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Cefuroxime-Induced Toxic Maculopathy: A Cataract Co-Management Conundrum

Abstract

Background: Cefuroxime-induced toxic maculopathy can occur when an incorrect dilution is injected intracamerally after cataract surgery. This results in a guarded visual prognosis. Cefuroxime-induced toxic maculopathy can also occur sporadically even when the medication dilution is the standard correct dosage. These patients usually recover their vision by the first postoperative week.

Case Report: A 72-year-old Caucasian female developed cefuroxime-induced toxic maculopathy after uneventful cataract surgery. Investigation of the case confirmed that she had a sporadic response to the cefuroxime. It was verified that she received the standard correct dilution of cefuroxime intracamerally. The patient showed a significant improvement in her vision by her first postoperative weekly visit. By her last postoperative visit at 7 weeks her best corrected visual acuity was 20/20.

Conclusion: Optometrists should be aware of cefuroxime-induced toxic maculopathy and distinguish between sporadic cases and those where the patient received an incorrect medication dilution dosage.

Keywords

intracameral cefuroxime, toxic maculopathy, cataract surgery, co-management

Cover Page Footnote

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INTRODUCTION

Up to 78% of optometrists co-manage patients who have had cataract surgery.[1] In a recent co-management survey, 93% of optometrists who responded said that they participate in some form of co-management at least once a month[2] and, 80% of these optometrists reported that the number of patients they co-manage has been increasing during the past few years.[2] A number of studies have confirmed that optometrists provide excellent postoperative care when seeing the post-cataract surgery patient within 24 to 36 hours after their surgery.[3,4] This next day post-cataract surgery follow-up by community optometrists provides the advantages of care closer to home and avoids unnecessary hospital visits for patients after undergoing uncomplicated cataract surgery.[4]

Co-managing optometrists are well-versed in identifying the typical next day after cataract surgery postoperative findings and complications. The typical postoperative findings may include pain from corneal abrasion, corneal edema and Descemet's folds, raised intraocular pressure and mild anterior chamber inflammation.[5] More common and worrisome next postoperative day complications may include surgical wound leak, iris prolapse, vitreous prolapse, retained lens material, intraocular lens displacement and hypopyon/endophthalmitis.[5]

To try to prevent postoperative infections many ophthalmologists will inject an antibiotic into the anterior chamber (intracamerally) at the conclusion of the cataract surgery.[6] Cefuroxime is a commonly-used intracamerally injected antibiotic that is well tolerated at the recommended dose of 0.1mL (1mg/0.1 mL).[7,8] It is a second-generation cephalosporin and is considered one of the most cost-effective prophylactic therapies in helping prevent postoperative endophthalmitis.[9] It has been recommended that intracameral cefuroxime be routinely used in modern phacoemulsification cataract surgery.[10] Various anterior and posterior segment ocular adverse effects have been reported with the inadvertent intracameral injection of a higher than recommended dose of cefuroxime.[11-16] However, because of individual patient differences in conventional drug dose tolerance, some surgical cases may not have an adverse ocular event even with a higher than recommended dose of intracameral cefuroxime injection.[17] On the other hand, some patients will develop adverse ocular events with use of the recommended intracameral dose of cefuroxime.[18-20]

Co-managing optometrists should know if the cataract surgeon uses intracameral cefuroxime at the conclusion of their surgical cataract cases. Co-managing

optometrists need to be aware of the ocular adverse events that the patient may experience from cefuroxime.

CASE REPORT

A 72-year-old Caucasian female was referred for cataract surgery evaluation by her optometrist. The patient complained of decreased and cloudy vision. She felt her depth perception was decreased and she needed more light to read. Her vision problems started in both eyes two years earlier and had slowly progressed to the point where these visual symptoms were constant throughout the day. She had tried wearing her glasses more often, changing her glasses prescription, and using her artificial tears more frequently, but only obtained minimal improvement.

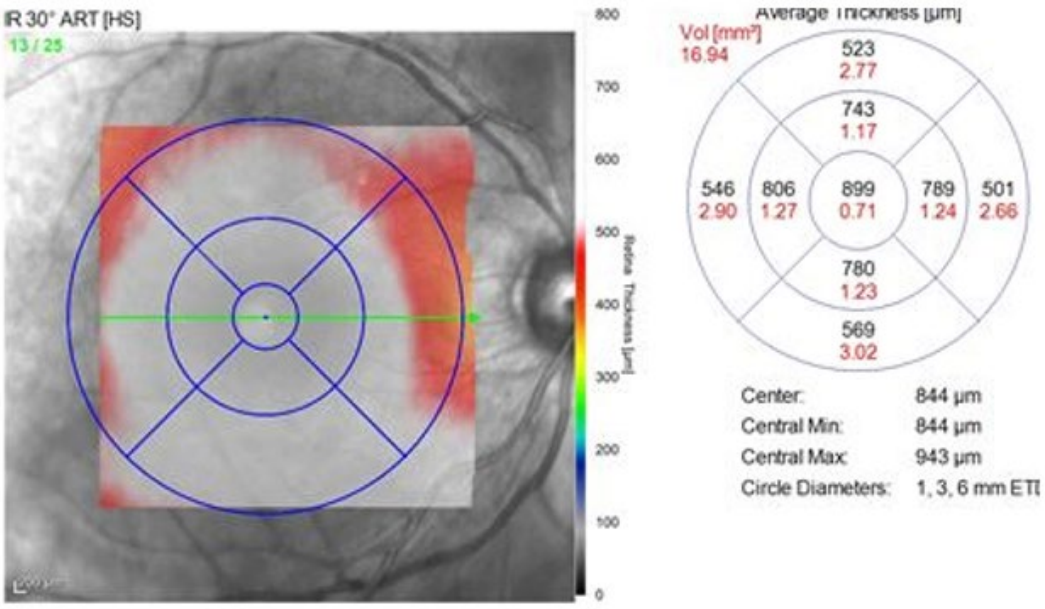
Her medical history was unremarkable, and she was on no medication. She only used topical tear supplements intermittently but was never diagnosed with dry eye syndrome. She had no medication allergies.

Her best-corrected visual acuities (BCVA) were $20/40^{+1}$ in the right eye and $20/40^{+2}$ in the left eye. Pinhole improved her vision to 20/25, right eye and $20/25^{+1}$, left eye. Glare testing using the Brightness Acuity Test measured at the medium setting was 20/60, right eye and 20/60, left eye. With the high setting, her visual acuity decreased to 20/125, right eye and 20/100, left eye. Her pupils, motility and confrontation visual fields were all normal. Her intraocular pressures using an ICARE tonometer were 16 mm Hg, right eye, and 15 mm Hg, left eye. The slit lamp exam was unremarkable except for 1+ nuclear sclerotic and 1+ posterior subcapsular cataracts in both eyes, with lenticular cortical spokes encroaching into the visual axis in both eyes. Her dilated fundus exam was unremarkable in both eyes. The optic nerves appeared healthy, and the maculae were flat and without edema. No other macular changes were appreciated.

The patient was scheduled for cataract surgery in the right eye and was instructed to start moxifloxacin 0.5% and ketorolac 0.5%, one drop each medication, four-times-a-day in the right eye, three days before the cataract surgery. The patient underwent uncomplicated small-incision phacoemulsification surgery in her right eye. At the conclusion of the surgery a 0.1mL (1mg/0.1mL) intracameral injection of cefuroxime was administered into the anterior chamber of her right eye. Postoperatively she was placed on prednisolone acetate 1%, moxifloxacin 0.5% and ketorolac 0.5%, one drop each medication, four-times-a-day to her right eye.

The patient was seen by the ophthalmic surgeon for her first postoperative visit the next day after surgery. The patient felt her vision was blurry, but she

experienced no eye pain. Her uncorrected vision in the right eye was 20/125. Pinhole showed minimal improvement to 20/100. With refraction the patient still only could read 20/125 at distance. Her intraocular pressure by ICARE tonometry was 22 mm Hg in the right eye. Slit lamp exam showed trace diffuse corneal edema with no Descemet's folds. Seidel sign was negative. There was a trace cell in the anterior chamber. The intraocular lens was well positioned, and the posterior capsule was clear and intact. Funduscopy confirmed a healthy appearing optic disc and an unremarkable retina except for the macula. The macula appeared edematous, and the patient felt she saw a "fat spot" in the middle of the vertical line of light on Watzke-Allen testing. An optical coherence tomography (OCT) of the macula confirmed cystoid macular edema with an extensive serous neurosensory macular retinal detachment (Figure 1).



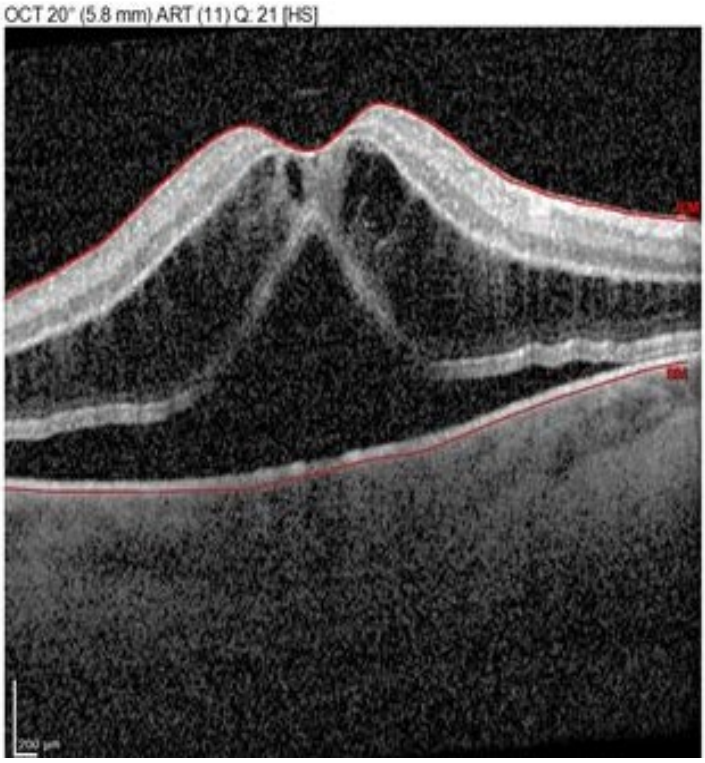


Figure 1: Optical coherence tomography of macula, 1st postoperative day: Cystoid macular edema with serous neurosensory macular retinal detachment.

The patient was instructed to increase her prednisolone acetate 1% to one drop every two hours while awake in her right eye. She was also instructed to continue both her moxifloxacin and ketorolac as before. She was referred for a retina consultation.

The patient was seen the following day by the retina specialist who repeated the macular OCT and obtained the same results (Figure 2).

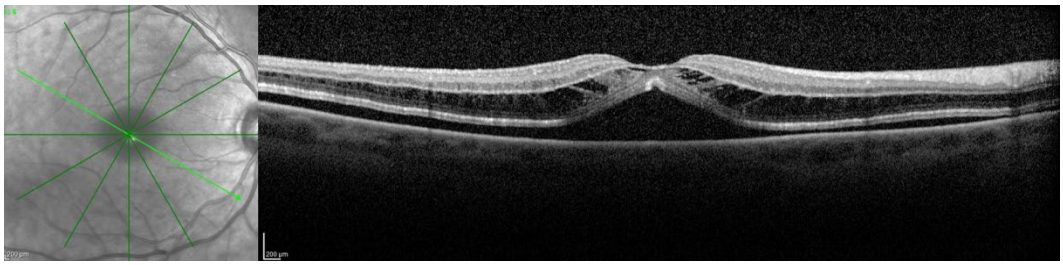


Figure 2: Optical coherence tomography of macula, 2nd postoperative day: Persistent cystoid macular edema with serous neurosensory macular retinal detachment.

The retina specialist also did an ultra-widefield photograph (Figure 3) and fluorescein angiography (Figures 4A, 4B, 4C) which confirmed no leakage.

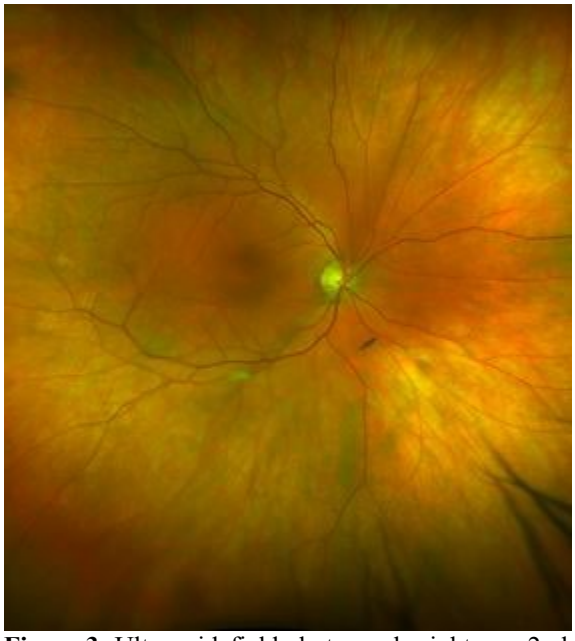


Figure 3: Ultra-widefield photograph, right eye, 2nd postoperative day: No macular exudates or hemorrhages

He diagnosed suspected cefuroxime-induced toxic maculopathy. The lack of leakage on fluorescein angiography is a distinguishing feature of cefuroxime-induced toxic maculopathy. He requested that the patient continue her eye drop medication as previously directed.

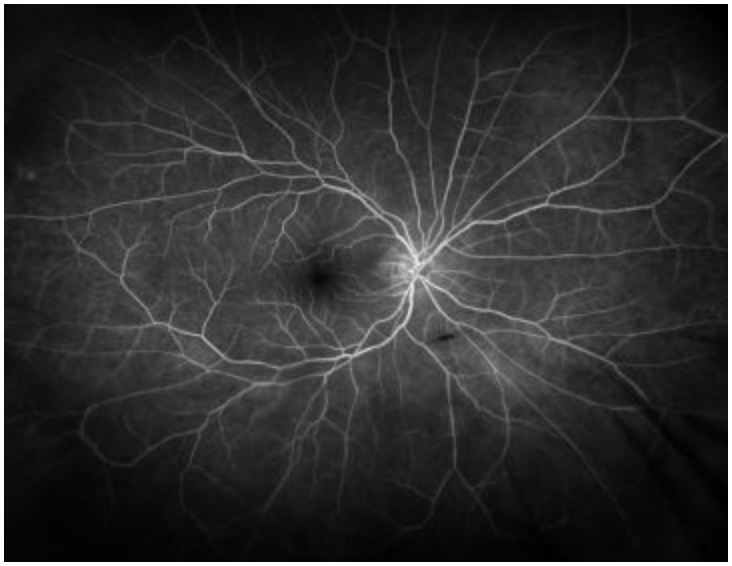


Figure 4A: Ultra-widefield fluorescein angiogram, early transit-phase, right eye, 2nd postoperative day: No macular leakage.

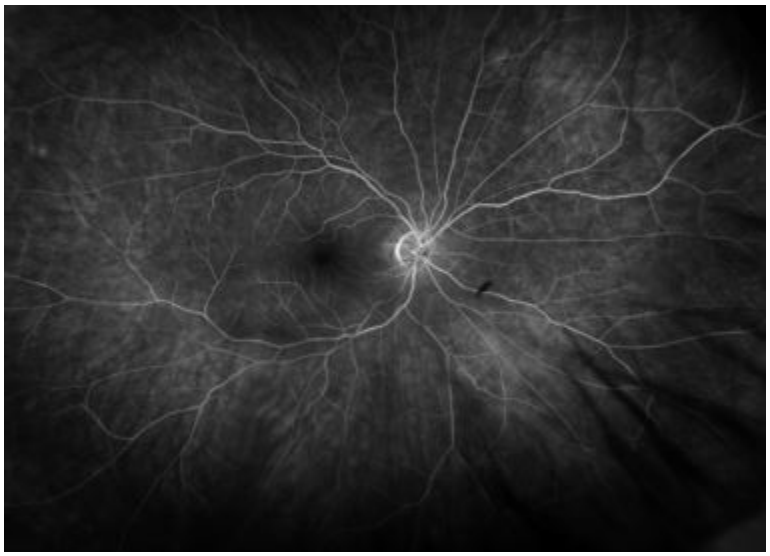


Figure 4B: Ultra-widefield fluorescein angiogram, mid-phase, right eye, 2nd postoperative day: No macular leakage.

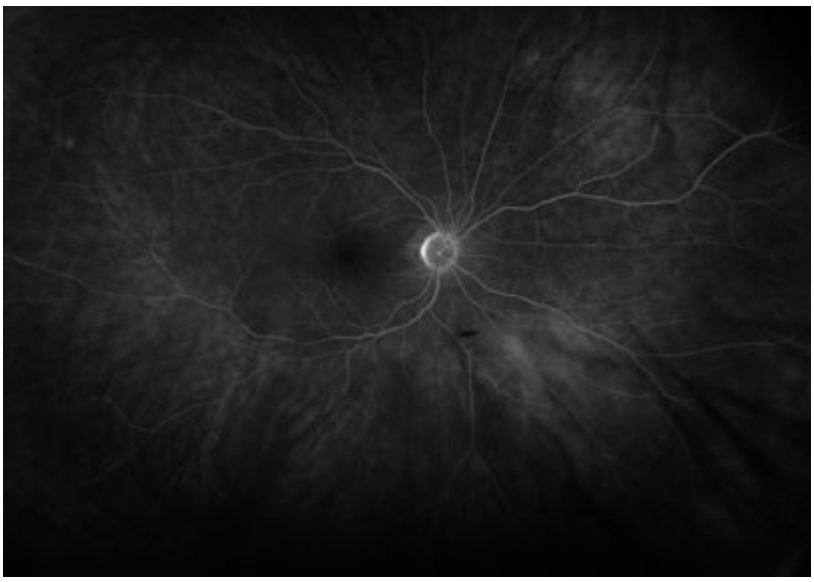


Figure 4C: Ultra-widefield fluorescein angiogram, late-phase, right eye, 2nd postoperative day: No macular leakage.

The patient was seen back at the original eye clinic one week after her first postoperative visit. The patient complained of seeing a red ring around her vision with the right eye and also experiencing shadowing behind objects. Overall, she felt her vision had improved enough that she was now able to work on her computer.

At this one week visit, her right eye uncorrected vision was $20/30^{+2}$ with no improvement on pinhole testing. BCVA after refraction was 20/25. Slit lamp evaluation showed resolution of all corneal edema and only a rare cell in the anterior chamber. The fundus examination showed less macular edema with a negative Watzke-Allen test. An OCT of the macula confirmed resolution of the cystoid macular edema and a greatly reduced macular retinal detachment with minimal subfoveal fluid (Figure 5 and Figure 6).

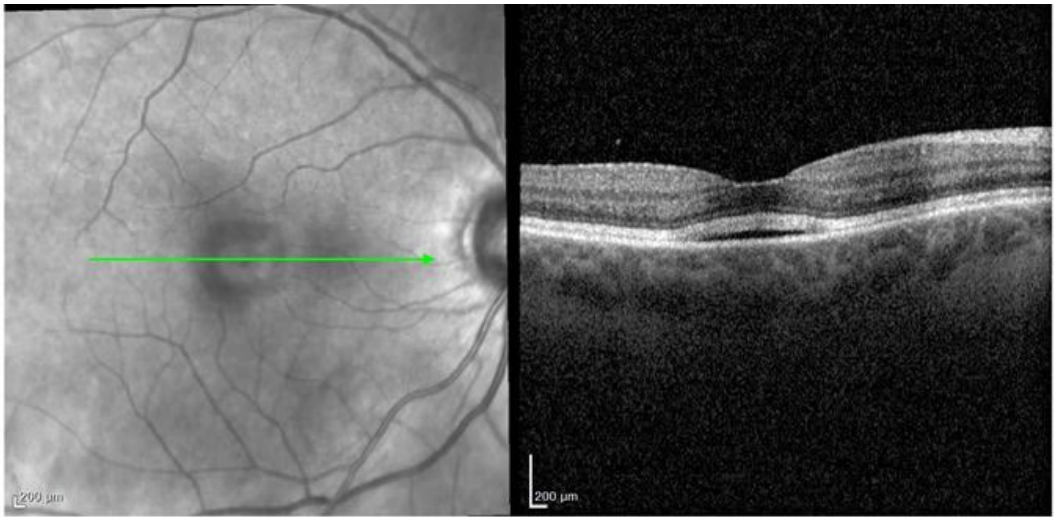


Figure 5: Optical coherence tomography of macula, 1st postoperative week: Cystoid macular edema resolution and reduced macular retinal detachment with minimal subfoveal fluid.

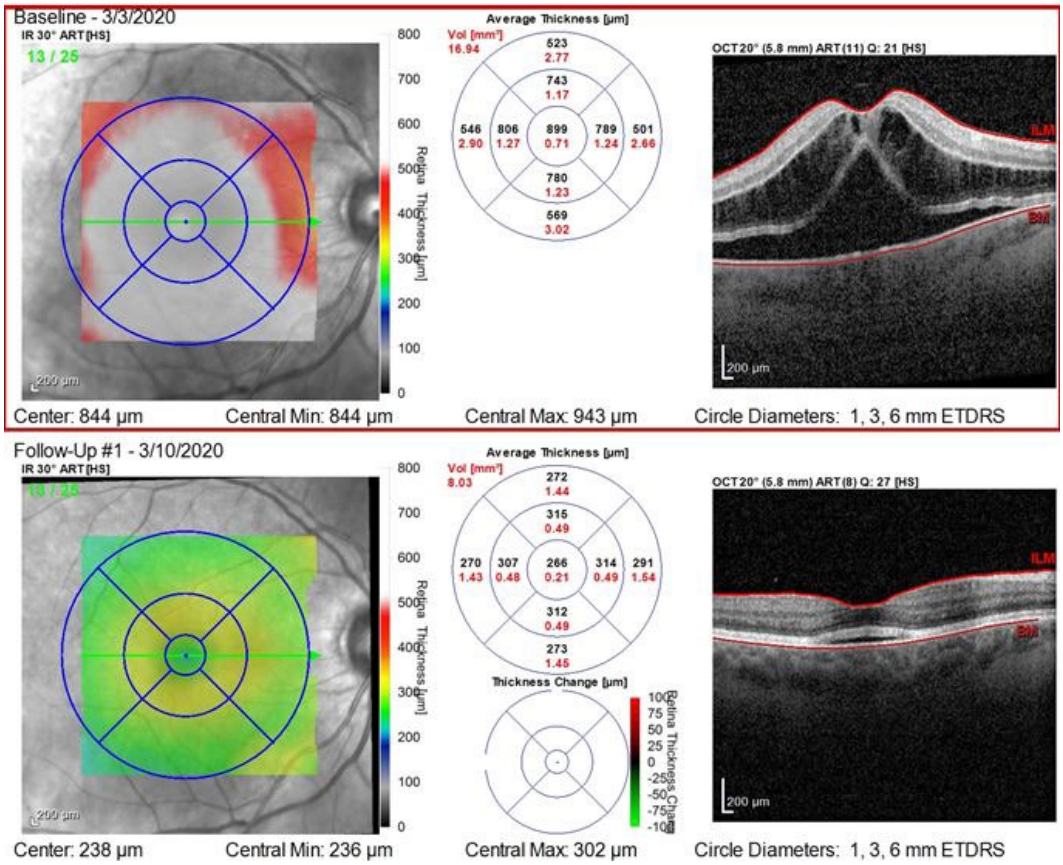


Figure 6: Comparison of optical coherence tomography. Top image: 1st postoperative day. Bottom image: 1st postoperative week.

The patient was instructed to taper the prednisolone acetate 1% over the next four weeks, to discontinue the other postoperative eye drop medication, and to restart her artificial tears as necessary.

The patient was seen for her final postoperative visit seven weeks after her first postoperative visit. At this visit the patient felt her vision had improved. She no longer saw red color distortion or any shadowing. She was only using artificial tears occasionally.

Her uncorrected visual acuity was $20/20^{-2}$ in the right eye. After refraction she could see 20/20 at both distance and near. Her slit lamp findings were unremarkable as were her macular findings on ophthalmoscopy (Figure 7).

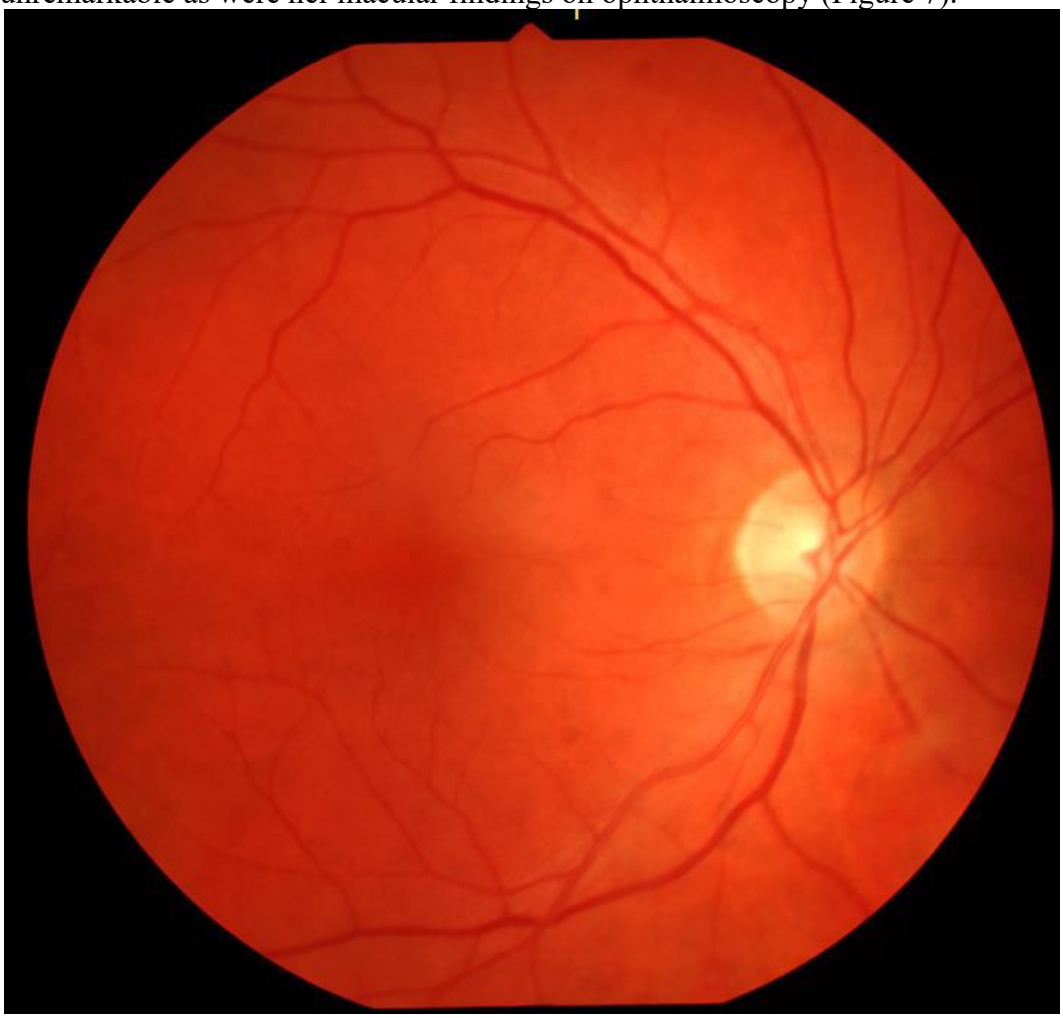


Figure 7: Fundus photograph, right eye, 7 weeks postoperatively. Normal disc and macula.

OCT of the macula showed no cystoid macular edema or subretinal fluid with normal foveal configuration (Figure 8).

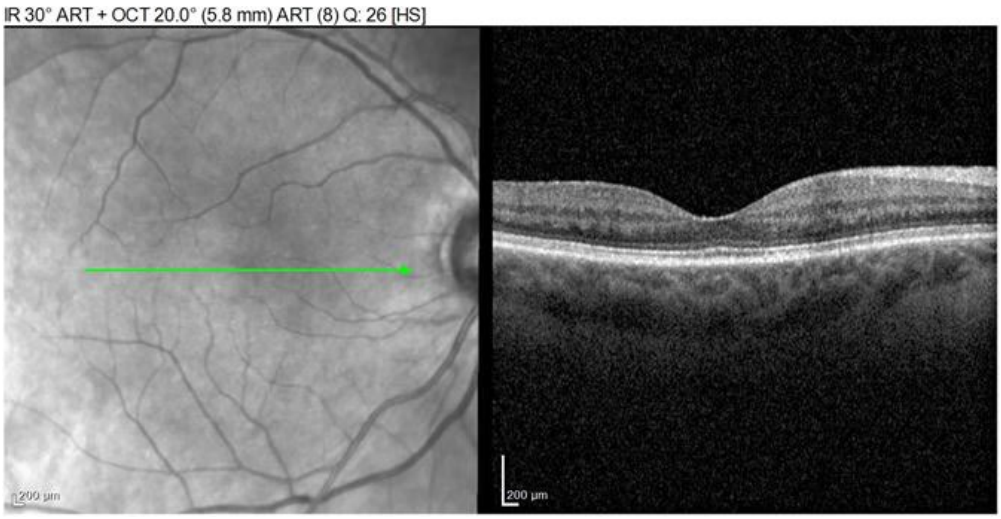


Figure 8: Optical coherence tomography of macula, 7 weeks postoperatively. No cystoid macular edema, no subretinal fluid with normal foveal configuration.

DISCUSSION

Cefuroxime-induced ocular adverse effects are rare complications most commonly seen when a drug dilution error occurs. Since cefuroxime is not available as an FDA-approved ophthalmic medication, it has to be used off-label and, therefore, needs to be diluted appropriately by the hospital pharmacy or the ophthalmic surgeon. This can lead to dilution errors which may result in anterior segment inflammation. This anterior segment inflammation can cause corneal edema and endothelial cell loss due to direct damage of the endothelial cells.[13,14] This patient had minimal corneal edema and no Descemet's folds on her first postoperative day. The amount of corneal edema the patient presented with can typically be seen on the first postoperative day after uneventful cataract surgery. Also, this patient's corneal edema resolved quickly. In addition, the patient had a typical postoperative anterior chamber inflammatory reaction. This, too, resolved quickly, albeit on a more intense prednisolone acetate 1% regimen. Color vision alteration has been previously recorded in patients from intracamerally injected cefuroxime.[14] This patient had a red color distortion initially which resolved on its own uneventfully.

Posterior segment cefuroxime-induced adverse effects include vitritis, retinal pigment epithelial changes, cystoid macular edema, serous macular retinal detachment and macular infarction.[11-16] A review of the literature indicates that

cystoid macular edema and serous macular retinal detachment are the most commonly occurring posterior segment adverse effects.[14,19] It has been noted that cefuroxime has a predilection for photoreceptors and retinal pigment epithelium.[11-14,17] The cystoid macular edema usually presents with severe outer nuclear layer edema with an accompanying serous retinal detachment in the posterior pole.[19] Previous fluorescein and indocyanine green studies of patients with cefuroxime-induced toxic maculopathy have shown no retinal or choroidal hyperpermeability or retinal pigment epithelial leakage.[11,20] This patient also had an ultra-widefield fluorescein angiogram performed on her second post-operative day and it showed no leakage. Therefore, it has been hypothesized that there is no organic damage to the inner or outer retinal barriers in these patients. Instead, the marked accumulation of subretinal and inner retinal fluid right after cataract surgery indicates impairment of the retinal pigment epithelial sodium-potassium pump.[19] An important distinction for visual prognosis is whether the patient received an incorrect toxic intracameral dose or if the dose was correct, but the patient experienced a sporadic adverse response to the medication. In one study, 42.10% of patients who received a greater than standard intracameral injection of cefuroxime (10mg to 12.5mg/0.1mL) exhibited ocular side effects.[14] The majority of these cases exhibited a toxic maculopathy response which was documented with macular OCT.[14] All these patients had a significant decrease of their visual acuity as measured on their first postoperative day after the cataract surgery. All but one patient showed some improvement in their final BCVA by postoperative week 12.[14] The patient who showed no improvement remained at BCVA of 20/200. Of the patients whose vision recovered (at least partially), one patient improved from BCVA 20/200 to BCVA 20/20 by postoperative week 12.[14] The rest were in the 20/40 to 20/120 range of BCVA by postoperative week 12.[14]

In contrast, in a different study of 20 patients who developed toxic maculopathy after receiving an intracameral injection of correctly diluted cefuroxime, all 20 patients recovered the majority of their BCVA by their first postoperative weekly visit.[19] The current patient also had a significant recovery of BCVA within her first postoperative week (from 20/125 to 20/25). By her seventh postoperative week her BCVA was 20/20. It was suspected that this patient's toxic maculopathy was most probably sporadic since none of the other patients who had cataract surgery on the same day and received an intracameral injection of cefuroxime at the conclusion of the surgery exhibited any anterior or posterior segment toxic response. The hospital pharmacy was also asked to double-check their dilution protocol and to verify that the correct dilution was prepared on the day of this patient's surgery. The hospital pharmacy confirmed that the usual dilution protocol was followed on that day and that the cefuroxime dilution was of a correct dosage. Had the dilution been

incorrect, the patient's potential for visual acuity recovery would have been more guarded.

It has been suggested that in clinical practice, many such sporadic cefuroxime-induced toxic maculopathy cases might go undetected because the poor visual acuity on the first day postoperative visit may be attributed to more typical postoperative factors such as pupil dilation, elevated intraocular pressure, corneal edema or inflammation.[19] By the first postoperative week, these patients will probably have a significant improvement in their BCVA along with nearly resolved cystoid macular edema and serous macular retinal detachment. Therefore, all co-managing clinicians should maintain a high level of suspicion in any postoperative case where there appears to be a mismatch between the patients' BCVA and the examination findings. This patient's BCVA on her first postoperative day did not match the minor anterior segment findings (trace diffuse corneal edema with no Descemet's folds and trace anterior chamber cell). This mismatch warrants further investigation which includes a dilated fundus exam and macular OCT. If toxic maculopathy is identified, the co-managing optometrist should communicate with the cataract surgeon and refer to a retina specialist for additional testing. It is the cataract surgeon's responsibility to verify whether the cefuroxime dilution was correct or incorrect. This information is important since it helps significantly in anticipating the final surgical outcome.

The cefuroxime-induced toxic maculopathy can mimic postoperative pseudophakic cystoid macular edema (PCME) also known as Irvine-Gass syndrome. There are, however, some critical differences between these two postoperative clinical entities. Cefuroxime-induced toxic maculopathy occurs on the first postoperative day. PCME has a peak incidence of six to 10 weeks following cataract surgery.[21] While both entities can be seen in uncomplicated cataract surgeries, PCME is more commonly present in patients with a history of complicated surgery involving vitreous loss, iris incarceration and chronic inflammation.[5] PCME is also more frequently seen in patients with diabetes mellitus.[22]

As described earlier, the cystoid macular edema that results from cefuroxime-induced toxicity mainly involves the outer nuclear layer of the retina and there is an accompanying serous macular retinal detachment.[19] PCME is caused by a breakdown in the blood-aqueous barrier due to inflammation.[23] This inflammation is a result of either a complicated cataract surgery or a patient's underlying systemic condition.[5,22] The chronic inflammation causes increased vascular permeability and leakage, specifically from the perifoveal capillaries with eosinophilic exudative fluid collecting in the loosely arranged outer plexiform and inner nuclear layers.[19,23]

Furthermore, there is usually no evidence of this leakage on fluorescein angiography in the cefuroxime-induced toxicity cases while the PCME cases will show leakage of the perifoveal capillaries during the early fluorescein angiogram phase (Figure 9)

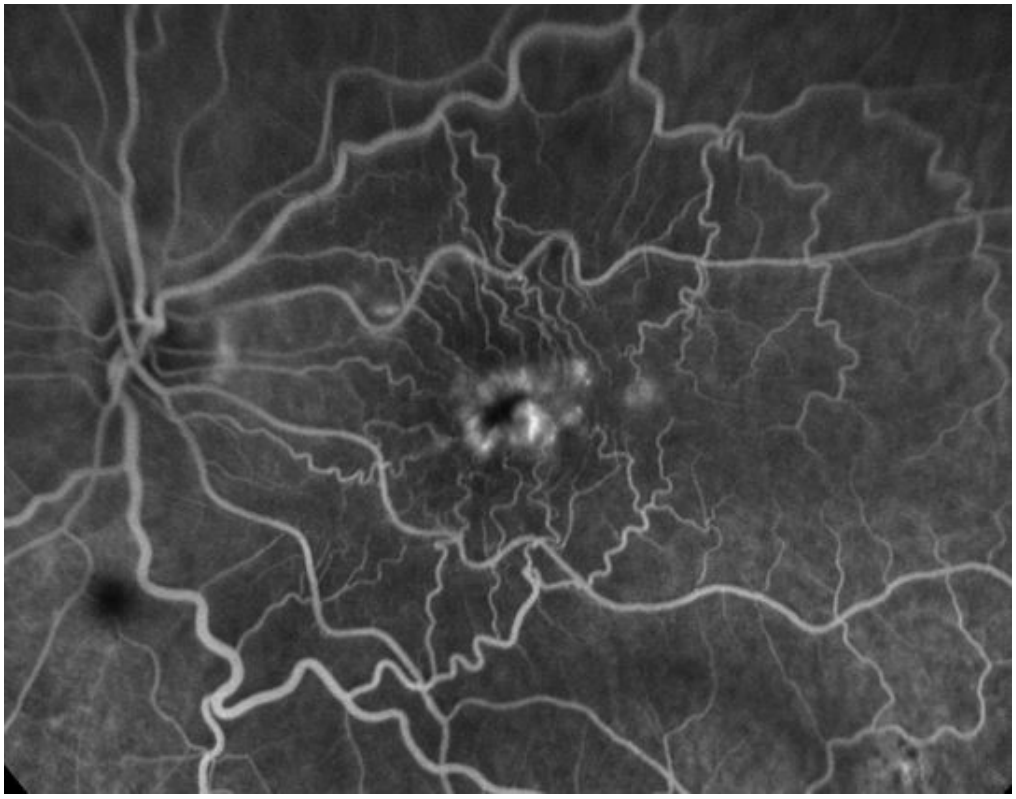


Figure 9: Fluorescein angiogram - early phase in patient with pseudophakic cystoid macular edema.

and a petaloid-shaped pattern of hyperfluorescence over the macula during the late phase (Figure 10).[21,23]

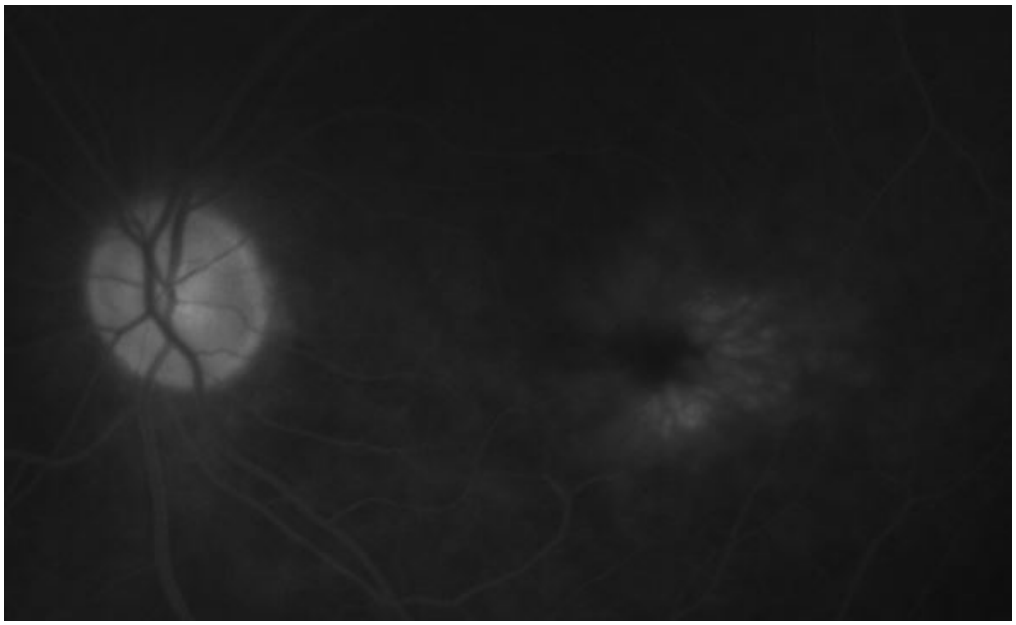


Figure 10: Fluorescein angiogram - late phase in patient with pseudophakic cystoid macular edema.

The OCT of PCME patients will also differ by exhibiting thickening and fluid accumulation in the form of cystic spaces predominantly in the outer plexiform and inner nuclear layers instead of the outer nuclear layers (Figure 11 and Figure 12).[22] PCME patients do not exhibit an accompanying serous macular retinal detachment.

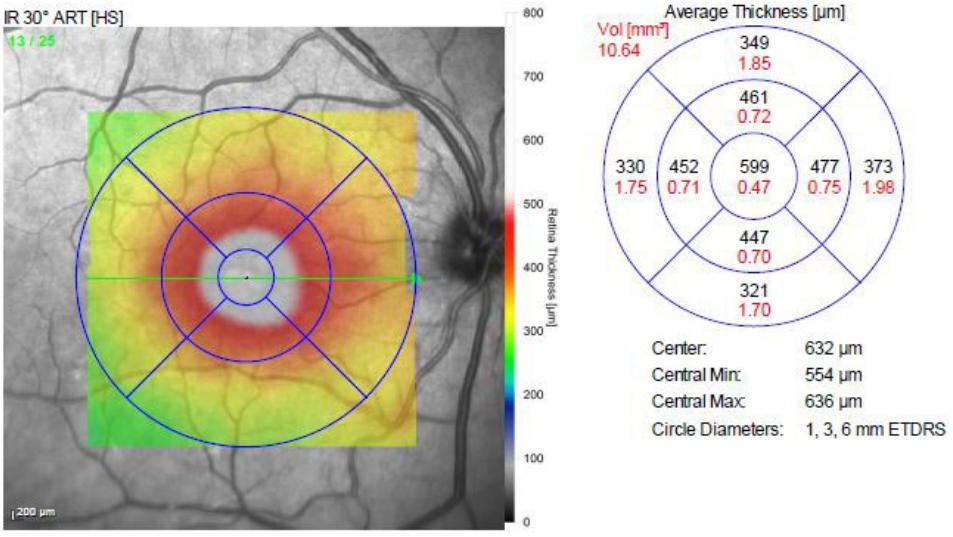


Figure 11: Optical coherence tomography of macula in patient with pseudophakic cystoid macular edema. Right eye.

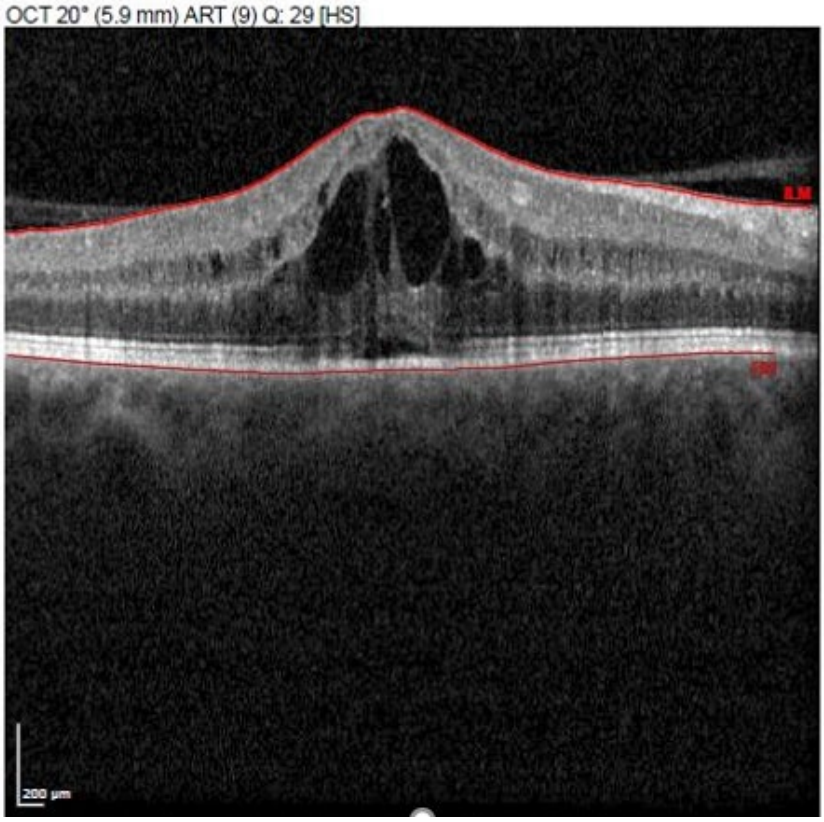


Figure 12: Optical coherence tomography of macula in patient with pseudophakic cystoid macular edema. Right eye.

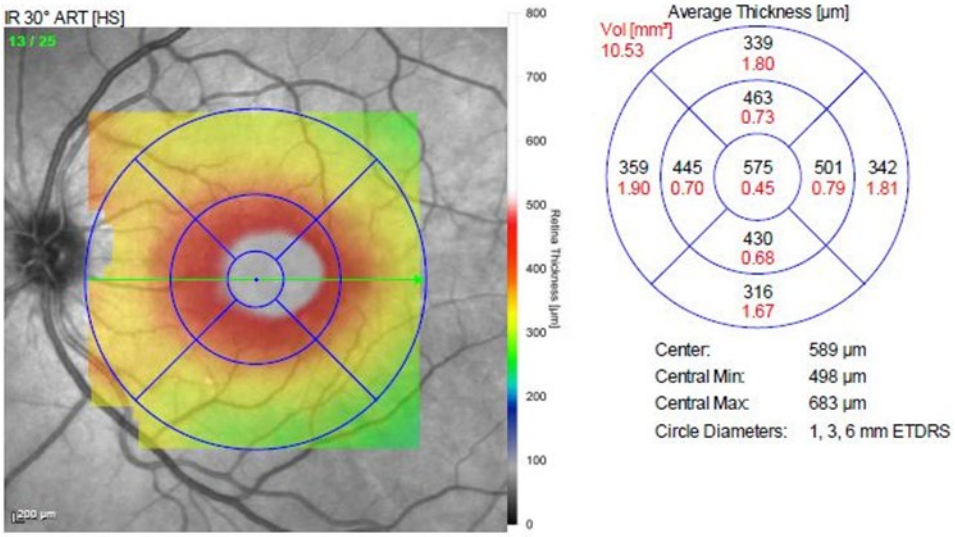


Figure 13: Optical coherence tomography of macula in patient with pseudophakic cystoid macula edema. Left eye.

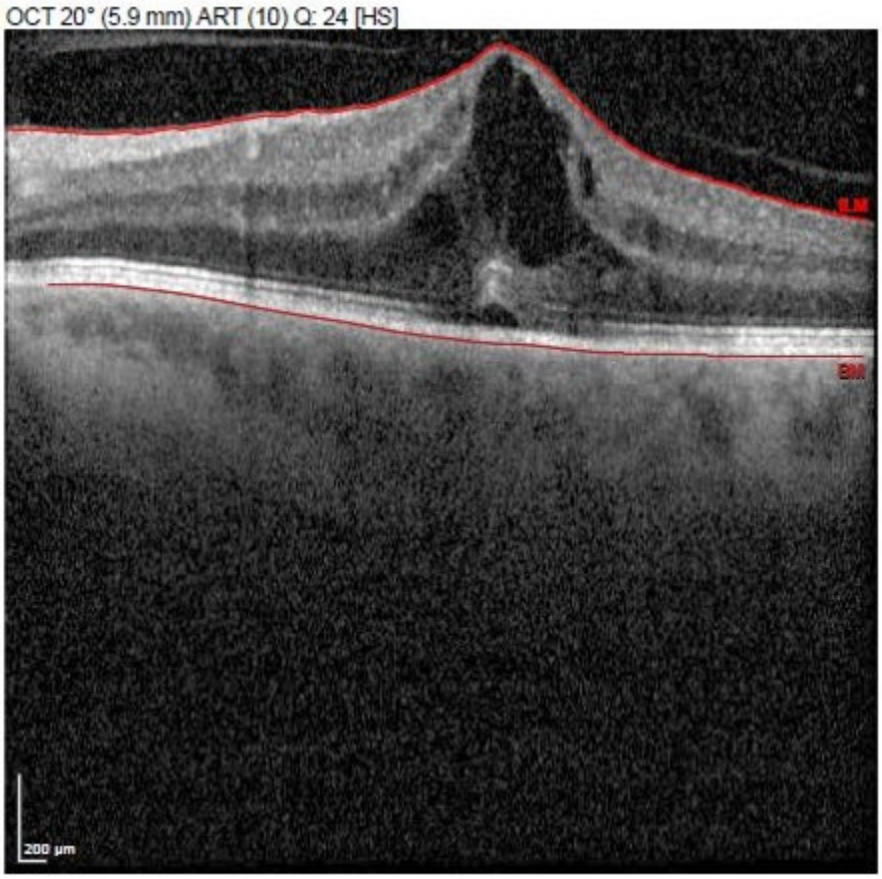


Figure 14: Optical coherence tomography of macula in patient with pseudophakic cystoid macula edema. Left eye.

Sporadic cefuroxime-induced toxic maculopathy shows significant resolution by the first postoperative week.[19] PCME often resolves spontaneously within six months.[21] To speed resolution of PCME, topical or oral non-steroidal anti-inflammatory medications in combination with topical or repository injections of steroids have shown adequate efficacy of resolution.[21-23] Permanent macular degeneration may arise secondary to prolonged chronic cystoid macular edema.[22]

There is no known treatment for cefuroxime-induced ocular toxicity. If the patient has significant anterior segment changes such as corneal edema, Descemet's folds and anterior chamber inflammation then increasing the frequency of the postoperative topical steroid dosing should be initiated. This should also be done in cases with severe vitritis. In addition, a repository steroid injection can be

performed. An endophthalmitis (infectious vs. sterile) evaluation should be considered in cases of severe vitritis or other non-classic posterior segment involvement.

CONCLUSION

Cefuroxime-induced ocular toxicity that results from a greater than standard dilution dosage administration has a more guarded visual prognosis. Sporadic cases usually resolve quickly with an improved visual acuity by the first postoperative week after cataract surgery.

Co-managing optometrists should know if their referring cataract surgeon uses intracameral cefuroxime at the conclusion of their phacoemulsification surgery. If so, co-managing optometrists should maintain a high level of suspicion regarding possible cefuroxime-induced ocular toxicity whenever there is a significant mismatch between the patient's first day postoperative BCVA and their clinical findings.

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