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CASE REPORT

Macular phototoxic injury due to cataract extraction and trifocal intraocular lens implantation

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Abstract

A 53-year-old woman underwent to the right uncomplicated cataract surgery and a trifocal intraocular lens (IOL) implantation. Twenty-six days after the surgery, the patient was admitted to our department with reduced vision. Slit-lamp examination of anterior chamber showed a clear cornea with deep anterior chamber and a centralized IOL. Fundus examination showed macular hole-like lesion in the fovea. Optic coherence tomography showed parafoveal edema, photoreceptor integrity line disruption, and outer retinal atrophy in the fovea. Fluorescein angiography showed corresponding areas of hyperfluorescence without leakage, consistent with phototoxic maculopathy resulting from the operating microscope. She had been diagnosed with systemic lupus erythematosus (SLE) 10 years ago. We aimed to present a patient who had profound visual loss secondary to presumed macular phototoxicity following cataract extraction and IOL implantation possibly related to underlying SLE. Patients with SLE may be prone to phototoxic damage during eye surgery.

Keywords: Cataract extraction; macular phototoxicity; phototrauma; systemic lupus erythematosus.

Iatrogenic macular phototrauma induced by exposure to operating microscope illumination is a well-recognized cause of visual loss after ocular surgery. McDonald and Irvine were the first to describe light-induced retinopathy in human eyes that had undergone extracapsular cataract extraction with intraocular lens (IOL) implantation in the posterior chamber.[1]

Herein, we describe a patient who had profound visual loss secondary to presumed macular phototoxicity following cataract extraction and trifocal IOL implantation possibly related to underlying systemic lupus erythematosus (SLE).

Case Report

A 54-year-old woman without antecedents of ocular disease history underwent to the right uncomplicated cataract surgery and trifocal IOL implantation at a local clinic. Before cataract surgery, visual acuity (VA) was 0.4 logMAR in the right eye and optic coherence tomography (OCT) showed a normal macular image (Fig. 1). Twenty-six days after the surgery, the patient was admitted to our department with reduced vision.

VA of the affected eye was counting fingers at 3 m when she applied to us and on the 1st post-operative day. Slit-

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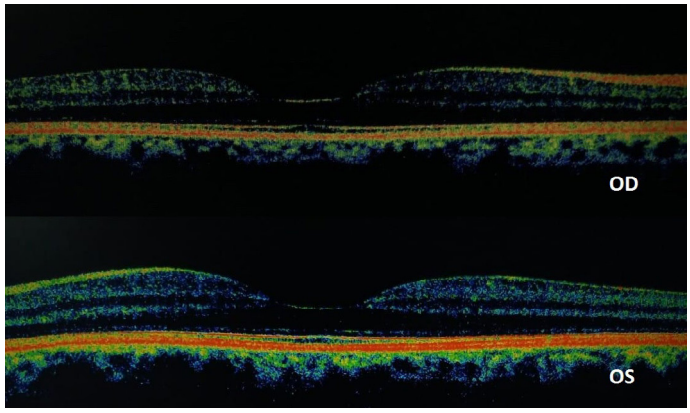

Fig. 1. Optical coherence tomography scan before cataract extraction.

lamp examination of anterior chamber showed a clear cornea with deep anterior chamber and a centralized IOL. The intraocular pressure was 12 mmHg, and there was no afferent pupil defect. Fundus examination showed macular hole-like lesion in the fovea. Fluorescein angiography showed corresponding areas of hyperfluorescence without leakage (Fig. 2). OCT showed parafoveal edema, photoreceptor integrity line disruption, and outer retinal atrophy in the subfoveal area (Fig. 3a). Four months after the first visit, VA did not improve and the cystic space healed with gliosis (Fig. 3b).

The foveal avascular zone (FAZ) was larger (0.436 mm^2) in the right eye than the left eye (0.387 mm^2) in OCT angiography (OCT-A). Superficial capillary plexus (SCP) vessel density (VD) was 51.2% in the right eye and 51.2% in the left eye. VD in deep capillary plexus (DCP) was 53% in the right

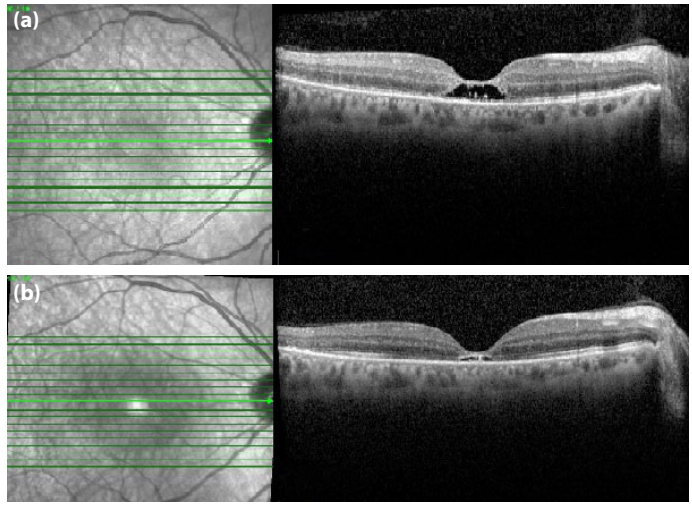

Fig. 3. **(a)** At first visit, optic coherence tomography (OCT) scan showed atrophy of outer retinal layers in subfoveal area, **(b)** 4 month after, the cystoid space was reduced due to gliosis in OCT examination.

eye and 52.9% in the left eye. There was no morphological alteration in SCP. The flow area of choriocapillaris plexus (CCP) was lower 2.129 mm^2 in the right eye than the left eye (2.299 mm^2). Irregular, granular hyper-reflectivity and hyporeflective cystic spaces were seen in the subfoveal area in en face image of outer retina (Fig. 4).

The surgery lasted in 20 min without complication. A coaxial light microscope (Leica, M220 F12) was used. A new model IQ PanOptix trifocal lens (Alcon) was implanted, with 21 diopters, The patient was diagnosed as SLE disease 10 years earlier and was receiving oral azathioprine

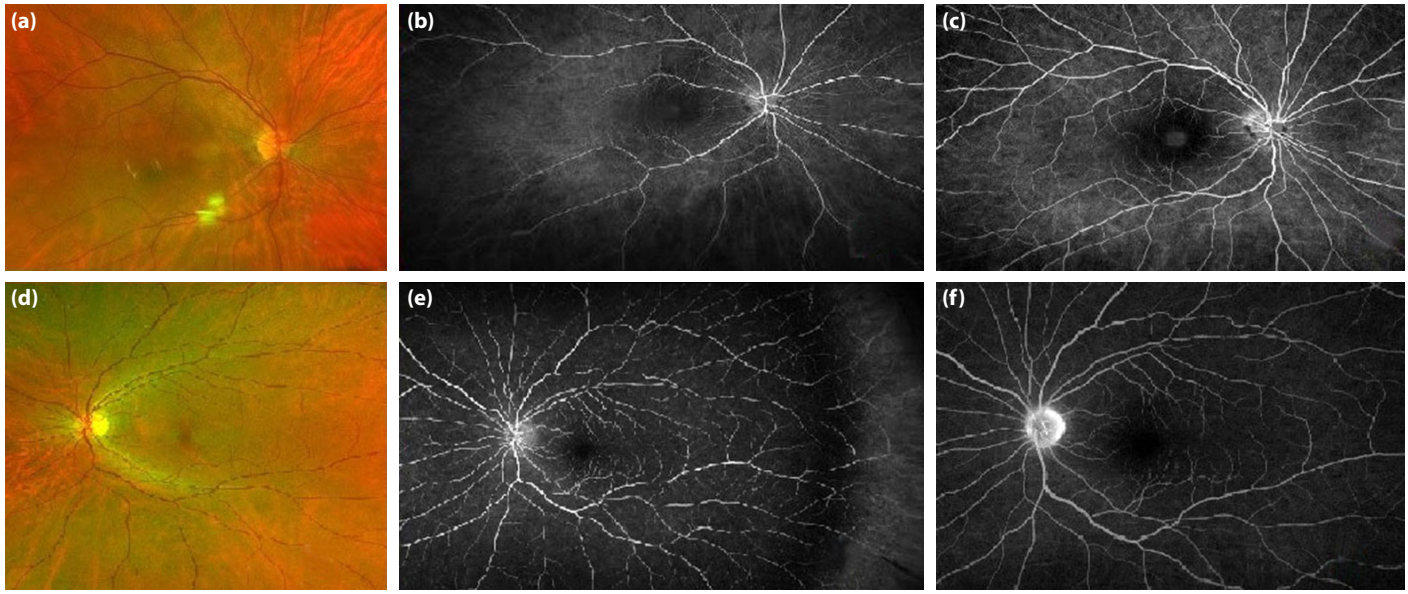

Fig. 2. **(a)** Color fundus photo of the right eye of case showed hyperpigmented macular lesion in the fovea. **(b)** Fluorescein angiography (FA) of the right eye: Early phase, **(c)** late phase, FA showed corresponding areas of hyperfluorescence without leakage. **(d)** Color fundus photo of the left eye, **(e)** FA of the left eye; early phase, **(f)** late phase of the left eye.

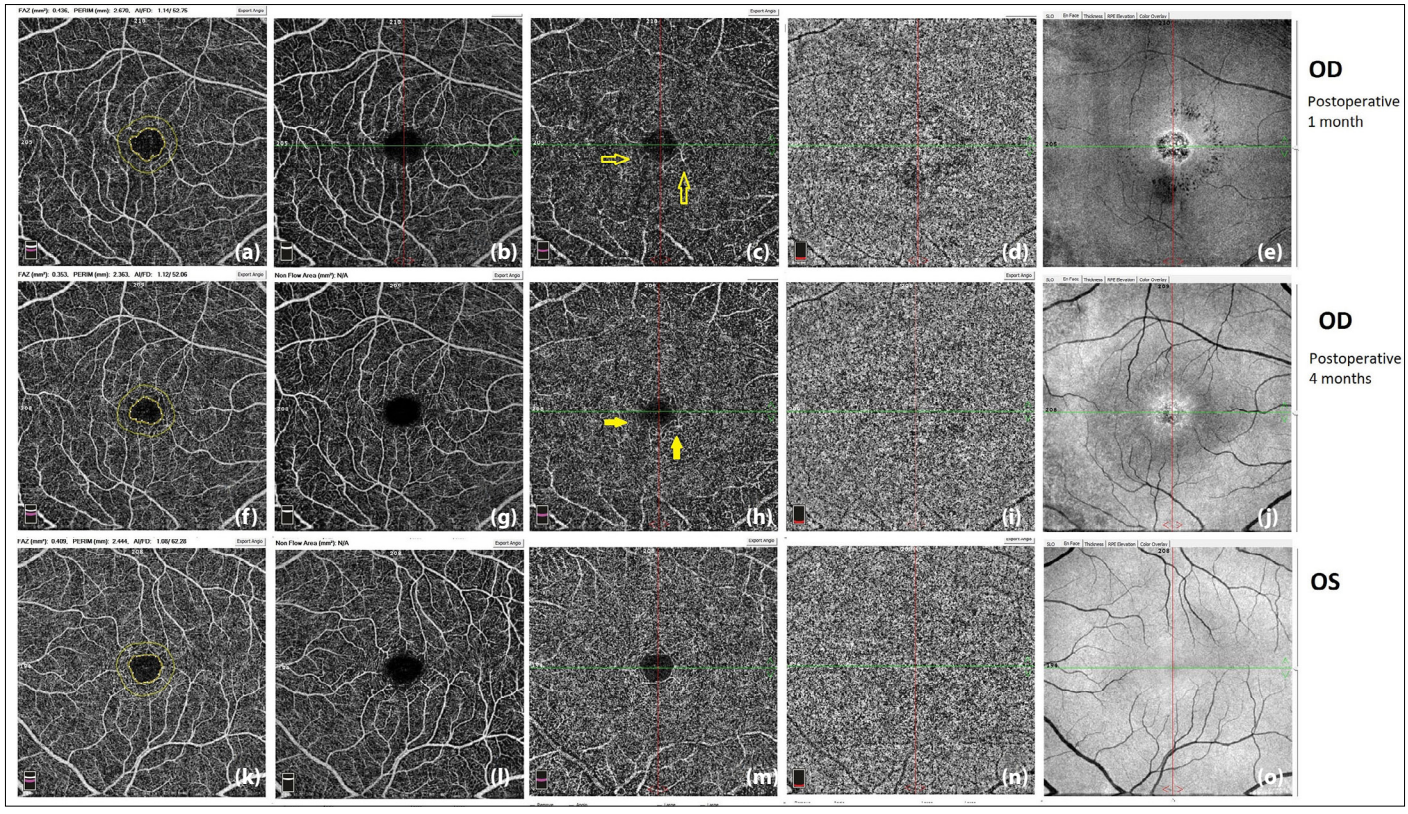

Fig. 4. **(a-o)** Optic coherence tomography angiography (OCT-A) images of right at post-operative 1 months and 4 months. The OCT-A image of the left eye at 4 months. Yellow arrows show the irregulation and disruption of foveal avascular zone

100 mg and colchicine 0.5 mg daily for 1 year. Our patient had never received hydroxychloroquine treatment before.

After 4 months, the FAZ was 0.353 mm^2 in the right eye in OCT-A. According to the first examination, the FAZ was found smaller. VD in SCP was 55.5%. VD in DCP was 57% in the right eye, and VD was increased compared to the first examination. The CCP flow area was 2.191 mm^2. Irregular, granular hyperreflective area was observed on en face image of outer retina. Healing with gliosis was examined with OCT-A after 4 months (Fig. 4). There were no abnormalities obtained by OCT-A in the left eye.

Discussion

Phototoxic injury to the macula may occur after cataract extraction. The previous studies reported that light-induced retinal toxicity was associated with surgical factors, such as operating time, focus, exposure to certain wavelengths of light, and level of light intensity emitted by the operating microscope.[2] Two similar cases previously presented in the literature.[3,4] Further research has revealed that phototrauma might be associated with fundus pigmentation, retinal vascular disease, and coexisting vascular diseases such as diabetes mellitus, hypertension, and a history of hydrochlorothiazide therapy.[5] However, these risk factors were not present in our patient.

The pathogenesis of phototoxicity is suggested as a consequence of imbalance between the light-induced reactive oxygen species and antioxidants.[6] Retinal phototoxicity studies in human eyes have demonstrated localized necrosis of retina pigment epithelium, intense deterioration of the outer layers of photoreceptors, edema, and swollen mitochondria in the inner segments of the photoreceptors.[7]

In this patient, presumed light toxicity may be associated with SLE disease and azathioprine treatment. Manzouri et al.[8] previously reported a patient with SLE who had retinal phototoxicity which was linked to operating microscope light following cataract extraction with IOL implantation. Unlike their case; our patient was not under hydroxychloroquine treatment which has been reported to cause predisposition to the development of phototoxic retinal lesions. It has been reported that patients with SLE have increased numbers of chromosome breaks and rearrangements correlated with a low-molecular-weight chromosome damaging agent present in lymphocytes that sensitize them to near ultraviolet light (360–400 nm) light.[9]

The previous studies reported that the photosensitization efficacy of thiopurines and the correlations between pho-

totoxicity. The thiopurines (azathioprine, 6-mercaptopurine, and 6-thioguanine) act as a Type II ultraviolet A photosensitizer and induce a synergistic toxicity. Photochemical activation with initiated production of reactive oxygen species and protein oxidation; it induces DNA breakage.[10,11] In addition, thiocolchicoside has photosensitizing activity. Studies on in vitro cells demonstrated phototoxic effects on cells. There is a similar mechanism as an underlying factor for the condition. (Type I and II photosensitization pathways mediated by free radicals and singlet molecular oxygen).[12] Our patient's use of azathioprine and colchicine may have facilitated the formation of phototoxic damage. Since the previous studies were in vitro experiments and there were multiple risk factors, it is difficult to make a definitive judgment in our case.

Cellini et al.[13] reported multifocal electroretinogram findings in arc welding macular injury in a case report. In their study, they observed a reduction in the amplitude in the central on multifocal electroretinography (mfERG). This reduction has been improving overtime as confirmed by control mfERG made a month later. The previous studies confirmed the importance of mfERG in the diagnosis and the follow-up of retinal phototoxic injury.[14,15] The limitation of this report is the lack of performing mfERG, it would make an additional contribution to our case report.

Conclusion

We demonstrated that the microvascular changes (perifoveal capillary arcade disruption) in addition to atrophy of the photoreceptor and retinal pigment epithelium layers. This case report would be valuable because there has been no similar published paper reporting an association between macular phototoxicity and SLE followed by recovery with OCT-A. Multimodal imaging may be helpful in the differential diagnosis. The presence of SLE, treatment of azathioprine or colchicine, may be risk factors for phototoxic damage during eye surgery. The phototoxic injury could be aggravated with these conditions. Surgeons should be aware of the possibility of phototoxic damage in the macula in patients with SLE.

Informed Consent: Written informed consents were obtained from the parents for publication of this case report and accompanying images.

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