PREOPERATIVE VITREORETINAL INTERFACE ABNORMALITIES ON SPECTRAL DOMAIN OPTICAL COHERENCE TOMOGRAPHY AS RISK FACTOR FOR PSEUDOPHAKIC CYSTOID MACULAR EDEMA AFTER PHACOEMULSIFICATION

SERGIO COPETE, MD, PHD,* PABLO MARTÍ-RODRIGO, MD,* ROMINA MUÑIZ-VIDAL, MD, PHD,* SALVADOR PASTOR-IDOATE, MD, PHD,* JAUME RIGO, MD,† MARTA S. FIGUEROA, MD, PHD,‡§ JOSÉ GARCÍA-ARUMÍ, MD, PHD,* MIGUEL A. ZAPATA, MD, PHD*¶

Purpose: We assessed the role of vitreoretinal interface status in the development of pseudophakic cystoid macular edema (PCME) after cataract surgery.

Methods: Prospective cohort study in which 112 patients (112 eyes) scheduled for cataract surgery were selected at random to undergo spectral domain optical coherence tomography (OCT) within 1 week preoperatively and at 1 and 3 months postoperatively. Spectral domain OCT macular images included no vitreoretinal contact, focal and diffuse vitreomacular adhesion, focal and diffuse vitreomacular traction, epiretinal membrane, macular hole, and macular edema.

Results: The incidence of PCME was 11.6% (13 eyes), all of them being diagnosed at 1 month, and 7 eyes resolved at 3 months. The only risk factor for PCME was detection of nonsurgical epiretinal membrane by spectral domain OCT before phacoemulsification, being developed in 5 of 16 eyes ($\chi^2 = 0.08$, odds ratio 4.53, 95% confidence interval 1.28–16.13). Other variables such as posterior vitreous detachment, subfoveal choroidal thickness, diabetes, or hypertension were not significantly associated with PCME.

Conclusion: In this cohort, preoperative detection of epiretinal membrane by spectral domain OCT was a risk factor for PCME after cataract extraction. It is recommended to perform a spectral domain OCT before cataract surgery because the presence of an epiretinal membrane may be passed unnoticed by fundus examination.

RETINA 00:1–8, 2018

Lens opacity is the leading cause of visual loss, and it is estimated that today 20 million people are blind from this condition.[1] Cataract surgery remains the most effective way to help restore vision for those with cataracts and is the most common procedure performed by the ophthalmic surgeon worldwide. According to the World Health Organization (WHO) for the control of cataract avoidable blindness, it is necessary to increase cataract operations to 20 million in 2010 with a final target of 32 million cataract surgeries annually by the year 2020.[1] However, the foreseeable increase in cataract operations will be also associated with an increased risk of complications. Although technical advances have largely improved the final functional results, the development of postoperative complications such as postoperative macular edema still remains a potential sight-threatening problem of major concern. In a retrospective database study of 81,984 eyes undergoing cataract surgery in 8 independent United Kingdom clinical sites, a previous diagnosis of epiretinal membrane (ERM) was associated with a risk ratio of 5.60 for the development of pseudophakic macular edema.[2]

Pseudophakic cystoid macular edema (PCME), also known as Irvine–Gass syndrome, is one of the most common complications after cataract surgery, being diagnosed in 0.1% to 5% of cases with fundoscopy, reaching more than 20% when ancillary tests are used.[3,4] It can occur during the first year after surgery, but is more frequent within the first 3 months postoperatively.[5] Although it is usually a self-limiting disease, in some cases, it can persist despite treatment causing decreased visual acuity. The etiology is not yet fully understood. Inflammatory mediators and/or anterior–posterior traction induced during the surgery have been suggested as main risk factors.[2,6,7] These factors could disrupt the blood–retinal barrier, increase vascular permeability, and cause intraretinal cysts in predisposed eyes. Other conditions, such as previous uveitis, diabetes mellitus, or traumatic surgery, have also been implicated, but a definitive pathophysiologic mechanism has not been identified.[8]

Optical coherence tomography (OCT) has modified diagnoses of retinal alterations showing that vitreomacular interface abnormalities can cause vision impairment.[9] Optical coherence tomography has also revealed that macular thickness can be modified after cataract surgery, and that the presence of an ERM is more frequent in eyes with PCME. However, little is known about the role of preexisting vitreoretinal interface abnormalities in the development of PCME after cataract surgery.

We hypothesized that the occurrence of PCME after cataract surgery may be more frequent among patients with a previous abnormal vitreoretinal interface. To this purpose, a prospective study was designed to determine the relationship between the presence of vitreoretinal interface abnormalities detected by OCT before surgery and the development of PME after cataract operation.

From the *Retina Department, Service of Ophthalmology, Hospital Universitari Vall d'Hebron, Universitat Autònoma de Barcelona, Barcelona, Spain. Dr. Copete is now with the Department of Ophthalmology, Castilla-La Mancha University, Albacete, Spain; †Glaucoma Department, Service of Ophthalmology, Hospital Universitari Vall d'Hebron, Universitat Autònoma de Barcelona, Barcelona, Spain; ‡Retina Division, Department of Ophthalmology, Hospital Universitario Ramón y Cajal, Madrid, Spain; §Vissum Madrid, Madrid, Spain; and ¶Oftalis, Institut d'Oftalmologia, Clinica Girona, Girona, Spain.

None of the authors has any financial/conflicting interests to disclose.

Reprint requests: Sergio Copete, MD, PhD, Department of Ophthalmology, Facultad de Medicina de Albacete, Castilla-La Mancha University, C/Almansa 14, E-02006 Albacete, Spain; e-mail: sergiocp.ab@gmail.com

Patients and Methods

Study Design and Patients

This prospective cohort study was performed with the approval of the Ethics Committee of Hospital Universitari Vall d'Hebron (Barcelona, Spain). The study was conducted in accordance with the principles of the Declaration of Helsinki for the protection of human subjects, and written informed consent was obtained from all participants.

Patients of both sexes prospectively scheduled for routine cataract surgery over a period of 4 months (between July 1, 2016, and October 31, 2016) at the Department of Ophthalmology of an acute-care tertiary hospital in Barcelona (Spain) were selected at random (one or two patients) from the operating lists of four ophthalmologists who perform cataract surgery at our institution. Patients were selected provided they agreed to the previous condition as to come to the hospital for two extra visits in which ophthalmologic evaluation and OCT examination were performed. Optical coherence tomography scans should be of sufficiently good quality to assess the choroid. Also, in patients selected for inclusion, a further ophthalmologic visit within 1 week before surgery was added.

Inclusion criteria were age >18 years, best-corrected visual acuity (BCVA) equal or better than 20/400 Snellen (1.3 logarithm of the minimum angle of resolution [LogMAR]), and lens opacity allowing for visualization of fundus details and to obtain OCT images for assessment of vitreoretinal interface. Patients with very low vision (counting fingers) were excluded because performance of OCT may be more complex and also because macular pathology is more common. Patients with high myopia (>6.0D and/or axial length > 26 mm), history of previous intraocular inflammation, ocular surgery, or indication of treatment with intravitreal injection of antiangiogenic drugs were excluded from the study. Treatment with the prostaglandin analog, latanoprost, was not allowed. Also, patients with macular cysts, retinal vein occlusion, geographic atrophy, dense hemorrhages, subfoveal fibrosis, choroidal neovascular membrane, full-thickness macular hole, and ERM that modified the foveal profile (normal foveal profile: the one in which there was neither correction/elevation of the foveal profile nor presence of ectopic material) were excluded, as were those in which the quality of images was considered inadequate by the investigators. One eye per patient was included.

Procedures and Outcome

The study included a preoperative visit (baseline) and 3 postoperative visits at 1 week and at 1 and 3

months after cataract surgery. The baseline visit included a complete medical anamnesis and an ophthalmologic examination and spectral domain OCT (SD-OCT). History of diabetes mellitus and hypertension referred by the patient was recorded. Best-corrected visual acuity was measured as Snellen fraction (LogMAR in parenthesis). Spectral domain OCT was performed with the Cirrus HD-OCT 4000 (Carl Zeiss Meditec, Dublin, CA) under pupil dilatation. Images were captured using macular cube (512 × 512) and the HD 5 Line Raster scan protocol with enhanced depth imaging. Central macular thickness (CMT) was measured automatically, whereas subfoveal choroidal macular thickness (SFCT) was performed manually with the OCT caliper, below the fovea from the outer limit of the retinal pigment epithelium–Bruch membrane complex to the inner of the sclera at the subfoveal region. Spectral domain OCT macular images were classified into the following categories: no vitreoretinal contact visible, focal and diffuse vitreomacular adhesion (VMA), focal and diffuse vitreomacular traction (VMT), nonsurgical ERM, macular hole, and macular edema. All images were reviewed by two independent ophthalmologists (S. C. and P.M.-R.), with a third ophthalmologist (M.A. Z.) in case of discrepancy.

Complete ophthalmologic examination was performed at all postoperative visits, and SD-OCT at 1- and 3-month visits.

Standard small-incision cataract phacoemulsification with foldable intraocular lens implantation was performed in all patients. The INFINITI Vision System (Alcon, Irvine, CA) was used in all cases. The intraoperative lens implanted were Acrysof SA60AT (Alcon Laboratories, Fort Worth, TX) or AMO TECNIS-1 (Abbott Medical Optics, Santa Ana, CA) at the surgeon's discretion. Postoperative treatment included topical dexamethasone and tobramycin (Tobradex, Alcon Cusi, S. A., El Masnou, Barcelona, Spain), 5 times a day for the first week, 4 times a day for the second week, 3 times a day for the third week, and twice a day for the fourth week. Topical nepafenac was added when PME was documented at the 1-month follow-up visit and treatment was maintained for 2 months.

Vitreomacular interface abnormalities were classified according to criteria of The International Vitreomacular Traction Study Group.[10] Posterior vitreous detachment (PVD) was assessed clinically and by OCT. Nonsurgical ERM was defined when it did not affect the foveal profile or justified a decrease in visual acuity.

Statistical Analyses

Patients who completed the 1- and 3-month follow-up visits were included in the analysis. Categorical data are expressed as frequencies and percentages and continuous data as mean and SD. The chi-square (χ^2) test or Fisher's exact tests were used for the comparison of categorical variables between the groups of patients with and without preoperative vitreoretinal interface abnormalities, and the Mann–Whitney *U* test or the Wilcoxon test for the comparison of quantitative variables. Multiple logistic regression analysis was used to assess variables independently associated with development of PCME. Statistical significance was set at $P < 0.05$. Statistical analyses were performed with the Statistical Package for the Social Sciences, version 20.0 software (SPSS, Inc, Chicago, IL).

Results

Of a total of 132 patients were assessed for eligibility, 5 (3.8%) were excluded because of previous vitreoretinal surgery (n = 3) and indication of intravitreal treatment with antiangiogenic drugs (n = 2). The remaining 127 patients underwent cataract surgery. Intraoperative complications occurred in four patients, including zonular disinsertion in two, tearing of the anterior lens capsule in one, and rupture of the posterior capsule in one. Anterior vitrectomy was necessary in two patients. None of these four patients developed cystoid macular edema.

Fifteen patients (11.8%) did not complete the 1- or 3-month follow-up visits for unknown reasons and were excluded from the analysis. Therefore, the study population consisted of 112 patients (112 eyes), 40 men and 72 women, with a mean (SD) age of 74.6 (7.6) years (range 50–87 years). Baseline (preoperative) characteristics of these patients are shown in Table 1. The preoperative characteristic of the 15 patients lost to follow-up were similar to those of the patients included in the study. There were 30 patients with Type 2 diabetes, 4 of which had mild or moderate nonproliferative diabetic retinopathy. Early or intermediate age-related macular degeneration (AMD) was found in 25% of patients. Posterior vitreous detachment was present in 26 eyes (23.2%). The mean BCVA was 20/55 (20/26) (range 20/28–20/40) (0.44 [0.12] logMAR, range 0.15–1.3 logMAR). The mean CMT was 260.96 (28.02) μm (range 126–353 μm), and the mean SFCT was 225.70 (85.87) μm (range 53–455 μm).

Table 1. Baseline Characteristics of the 112 Study Patients

Variables	Number (%)	Mean (SD)
Sex		
Men	40 (35.7)	
Women	72 (64.3)	
Age, years		74.6 (7.6)
Underlying diseases		
Type 2 diabetes mellitus	30 (26.8)	
Hypertension	66 (58.9)	
Ophthalmoscopic examination (fundoscopy and SD-OCT)		
Mild or moderate NPDR	4 (3.6)	
Early or intermediate AMD	28 (25)	
Complete PVD	26 (23.2)	
BCVA, Snellen		20/55 (20/26)
BCVA, LogMAR		0.44 (0.12)
CMT, μm		260.96 (28.02)
SFCT, μm		225.70 (85.87)
Vitreoretinal interface		
Absence of vitreoretinal contact	65 (58.0)	
Focal VMA	10 (8.9)	
Diffuse VMA	18 (16.1)	
Focal VMT	1 (0.9)	
Diffuse VMT	1 (0.9)	
ERM	16 (14.3)	
Lamellar hole	1 (0.9)	

NPDR, nonproliferative diabetic retinopathy.

The incidence of PCME was 11.6% (13 eyes). In all cases, PME was diagnosed at 1 month after cataract surgery. All these patients were treated with nonsteroidal anti-inflammatory drug drops until the 3-month visit. Edema resolved in 7 eyes at 3 months after cataract surgery. The mean BCVA in patients with resolved PCME was similar to that of patients without PCME (20/35 [20/35] vs. 20/30 [20/27], P = 0.67) (0.24 [0.24] vs. 0.18 [0.14] LogMAR). By contrast, patients with unresolved PCME showed worse BCVA at baseline as compared with those without PCME (20/57 [20/33] vs. 20/55 [20/29], P = 0.7; 0.46 [0.22] vs. 0.44 [0.17] logMAR). In the overall study patients, the BCVA increased significantly from 20/55 (20/29) (0.44 [0.17] logMAR) at baseline to 20/30 (20/27) (0.18 [0.14] logMAR) at 3 months after cataract surgery ($P < 0.05$). However, BCVA decreased in six eyes (5.4%), three of them with persistent PME, two with AMD, and one with ERM and a thick CMT at baseline (351 μm) but with normal foveal profile.

The distribution of the study variables at baseline and at the 3-month follow-up visit according to the presence or absence of PCME is shown in Table 2. The incidence of PCME was higher among patients with diabetes, but the difference was not significant (38.4% vs. 25.2%). Of the five patients with diabetes and PCME, resolution was recorded in two patients (40%), whereas among eight patients without diabetes and PCME, resolution was observed in five patients (62.5%). Two of the four patients with nonproliferative diabetic retinopathy showed PCME. The occurrence of PCME was unrelated to the presence of hypertension, AMD, previous vitreous status, or development of PVD at follow-up (Table 2).

The mean CMT was 260.96 (28.02) at baseline and increases to 282.34 (65.32) at 1 month and to 275.38 (42.14) at 3 months after cataract surgery ($P < 0.05$). Patients with PCME as compared to those without PCME showed a significantly thicker retina at baseline (279.31 [32.02] vs. 258.55 [26.71] μm, P = 0.045) and at 3 months after cataract surgery (344.15 [71.11] vs. 266.35 [27.92] μm, $P < 0.001$), even in eyes with PCME resolution in which CMT at 2 months was 305.14 (36.1) (P = 0.001).

Subfoveal choroidal macular thickness was measurable in 100 eyes (89.3%), and significant changes at 1 month and 3 months after cataract surgery as compared with baseline were not found. Changes according to the presence or absence of PME were not found either (Figure 1). Development of PCME was neither more frequent when the choroidal–scleral interface was not visible.

In relation to vitreoretinal interface status, there was only one case of discrepancy between the two independent ophthalmologists, one established a diagnosis

Table 2. Distribution of the Study Variables in Patients With and Without PCME

Variables	Pseudophakic Macular Edema (PME)		P
	Absent (n = 99)	Present (n = 13)	
Age, years, mean (SD)	74.5 (7.9)	76.2 (5.0)	0.59
Type 2 diabetes mellitus, %	25.2	38.4	0.31
NPDR, %	8	40	0.055
Hypertension, %	58.6	61.5	0.84
AMD, %	27.2	7.7	0.42
PVD at baseline, %	23.2	23.1	0.99
PVD at follow-up, %	12.1	15.4	0.74
BCVA, Snellen, mean (SD)			
Baseline	20/55 (20/29)	20/59 (20/34)	0.78
At 3 months	20/30 (20/27)	20/43 (20/35)	0.25
BCVA, logMAR, mean (SD)			
Baseline	0.44 (0.17)	0.47 (0.24)	0.78
At 3 months	0.18 (0.14)	0.34 (0.25)	0.25
CMT, μm, mean (SD)			
Baseline	258.55 (26.71)	279.31 (32.02)	0.045
At 3 months	266.35 (27.92)	344.15 (71.11)	<0.01
SFCT, μm, mean (SD)			
Baseline	223.64 (84.79)	220.77 (103.03)	0.74
At 3 months	223.67 (82.96)	224.77 (119.75)	0.88
Vitreoretinal interface at baseline, no. (%)			
Absence of vitreoretinal contact, n = 65	59 (90.8)	6 (9.2)	—
Focal VMA, n = 10	9 (90)	1 (10)	—
Diffuse VMA, n = 18	18 (100)	0	—
Focal VMT, n = 1	1 (100)	0	—
Diffuse VMT, n = 1	0	1 (100)	—
ERM, n = 16	11 (68.7)	5 (31.2)	—
Lamellar hole, n = 1	1 (100)	0	—

NPDR, nonproliferative diabetic retinopathy.

of VMT and the other established a diagnosis of ERM (degree of agreement, kappa 0.98). The third ophthalmologist confirmed the diagnosis of ERM. No visible vitreoretinal contact was detected in 65 eyes (58%) at baseline. Focal VMA was observed in 8.9% of eyes, diffuse VMA in 16.1%, focal and diffuse VMT in 0.9% each, ERM in 14.3%, and lamellar hole in 0.9%. Previous VMA or VMT detected by SD-OCT was not associated with the development of PCME. The presence of nonsurgical ERM at baseline was the only factor associated with PCME, being developed in 5 of 16 eyes (31.2%) as compared with 2 of 49 eyes without ERM (4.1%) (χ^2 = 0.08, odds ratio = 4.53, 95% confidence interval 1.28–16.13).

Discussion

This prospective cohort study was performed to identify risk factors for the development of PCME after cataract surgery.[9,11] Identification of a nonsurgical ERM on preoperative SD-OCT was the only variable associated with PCME. Inflammatory, vascular,[12–14] and mechanical[15] factors have been implicated in the appearance of macular edema after cataract extraction, but the precise etiology remains unclear. Anterior segment manipulation during cataract surgery or changes induced in the vitreous after lens removal could induce alterations in the retina and cause edema in the posterior pole. Increased inflammatory mediators in the vitreous after cataract surgery, such as prostaglandins and leukotrienes, may disrupt the blood–retinal barrier, resulting in dilatation of parafoveal retinal capillaries, increased permeability, changes in the inner retina,[16] and formation of cystic spaces in the central macula.[17] In fact, the role of triamcinolone and dexamethasone in the treatment of PCME is based mainly on their inhibition of the biosynthetic pathways of leukotrienes and prostaglandins and stabilization of the blood–retinal barrier.[18–21] However, the role of vitreous has not been elucidated, although vitreoretinal interface has been suggested to play an important role in the onset of Irvine–Gass syndrome.[8] In this study, however, previous PVD or vitreoretinal contact did not seem to be a risk factor for PCME. This may indicate that the development of cystoid macular edema may

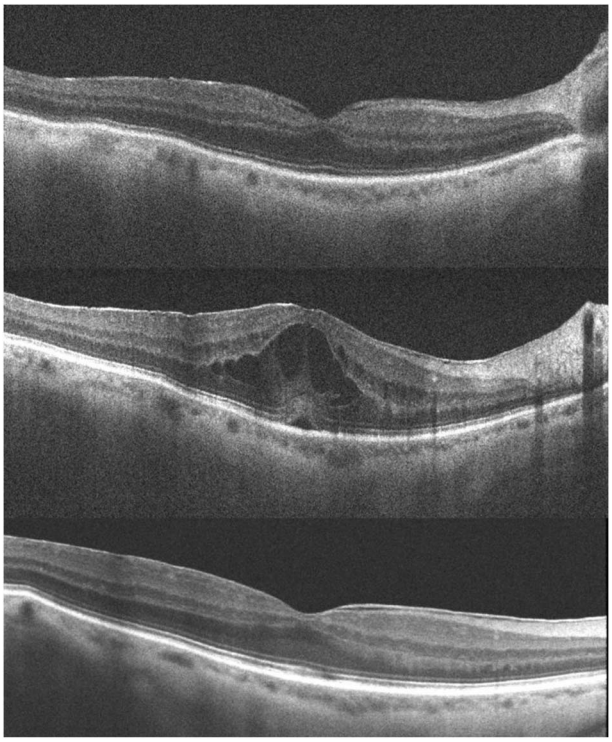

Fig. 1. Optical coherence tomography image showing ERM. Central macular thickness was 346 μm at baseline (top), but foveal profile and external retina were preserved. This patient developed PCME (middle) that resolved at 3 months (bottom).

be influenced by other factors besides status of the vitreoretinal interface.

In agreement with data reported by others,[3,11,22] mean CMT showed a statistically significant increase at 1 month after cataract extraction, which was maintained after 3 months, pointing out a possible leakage. Also, baseline CMT was thicker in eyes developing PCME, suggesting that increased CMT thickness may be a predisposing factor for PCME or the presence of subclinical PCME, which may not be detected by current OCT imaging systems. Inflammatory mediators may increase vascular permeability leading to an increase in macular thickness and cyst formation in eyes with predisposing conditions or previous retinal vascular disease.

A history of ERM has been associated with an increased risk of PCME.[2,3] In a retrospective database study of electronic medical records of 81,984 eyes undergoing cataract surgery, a previous diagnosis of ERM showed a relative risk of 5.60 (95% confidence interval 3.45–9.07) for PCME after cataract surgery.[4] In this retrospective database study, however, OCT was not routinely performed before or after surgery, so that the incidence of ERM could have been underreported. In our study, the presence of ERM in the macular area with no foveal alterations was the only risk factor for PCME and may implicate a previous subclinical damage or increased predisposition to both conditions. It has been estimated that subclinical ERM as a predisposing factor to the onset of PCME can be present in 2% to 35% of cases,[8,23,24] with a higher frequency in diabetics versus nondiabetics, as well as in eyes with diabetic retinopathy and AMD.[4,25–29] We also found a higher occurrence of PCME in patients with diabetes, particularly when diabetic retinopathy was present, but a significant association was not observed.

Intraoperative complications such as posterior capsule rupture, vitreous loss, and lens material luxation are well-known risk factors for PCME,[4] although the incidence of these complications have decreased in the past years.[28–30] In the present series, anterior vitrectomy was necessary in 2 eyes, but in none of them cystoid macular edema has developed at 3 months after surgery.

Treatment of PCME also remains controversial because macular edema may resolve spontaneously. However, there is evidence that topical nonsteroidal anti-inflammatory drugs are more effective than topical corticosteroids in preventing PCME after cataract surgery,[31] although differences in final BCVA have not been observed. In our patients, preoperative treatment was not prescribed. Postoperatively, patients were treated with a combination of tobramycin and dexamethasone, which was tapered in 1 month. In the 13 patients in whom PCME was documented at 1-month visit, topical nonsteroidal anti-inflammatory drug drops were administered, with resolution of macular edema in 7 (53.8%). In these patients, the mean BCVA was similar to those without PCME, although CMT was higher. In the three eyes without resolution of macular edema, BCVA decreased as compared with baseline, which may suggest that persistence of edema may be more relevant than its development.

It has been shown that SFCT increase after cataract surgery and changes in SFCT are greater in patients with PCME and preceded the increase in central retinal thickness.[32] In our study, significant changes in SFCT were not observed in the overall study patients and in those with PCME. The reason for this lack of association is unknown. Also, PCME was neither more frequent in eyes in which SFCT could not have been measured because choroidal–scleral interface was not visible. However, swept-source OCT is superior to SD-OCT for measurement of choroidal thickness.[33]

In our center, patients are scheduled for operation according to their order of inclusion in the waiting lists. The first or the second patient in the list who fulfilled the inclusion criteria and agreed to follow the study protocol was selected. Patients were

selected at random from the lists of four staff ophthalmologists who perform cataract surgery at our institution. The selection of more than one or two surgeons aimed at achieving conditions that are more similar to the real-world setting. Also, although more patients could have been included for each day of surgery, this limited number ensured the proper quality of care necessary for the study. Limitations of the study include the relatively small number of patients and the fact that other perioperative risk factors, such as phacoemulsification time, were not assessed. Also, 15 patients were lost to follow-up and were excluded from the analysis, but their preoperative characteristics were similar to the overall study population of 112 patients who completed the 2 follow-up visits. When advanced cataracts are present, quality of images of vitreoretinal interface by SD-OCT may be suboptimal. However, poor image quality was an exclusion criterion in our study. Other limitations include the fact that fluorescein angiography was not performed in the presence of postoperative edema and that exact SFCT measurement was not possible in all cases.

In conclusion, in the present cohort, preoperative detection of a nonsurgical ERM by SD-OCT was a risk factor for PCME after cataract extraction. It is recommended to perform a SD-OCT before cataract surgery because the presence of an ERM may be passed unnoticed by standard ophthalmologic examination. Pretreatment with topical nonsteroidal anti-inflammatory drug can be advocated for patients with ERM on preoperative OCT scans. Further studies, however, are needed to confirm the impact of vitreoretinal interface abnormalities on the development of postsurgical macular edema.

Key words: epiretinal membrane, phacoemulsification, pseudophakic macular edema, spectral domain OCT, vitreomacular adhesion, vitreomacular traction, vitreoretinal interface abnormalities.

Acknowledgments

The authors thank Marta Pulido, MD, for editing the manuscript and for her editorial assistance.

References

1. Blindness: VISION 2020—The Global Initiative for Elimination of Avoidable Blindness. World Health Organization. Available at: http://www.who.int/mediacentre/factsheets/fs213/en/. Accessed April 14, 2017.
2. Chu CJ, Johnston RL, Buscombe C, et al. Risk factors and incidence of macular edema after cataract surgery: a database study of 81984 eyes. Ophthalmology 2016;123:316–323.
3. Lobo CL, Faria PM, Soares MA, et al. Macular alterations after small-incision cataract surgery. J Cataract Refract Surg 2004; 30:752–760.
4. Bélair ML, Kim SJ, Thorne JE, et al. Incidence of cystoid macular edema after cataract surgery in patients with and without uveitis using optical coherence tomography. Am J Ophthalmol 2009;148:128–135.e2.
5. Henderson BA, Kim JY, Ament CS, et al. Clinical pseudophakic cystoid macular edema. Risk factors for development and duration after treatment. J Cataract Refract Surg 2007;33: 1550–1558.
6. Grzybowski A, Sikorski B, Ascaso F, Huerva V. Pseudophakic cystoid macular edema: update 2016. Clin Interv Aging 2016; 11:1221–1229.
7. Loewenstein A, Zur D. Postsurgical cystoid macular edema. Dev Ophthalmol 2010;47:148–159.
8. Framme C, Wolf S. Retinal complications after damaging the vitreolenticular barrier. Ophthalmologica 2011;227:20–33.
9. Meuer SM, Myers CE, Klein BEK, et al. The epidemiology of vitreoretinal interface abnormalities as detected by spectral-domain optical coherence tomography: the Beaver Dam Eye Study. Ophthalmology 2015;122:787–795.
10. Duker JS, Kaiser PK, Binder S, et al. The International Vitreomacular Traction Study Group classification of vitreomacular adhesion, traction, and macular hole. Ophthalmology 2013; 120:2611–2619.
11. Anastasilakis K, Mourgela A, Symeonidis C, et al. Macular edema after uncomplicated cataract surgery: a role for phacoemulsification energy and vitreoretinal interface status? Eur J Ophthalmol 2014;25:192–197.
12. Rossetti L, Autelitano A. Cystoid macular edema following cataract surgery. Curr Opin Ophthalmol 2000;11:65–72.
13. Miyake K, Ibaraki N. Prostaglandins and cystoid macular edema. Surv Ophthalmol 2002;47:S203–S218.
14. Neal RE, Bettelheim FA, Lin C, et al. Alterations in human vitreous humour following cataract extraction. Exp Eye Res 2005;80:337–347.
15. Schubert HD. Cystoid macular edema: the apparent role of mechanical factors. Prog Clin Biol Res 1989;312:277–291.
16. Sigler EJ, Randolph JC, Kiernan DF. Longitudinal analysis of the structural pattern of pseudophakic cystoid macular edema using multimodal imaging. Graefes Arch Clin Exp Ophthalmol 2016;254:43–51.
17. Munk MR, Jampol LM, Simader C, et al. Differentiation of diabetic macular edema from pseudophakic cystoid macular edema by spectral-domain optical coherence tomography. Invest Ophthalmol Vis Sci 2015;56:6724–6733.
18. Conway MD, Canakis C, Livir-Rallatos C, Peyman GA. Intravitreal triamcinolone acetonide for refractory chronic pseudophakic cystoid macular edema. J Cataract Refract Surg 2003; 29:27–33.
19. Yüksel B, Uzunel UD, Kerci SG, et al. Comparison of subtenon triamcinolone acetonide injection with topical nepafenac for the treatment of pseudophakic cystoid macular Edema. Ocul Immunol Inflamm 2016;25:1–7.
20. Medeiros MD, Navarro R, Garcia-Arumí J, et al. Dexamethasone intravitreal implant for treatment of patients with recalcitrant macular edema resulting from Irvine-Gass syndrome. Invest Ophthalmol Vis Sci 2013;54: 3320–3324.

21. Takkar B. Dexamethasone implant as an effective treatment option for macular edema in Irvine-Gass syndrome. J Cataract Refract Surg 2016;42:648.
22. Şahin M, Cingü AK, Gözüm N. Evaluation of cystoid macular edema using optical coherence tomography and fundus autofluorescence after uncomplicated phacoemulsification surgery. J Ophthalmol 2013;2013:376013.
23. Zapata MA, Figueroa MS, Esteban González E, et al. Prevalence of vitreoretinal interface abnormalities on spectral-domain optical coherence tomography in healthy participants over 45 years of age. Ophthalmol Retina 2016;1:249–254.
24. Liu L, Yue S, Wu J, et al. The prevalence and distribution of vitreoretinal interface abnormalities among urban community population in China. J Ophthalmol 2015;2015:742686.
25. Schmier JK, Halpern MT, Covert DW, Matthews GP. Evaluation of costs for cystoid macular edema among patients after cataract surgery. Retina 2007;27:621–628.
26. Baker CW, Almukhtar T, Bressler NM, et al. Macular edema after cataract surgery in eyes without preoperative central-involved diabetic macular edema. JAMA Ophthalmol 2013; 131:870–879.
27. Jiramongkolchai K, Lalezary M, Kim S. Influence of previous vitrectomy on incidence of macular oedema after cataract surgery in diabetic eyes. Br J Ophthalmol 2011;95:524–529.
28. Johnston RL, Taylor H, Smith R, Sparrow JM. The Cataract National Dataset electronic multi-centre audit of 55,567 operations: variation in posterior capsule rupture rates between surgeons. Eye (Lond) 2010;24:888–893.
29. Ti SE, Yang YN, Lang SS, Chee SP. A 5-year audit of cataract surgery outcomes after posterior capsule rupture and risk factors affecting visual acuity. Am J Ophthalmol 2014; 157:180–185.e1.
30. Day AC, Donachie PHJ, Sparrow JM, Johnston RL. The Royal College of Ophthalmologists' National Ophthalmology Database study of cataract surgery: report 1, visual outcomes and complications. Eye (Lond) 2015;29:552–560.
31. Kessel L, Tendal B, Jørgensen KJ, et al. Post-cataract prevention of inflammation and macular edema by steroid and non-steroidal anti-inflammatory eye drops: a systematic review. Ophthalmology 2014;121:1915–1924.
32. Pierru A, Carles M, Gastaud P, Baillif S. Measurement of subfoveal choroidal thickness after cataract surgery in enhanced depth imaging optical coherence tomography. Invest Ophthalmol Vis Sci 2014;55:4967–4974.
33. Copete S, Flores-Moreno I, Montero JA, et al. Direct comparison of spectral-domain and swept-source OCT in the measurement of choroidal thickness in normal eyes. Br J Ophthalmol 2014;98:334–338.