, and well-centered ICLs with ideal vaulting in both eyes. Vitreous was clear without evident opacities. However, the scanning laser ophthalmoscopy (SLO) photography of the left eye disclosed macular edema (Figure 1), and subsequent optical coherence tomography (OCT) confirmed the existence of significant CME with extensive serous retinal detachment, along with an increase in macular retinal thickness to 778 μm (Figure 2A).

Our patient was treated with 0.1% topical pranoprofen in the left eye six times per day for seven days and take 30 mg oforal prednisone daily for three days. We followed up the patient several times. On the fifth postoperative day, UDVA in the left eye was improving to 20/50, and CME decreased significantly and macular retinal thickness decreased to 238 μm (Figure 2B). Six months post-surgery, CME had vanished completely, the retinal structure reverted to normal, and the macular retinal thickness reached 220μm (Figure 2C), and the patient's UDVA in the left eye had improved to 20/20.

Discussion

CME is a rare complication after ICL surgery, and it can cause vision loss and visual distortion. Although its etiology has not been clearly defined, macular traction and postoperative inflammatory processes have been proposed as causative agents.[3,4] There are also reports showing various concentrations of cefuroxime can cause acute CME.[5] According to our knowledge, this is the first report of CME after non-toric ICL implantation.

After reviewing the available literature, we found limited reports on CME after ICL implantation. Julide Canan et al reported a case of CME after toric ICL implantation: visual acuity failed to improve on the first postoperative day, a 10° rotation of the ICL was noted and secondary repositioning was performed, yet vision remained unchanged; one week later the patient returned with further visual decline, and OCT confirmed CME.[2] They proposed that constant friction between the posterior iris surface and the phakic lens or between the haptic and the ciliary sulcus, as well as traction on vitreous, could be causing CME.[2] In our case, ICL was ideally located. Despite the presence of incomplete posterior vitreous detachment, we believe it is unlikely to cause acute CME. Numerous studies have highlighted the role of inflammation in Postoperative CME.[1,4,6] However, neither obviours anterior segment inflammation nor significant vitreous cells or flare was observed in this case, rendering an inflammatory etiology unlikely. Moreover, the potential retinal toxicity of cefuroxime should not be

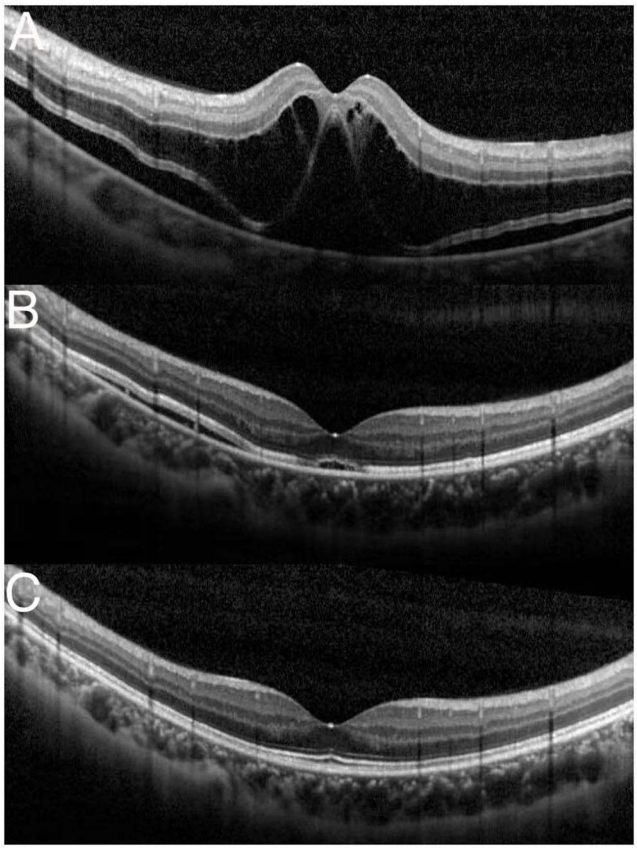

Figure 2 (**A**) One day after surgery,the OCT image showed significant CME with extensive serous retinal detachment. (**B**) On the fifth postoperative day, the CME had resolved, but subretinal fluid persists. (**C**) At the 6-month follow-up, the subretinal fluid has completely resolved.

underestimated; The presentation in this case closely resembles "Intracameral cefuroxime ocular toxic syndrome (ICOTS)": it is characterized by acute, extensive, and serous retinal detachment with significant CME that usually resolves spontaneously.[7] Reports exist of CME and serous retinal detachment appearing as early as postoperative day 1 after intracameral injection of either the standard dose (1.0mg/0.1mL) or a high dose (9 mg/0.1 mL) of cefuroxime during cataract surgery, with spontaneous resolution in the vast majority of patients within 1–2 weeks.[7,8] Studies have indicated that prior vitrectomy is a susceptible factor for ICOTS, presumably due to disruption of the intraocular barrier.[7,9] However, Zuo et al reported that ICOTS can still occur despite intracameral cefuroxime at standard concentration and an anatomically intact vitreous.[10] The cause might be related to transient retinal pigment epithelium sodium–potassium pump dysfunction resulting from a large injection volume of a standard dose concentration or individual differences in conventional drug-dose tolerance.[10] Given this case of the clinical picture characterized by immediate postoperative onset and rapid resolution by day 5, we strongly suspect ICOTS as the cause of acute CME with serous retinal detachment in the left eye. Although the operative record indicates that both eyes received an intracameral cefuroxime dose of 0.15mg/0.1mL, only the left eye subsequently developed CME with serous retinal detachment. This disparity raises the suspicion that a compounding error may have led to an inadvertently elevated drug concentration in the left eye, thereby triggering CME. Additionally, Despite the patient's documented penicillin allergy, cefuroxime's unique R1 side-chain confers a negligible cross-reactivity rate; accordingly, penicillin hypersensitivity is unlikely to compromise tolerance to intracameral cefuroxime.[11]

Fluorescein fundus angiography can indirectly delineates the pathophysiology of macular edema by revealing patterns of vascular leakage and barrier disruption,[1] regrettably, the patient declined the examination at that time. Currently, there is no standard treatment for postoperative CME, and the natural history of postoperative CME indicates that most patients do not require treatment, but treatment may help vision recovery in early CME. In clinical practice, Steroids or nonsteroidal anti-inflammatory drugs may be effective in alleviating CME.[12]

Conclusion

Acute CME after ICL implantation, anatomically and temporally aligned with intracameral cefuroxime injection and lacking other inflammatory or vascular triggers, strongly points to ICOTS. Whether its eventual resolution reflects the natural history or a treatment-driven response remains to be clarified by controlled studies. Therefore, ophthalmologists should be aware that suboptimal visual recovery after ICL surgery may indicate CME and must attend to the dose safety of intracameral cefuroxime.

Abbreviations

ICL, implantable collamer lens; UDVA, uncorrected distance visual acuity; SLO,Scanning laser ophthalmoscopy; OCT, optical coherence tomography; CME, cystoid macular edema; BCVA, best corrected visual acuity; ICOTS, Intracameral cefuroxime ocular toxic syndrome.

Ethics and Consent Statements

Institutional Approval from Aier Eye Hospital of Jinan University was taken to publish the case report. The patient has provided informed consent for the use of this case report and associated images in scientific research, and consent for publication of this case report has been obtained from the patient.

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Disclosure

The authors report no conflicts of interest in this work.

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