The Importance of Anterior Segment Examination in Resistant Macular Edema

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ABSTRACT

A 28-year old male patient underwent cataract surgery and secondary IOL implantation in the sulcus at an external centre for a diagnosis of traumatic cataract to the right eye. The patient then had reduced vision and was diagnosed with CMO. Despite a total of 15 anti-VEGF applications at the external centre, the edema did not improve. In our examination, it was seen that the IOL had been placed in the sulcus and one leg was suspended on the iridotomy. IOP was 26mmHg. On FFA, there was seen to be leakage in the optic disc, peripheral retina and macula. It was thought that the CMO was secondary to inflammation caused by contact of the IOL on the iris. Surgery was performed by suturing the haptics to the sclera using the patient's own IOL. Then no CMO was observed throughout 2 years. The elevated IOP continued and the causes of this were thought to be steroid use, inflammation and damage at the micro level in the angular structure associated with the trauma.

Keywords: cystoid macular edema, etiological factors, Irvine-Gass syndrome, Pseudophakic cystoid macular edema.

INTRODUCTION

The accumulation of fluid in the outer plexiform and inner nuclear layers of the fovea, which occurs as a result of leakage from the parafoveal, retinal and optic nerve capillaries, is known as cystoid macular edema (CME). Various reasons such as vitreous traction, impaired retina pigment epithelial pump function and impaired blood retina barrier may play a role alone or together in the development of CME. Diagnosis is made from OCT and is supported by FFA. FFA is also important as it may provide additional findings of the etiology.[1]

The reasons for CME include vascular (diabetic retinopathy, retnal vein occlusion, choroid neovascularisation), postoperative (cataract surgery, keratoplasty, iridectomy-iridotomy, uveitis-hyphema-glaucoma syndrome, inflammatory, trauma, toxic, tumour and hereditary etiological causes.[2]

Case Report

A 28-year old male underwent cataract surgery and secondary IOL implantation in the sulcus at an external centre for a diagnosis of traumatic cataract following trauma to the right eye 5 years previously. Some time after the operation, the patient experienced reduced vision, and intravitreal anti-VEGF injection was administered as there was said to be macular oedema. The vision improved after the injection but after one month, blurred vision occurred again. Macular oedema was determined in the examination and anti-VEGF injection was again administered. It was also learned that the patient had been using steroid and non-steroid anti-inflammatory drops for a long time. As the vision improved followed by recurrence of blurred vision, the patient presented at another centre and throughout a 2-year period received a total of 15 anti-VEGF injections for a diagnosis of macular oedema. Despite these injections, as there was no regression of the macular oedema, the patient then presented at our clinic.

In the examination of the patient, there was determined to be haze in the cornea inferior paracentral and iridotomy in the superior iris of the right eye. The posterior capsule was seen to be open. The intraocular lens had been placed in the sulcus and one leg was seen to be placed in the iridotomy (Figures 1a-b-c-d).

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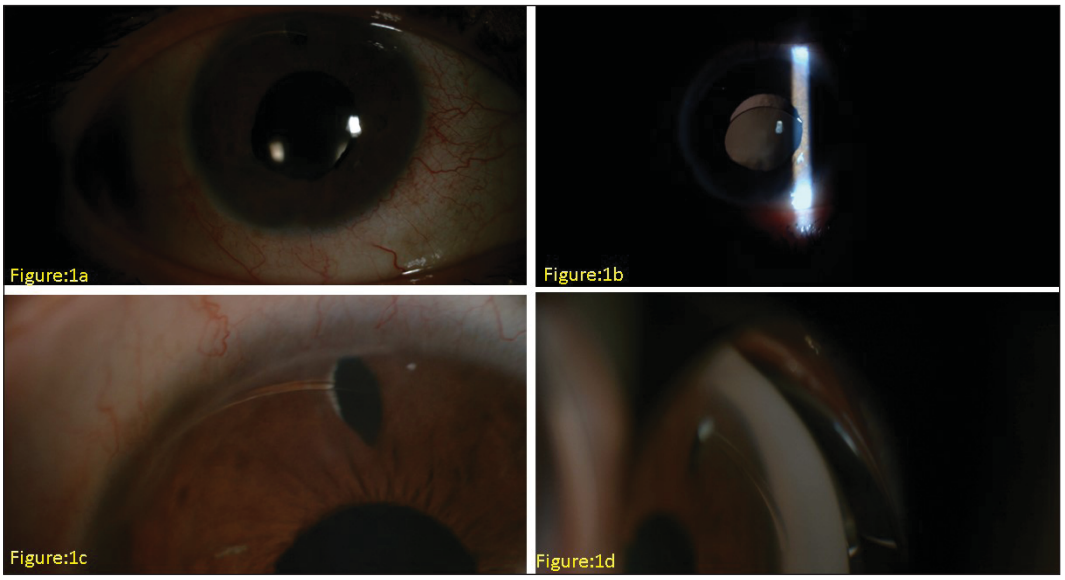

Figure 1: a. *Corneal haze in the inferior paracentral region and the haptics in the iridotomia area is seen in the anterior segment examination,* **b**. *The intraocular lens is seen to be subluxated to inferior,* **c**. *It is seen that the intraocular lens is placed in the sulcus and suspended by one of the haptics in the iridotomy area,* **d**. *The intraocular lens appears to touch the angle area and iris.*

There were no findings of uveitis or hyphema. Intraocular pressure (IOP) was measured as 26mmHg with applanation tonometry. As a result of fundus examination, OCT and FFA, cystoid macular edema was determinated (Figure 2a-b-c-d). On the wide-angle FFA, also there was leakage in the peripheral retina and optic disc. and scar appearance was observed in the infero-temporal peripheral retina. No extra pathology was determined in the peripheral retina scan with 3 mirror lens. No pathology was determined as a result of retina nerve fibre analysis, corneal topography and visual field tests. With a diagnosis of secondary elevated IOP, Timolol-Brimonidine drop combination was started at a dosage of 2+1.

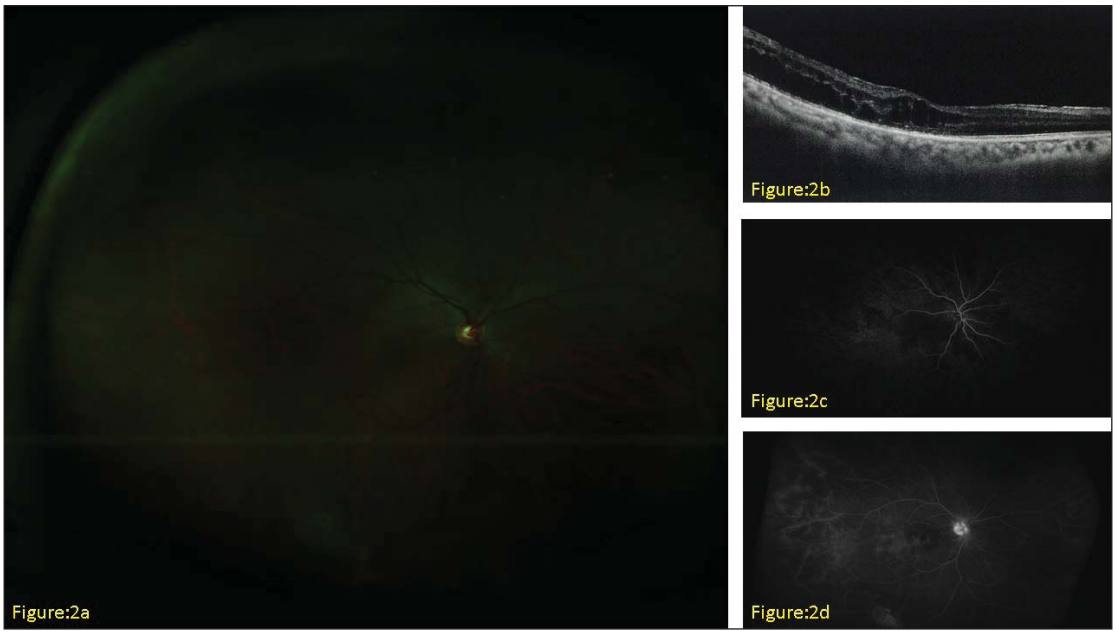

Figure 2: a. *The scar appearance was observed in the infero-temporal peripheral retina in the ultra wield angle fundus photo,* **b**. *The cystoid macular appearance was observed in macular OCT,* **c**. *In early stage wide angle FFA, no pathology is observed,* **d.** *In late stage wide angle FFA, leakage is observed in the macula, optic disc and peripheral retina.*

According to the angiography findings, the macular oedema present in the patient was thought to be secondary to the inflammation formed associated with the intraocular lens placed in the sulcus being mobile and having one end of the haptic suspended in the iridotomy. Surgery was performed, suturing the haptics to the sclera with the scleral fixation method using the patient's own intraocular lens. No macular oedema was determined on OCT examinations made in the postoperative follow-up examinations. To date, during a 2-year follow-up period, no macular oedema has developed (Figure 3a-b-c-d). The anti-glaucoma treatment was terminated, but in the follow-up examinations IOP increased again so the same topical medical treatment was continued. No cupping over the optic nerve was determined and no glaucomatous defect was seen in the visual field test with retinal nerve fibre layer. Damage at the micro-level in the angular structure associated with the trauma, inflammation and steroid use were considered among the reasons for the elevated IOP. It was thought that decreasing the pressure with medical treatment would prevent the progression of the patient to glaucoma.

DISCUSSION

In cases with resistant CMO, clues about the etiological cause should be gathered by taking a detailed anamnesis and making a detailed anterior segment examination. From the detailed anamnesis taken from the current patient, no vascular, rheumatological or hereditary disease was determined and no drug had been used in terms of toxic etiology. No findings of uveitis, hyphema, or tumour were determined in the examination as other etiological causes. As there was a history of trauma and surgery, pseudophakic CMO associated with the initial trauma and the subsequent cataract surgery, and secondary elevated IOP were initally considered as diagnoses.

Macular oedema associated with trauma is seen more in the form known as Berlin's oedema and rarely as cystoid macula oedema. An effect develops in the interface between the photoreceptor layer and the retina pigment epithelium due to the shock waves forming following blunt trauma, and this trauma causes an outward expansion of the peripheral structures and migration of the lens-iris diaphragm.[3] The retina is not flexible and by absorbing the whole effect of the shock waves, there is damage to the outer part of the photoreceptor in particular and to various layers of the RPE link. The affected outer retina layers become opaque, the retina vessels below the opaque area appear regular, and on fundus angiography, vessels appear normal and leakage cannot be seen.[4] As the current patient was seen to have leakage on FFA, and a long time had passed since the trauma, traumatic macular oedema was discounted from the diagnosis.

With the effect of the blow sustained in blunt trauma there

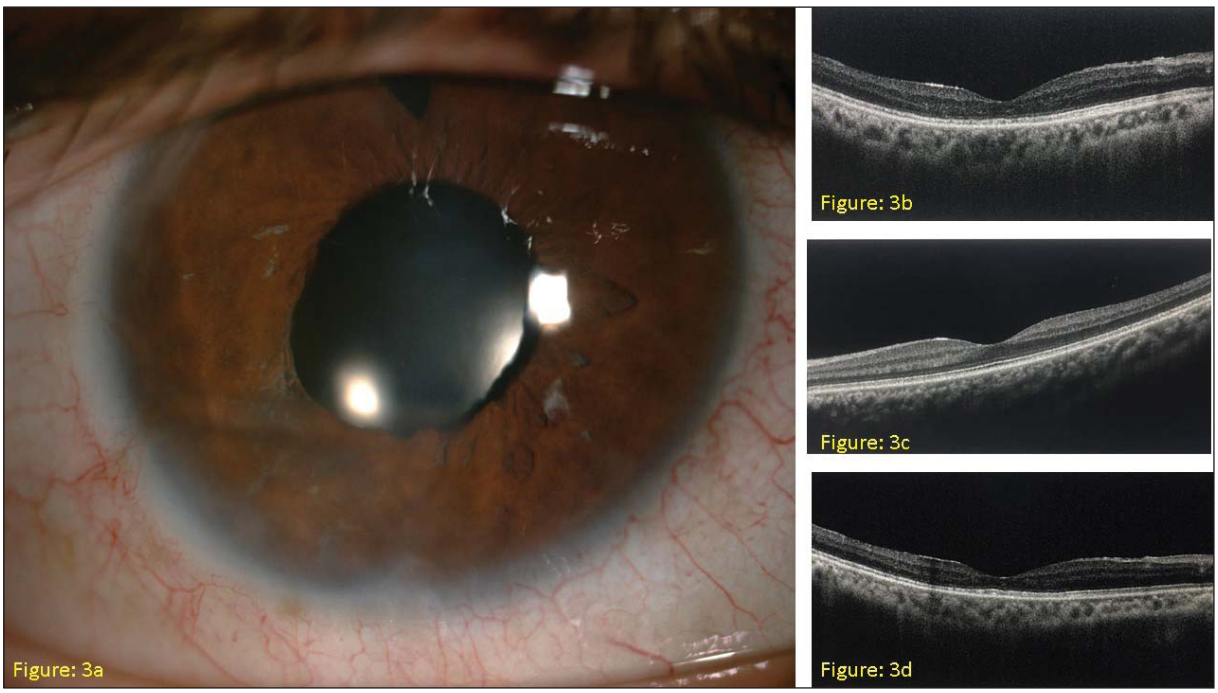

Figure 3: a. *Anterior segment appearance is observed in the postoperative 1st month (There is no haptic in the iridotomy area),* **b**. *It is observed that there is no macular edema in OCT in the postoperative 1st month control,* **c.** *It is observed that there is no macular edema in OCT in the postoperative 1st year control,* **d.** *It is observed that there is no macular edema in OCT in the postoperative 2 th year control.*

is a shortening in the anterior-posterior axis of the eye, and an expansion in the lateral axis. Damage may occur in the sphincter pupilla, iris root, trabeculum, and other angular elements that cannot adjust to these changes, and there may be tears in the ciliary body and zonular fibres, and breakages in the branches in the great artery ring of the iris. Elevated IOP secondary to trauma and glaucoma can occur associated with these findings.[5] Post-traumatic increases in IOP may be seen in the early or late period together with any one of these. However, as the IOP did not decrease after surgery in the current patient, it was thought that elevated IOP could have developed secondary to defects in the angular structure caused by the trauma. Inflammation and the use of steroids may also have contributed to the elevated IOP.

Pseudophakic CMO, also known as Irvine-Gass syndrome, is one of the most important causes of painless reduced visual acuity following cataract surgery. The incidence increases when there are complications during the operation. With improvements in cataract surgery, there has been a significant decrease in the size of incisions, especially during phacoemulsification. Clinically significant CMO is seen in only 1-2% of patients with reduced visual acuity and metamorphia and the highest incidence has been reported to emerge after mean 6 weeks postoperatively and despite the application of preventative treatment, subclinical CMO is determined in approximately 30% of patients with postoperative angiography and in 11-41% with OCT.[6] Known risk factors include posterior capsule rupture, vitreous loss, lens residue, iris or vitreous incarceration in the wound site, the use of iris retractors, the placement of iris-supported IOL, irritation of the iris by the IOL, and Nd:YAG laser capsulotomy.[7] As a healthy vitreous forms a barrier against the diffusion of inflammatory mediators, situations such as anterior migration, liquefaction or loss of the vitreous can facilitate the formation of CMO.[8] Similarly, in the current patient, by placement of the intraocular lens in the sulcus with suspension of the upper haptic in the iridotomy, the continuous movement of the lens irritating the iris may have caused pseudophakic CMO.

The most likely physiopathological hypothesis for pseudophakic CMO is the triggering of an inflammation cascade with the expression of pro-inflammatory cytokines during or after surgery. As a result of this onset of inflammation, changes occur in the blood-retina barrier with an increase in retinal vascular permeability, and an inflammatory response is formed.[10] Due to the increase in vascular permeability, leakages form in the optic nerve, macula and retina periphery, which can be determined on FFA. In the current patient, leakages were determined in the macula, optic nerve and peripheral retina on the wide-angle FFA examination. By fluid in the macula then accumulating over the outer plexiform layer and inner nuclear layer, cystic spaces can form as large spaces containing fluid.[10]

In the examination of the current patient, the IOL placed in the sulcus was continuously mobile resulting in chronic inflammation, and leakages in the optic disc, macula and retina periphery were observed on FFA. The patient had been unresponsive to 15 doses of anti-VEGF injections applied at other centres before treatment of the etiological cause. After stabilisation of the lens, which was continuously mobile in the sulcus, with the scleral fixation method, macular oedema had not developed again throughout a 2-year follow-up period. In addition, the elevated IOP determined in the patient had similarly formed secondary to inflammation. With the complete resolution of CMO after surgery and the findings of leakages in the optic disc, macula and peripheral retina, and IOP of 26mmHg, the inital diagnosis of pseudophakic CMO was confirmed. The persistence of elevated IOP postoperatively was considered to be due to histopathological changes in the angulation caused by the trauma together with the effects of inflammation and steroids.

In conclusion, an evaluation of a detailed anterior segment examination is important as the first step in resistant CMO treatment to avoid unecessary treatment and costs, and with the stabilisation of a mobile IOL suspended on the iris, as in the current patient, the development of CMO can be prevented.

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