Simultaneous existence of three intraocular lens inside one eye

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Abstract:
The purpose of the study was to report an unusual case of recurrent pseudophakic cystoid macular edema (PCME) in an eye with three simultaneous intraocular lenses (IOLs) inside. A 57-year-old female with diabetes mellitus (DM) and a history of complicated cataract surgery was diagnosed with cystoid macular edema (CME). Upon examination, an anterior chamber intraocular lens (ACIOL) with vitreous strand in her right eye was noted. The fluorescence angiography revealed CME of the right eye and microaneurysms in both eyes. Pars plana vitrectomy was performed to release the vitreous prolapse and traction around the ACIOL. During the surgery, two sunken posterior chamber IOLs in the vitreous were incidentally found and removed. The vitreous traction strand around the inappropriately placed anterior chamber ACIOL was also released. It was rarely reported that two dislocated IOL and ACIOL simultaneously existed in the same eye. Chronic recurrent PCME in this patient was possibly associated with posteriorly dislocated IOL, DM, and vitreous traction.

Keywords:
Macular edema, multiple intraocular lens, pars plana vitrectomy, pseudophakia, pseudophakic cystoid macular edema

Introduction

Cataract surgery is one of the most common procedures, and pseudophakic cystoid macular edema (PCME) is a common postoperative complication.¹,² Although most cases of acute PCME may spontaneously resolve, some patients will develop chronic PCME which may lead to visual loss.³ Patients with diabetes have an increased risk of developing PCME after cataract surgery.⁴ Other common risk factors of PCME including aging, uveitis, retinal vein occlusion, or retinal detachment.⁵

Since refractory macular edema is still a therapeutic challenge nowadays, reviewing the patients’ history and a comprehensive examination are essential for consideration of the possible factors of PCME.⁶ We report a case of a frequently recurrent PCME after the medical treatment associated with multiple dislocated intraocular lenses (IOLs). To the best of our knowledge, this is very likely the first report revealing three IOLs simultaneously existed in one eye. This report may remind our colleagues about this possible finding when encountered on an intractable PCME.

Case Report

A 57-year-old female with a history of diabetes mellitus sought consultation in our hospital due to blurred vision in the right eye for 1 year. One year before consultation, the patient underwent cataract surgery in her right eye and had two reoperations within 1 month at a local clinic. Since then, she has experienced blurring of vision.

Upon consultation, best-corrected visual acuity (BCVA) was 6/60 and 6/12 in the right and left eyes, respectively. Intraocular
pressure (IOP) was 18 and 19 mmHg in the right and left eyes, respectively. Slit-lamp examination revealed a clear cornea and the presence of an anterior chamber intraocular lens (ACIOL) with vitreous strand in her right eye. Dilated fundus examination was performed which revealed several dots and blot hemorrhages and some creamy whitish patches around the macular. However, because of the limitation of margin of ACIOL and difficulty for full dilatation of the pupil, peripheral retina could not be fully examined [Figure 1a]. An optical coherence tomography (OCT) showed cystoid macular edema (CME) in the right eye with a central retinal thickness of 343 μm [Figure 1b] and fluorescein angiography demonstrated CME in a petaloid pattern with severe leakage in her right eye [Figure 1c]. After discussing with the patient, intravitreal injection of aflibercept along with topical corticosteroid (1% prednisolone acetate) was administered to treat the CME. BCVA improved to 6/12, and the OCT showed improvement in CME with a central retinal thickness of 312 μm in the next month’s follow-up [Figure 2a].

However, 2 months following the first dose of aflibercept, macular edema recurred with central retinal thickness up to 363 μm as seen in the OCT [Figure 2b], and the BCVA decreased to 6/24. Therefore, a second dose of intravitreal injection of aflibercept was administered. The OCT revealed improved macular edema in the next month with a central retinal thickness of 301 μm [Figure 2c]. However, the patient experienced micrographia and metamorphopsia in her right eye. Three months after the second injection, OCT showed recurrent CME and subretinal fluid with a central retinal thickness of 363 μm [Figure 2d]. Thus, a third dose of intravitreal aflibercept injection was administered. Considering the presence of refractory macular edema and the possibility of vitreous traction, pars plana vitrectomy was performed to release the vitreous prolapse and traction.

During the surgery, we incidentally found two additional dislocated IOLs (a “Rayner” and a “Lentis” type, respectively) in the vitreous cavity [Figure 3] and a focal rhegmatogenous retinal detachment. The two intravitreal IOLs were removed followed by fluid air exchange, endolaser, and C3F8 tamponade for the retinal detachment. No epiretinal membrane was found, and the internal limiting membrane was not peeled.

Throughout the postoperative follow-up, ACIOL subluxation with protrusion through the sclera was noted. Four days after vitrectomy, the patient underwent secondary removal of the ACIOL. Unfortunately, moderate corneal edema was present postoperatively for over 6 months which rendered her vision to only 6/60 level despite that repeated OCT revealed no recurrence of CME in this period.

Figure 1: The initial visit image data. (a) Color fundus photography revealed central macular edema. (b) Optical coherence tomography of right eye showed cystoid macular edema with central retinal foveal thickness of 343 μm. (c) Fluorescein angiography revealed cystoid macular edema with severe leakage

Figure 2: The sequential optical coherence tomography during the treatment. (a) Optical coherence tomography showed improved macular edema after 1st injection of aflibercept with central retinal thickness of 312 μm. (b) Optical coherence tomography showed recurrent cystoid macular edema 2 month after 1st injection of aflibercept with central retinal thickness of 363 μm. (c) Optical coherence tomography showed improved macular edema the next month after 2nd injection of aflibercept with central retinal thickness of 301 μm. (d) Optical coherence tomography showed recurrent cystoid macular edema and subretinal fluid 3 months after 2nd injection of aflibercept with central retinal thickness of 363 μm.
Discussion

PCME, also known as Irvine-Gass syndrome, is a common complication following cataract surgery. Inflammation plays a vital role in its pathogenesis. Inflammatory mediators are released after the surgical manipulation which disrupt the blood–retinal barriers, leading to fluid leakage to the macular region. Therefore, topical corticosteroids and nonsteroidal anti-inflammatory agents are commonly used in most cases of chronic PCME.

In our case, a topical steroid was initially prescribed. In addition, intravitreal aflibercept injections were administered to reduce macular edema. Vascular endothelial growth factor (VEGF) plays a role in the inflammation process and vascular permeability, contributing to the development of macular edema. Anti-VEGF decreases the steroid use and reduces the risk of elevation in IOP. In our patient, the administration of anti-VEGF along with topical corticosteroid did achieve a transient subsidence of PCME. However, the PCME recurred frequently and repeated anti-VEGF was needed in a frequency of every 1–2 months.

There may be certain factors contributing to our patient’s recurrent macular edema. First, she had undergone a complicated cataract surgery. The postoperative inflammatory response may be more intensive and prone to a difficult postoperative recovery. Second, the risk of PCME is increased in patients with diabetes. It results in a more vulnerable vascular bed and a higher concentration of prostaglandin and other inflammatory mediators. Third, vitreous loss and traction associated with ACIOL are also related to chronic PCME. The mechanical force can lead to vitreoretinal disturbance and inflammatory response. Since vitrectomy has been shown to be effective for the treatment of chronic PCME, we, therefore, performed a pars plana vitrectomy for the intention of relieving the vitreous traction. This leads to the incidental finding of two additional dislocated IOLs in the vitreous cavity.

In addition to vitreous prolapse and traction around the ACIOL, the mobile IOLs may also be an important factor for the refractory macular edema in our case. Since IOLs tend to move with postural change, the friction between the IOLs and retina and between the IOLs and iris can lead to sustained inflammation. The inflammatory mediators were also elevated in the vitreous of pseudophakic eyes. Furthermore, the intraocular foreign body in the vitreous cavity could cause damage to the retina or uveal tissue, thus leading to retinal detachment, CME, or endophthalmitis.

To make things more complicated, in this case, we failed to identify the dislocated IOLs during the previous examination. The IOLs might be laid on the retina in the inferior vitreous cavity due to gravity or hidden behind the iris, while the patient was examined in a sitting position. In addition, the obvious vitreous loss and traction membrane around the ACIOL and to the corneal wound together with the difficulty for full dilatation of pupil could possibly mask the discovery of additional causes of chronic PCME in our patient. A high suspicion of the possibility of dislocated IOLs and further indirect ophthalmoscopic examinations using supine position, ultrasound biomicroscopy, anterior segment OCT, or ultra-widefield fundus photography might help for the early detection of dislocated IOL.

In conclusion, we reported an unusual case of recurrent PCME with three IOLs in the same eye. These retaining IOLs may be one of the main reasons for the refractory macular edema in our patient. Although several diluted fundus examinations had been attempted in our patient, the obscuration effect of poorly dilated pupil and the margin of the ACIOL still precluded the finding of the dislocated posterior chamber IOLs in the far inferior part of fundus. This report highlights the importance of high suspicions of additional dislocated IOL when treating patients with complicated cataract surgery and refractory PCME.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

Dr. Cheng-Kuo Cheng, an editorial board member at Taiwan Journal of Ophthalmology, had no role in the peer review process of or decision to publish this article.
References

1. Flach AJ. The incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery. Trans Am Ophthalmol Soc 1998;96:557-634.
2. Irvine SR. A newly defined vitreous syndrome following cataract surgery. Am J Ophthalmol 1953;36:599-619.
3. Shelsta HN, Jampol LM. Pharmacologic therapy of pseudophakic cystoid macular edema: 2010 update. Retina 2011;31:4-12.
4. Henderson BA, Kim JY, Ament CS, Ferrufino-Ponce ZK, Grabowska A, Cremers SL. Clinical pseudophakic cystoid macular edema. Risk factors for development and duration after treatment. J Cataract Refract Surg 2007;33:1550-8.
5. Negi AK, Browning AC, Vernon SA. Single perioperative triamcinolone injection versus standard postoperative steroid drops after uneventful phacoemulsification surgery: Randomized controlled trial. J Cataract Refract Surg 2006;32:468-74.
6. Spitzer MS, Ziemssen F, Yoeruek E,彼得meier K, Aisenbrey S, Szurman P. Efficacy of intravitreal bevacizumab in treating postoperative pseudophakic cystoid macular edema. J Cataract Refract Surg 2008;34:70-5.
7. Shah AS, Chen SH. Cataract surgery and diabetes. Curr Opin Ophthalmol 2010;21:4-9.
8. Nelson ML, Martidis A. Managing cystoid macular edema after cataract surgery. Curr Opin Ophthalmol 2003;14:39-43.
9. Jakobsson G, Sundelin K, Zetterberg H, Zetterberg M. Increased levels of inflammatory immune mediators in vitreous from pseudophakic eyes. Invest Ophthalmol Vis Sci 2015;56:3407-14.
10. Harbour JW, Smiddy WE, Rubsamen PE, Murray TG, Davis JL, Flynn HW Jr. Pars plana vitrectomy for chronic pseudophakic cystoid macular edema. Am J Ophthalmol 1995;120:302-7.
11. Soliman Mahdy M, Eid MZ, Shalaby KA, Hegazy HM. Intravitreal phacoemulsification with pars plana vitrectomy for management of posteriorly dislocated nucleus or lens fragments. Eur J Ophthalmol 2010;20:115-9.
12. Monshizadeh R, Samiy N, Haimovici R. Management of retained intravitreal lens fragments after cataract surgery. Surv Ophthalmol 1999;43:397-404.
13. Williams DF, Del Piro EJ, Ferrone PJ, Jafe GJ, McDonald HR, Peters MA. Management of complications in eyes containing two intraocular lenses. Ophthalmology 1998;105:2017-22.
14. Emanuelli A, Smiddy WE. Management of 2 intraocular lenses in the same eye. JAMA Ophthalmol 2013;131:86-7.