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Original Article

Evaluation of Changes of Macular Thickness in Diabetic Retinopathy after Cataract Surgery

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Purpose: To assess the macular thickness changes after cataract surgery in diabetic patients using optical coherence tomography (OCT).

Methods: We retrospectively reviewed the records of 104 diabetic patients who underwent cataract surgery. We examined the changes of macular thickness using OCT before cataract surgery and 1 week, 1-, 2- and 6-months after surgery. The central subfield mean thickness (CSMT) was used to evaluate macular edema which was defined as an increase of CSMT (ΔCSMT) > 30% from the baseline. The association between prior laser treatment or severity of diabetic retinopathy and macular thickness were also analyzed.

Results: Macular edema occurred in 19 eyes (18%) from the diabetic group and 63% of macular edema developed at 1 month after surgery. Thirteen (68%) out of 19 eyes with macular edema showed the resolution of macular edema by 6 months after surgery without treatment. ΔCSMT of eyes without a history of laser treatment was statistically greater compared to eyes with a history of laser treatment in at 1- and 2-months after surgery, but was not different than eyes who had laser treatment at 6-months after surgery. The severity of diabetic retinopathy was not significantly correlated to macular edema, but there was statistical difference when patients who had a history of prior laser treatment were excluded.

Conclusions: The incidence of macular edema after cataract surgery in diabetic patients was 18%. Its peak incidence was at 1 month post surgery and it resolved spontaneously in 68% of patients by 6 months post surgery. Prior laser treatment might prevent postoperative macular edema until 2 months after cataract surgery in diabetic patients. However, macular edema did not affect the severity of diabetic retinopathy.

Key Words: Cataract extraction, Diabetic retinopathy, Macular edema, Macular thickness

Macular edema is a common cause of unfavorable visual outcome after cataract surgery. Clinically significant cystoid macular edema (CSME) has a reported incidence of 1% to 2% after cataract surgery [1]. Diabetes has been associated with an increased incidence of postoperative macular edema. The incidence of macular edema on optical coherence tomography (OCT) was 22% in diabetic eyes undergoing cataract surgery [2]. The macular edema after cataract surgery in diabetic patients could be caused by the cataract surgery or diabetes itself, but it might be hard to differentiate between these two causes.

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OCT has been shown to be highly reproducible in measuring macular thickness in normal individuals and diabetic patients [3,4]. For detecting macular edema, OCT is superior to contact lens biomicroscopy and as effective as fluorescein angiography (FAG) [5]. Since OCT can assess macular thickness quantitatively, it can detect subtle changes of macular thickness and is especially useful in mild cases.

In a previous report, diabetic eyes with CSME at the time of cataract surgery showed persistent CSME throughout the first postoperative year and showed no tendency to resolve spontaneously [6]. For diabetic eyes without CSME at the time of cataract surgery, macular edema after cataract surgery resolved spontaneously in 50% of cases by 6 months and in 75% of cases by 1 year. However, a relatively small number of patients (n = 32) were enrolled in this study and it would be even smaller if the eyes with CSME at the time of surgery were excluded. Therefore, this is not enough evidence to determine the natural history of macular edema after

cataract surgery. Moreover, the macular edema was assessed by FAG in this study and it is difficult to detect subtle changes of macular thickness using FAG.

In this study, we assessed the changes of macular thickness in diabetic patients after cataract surgery using OCT and also examined the influence of preoperative laser treatment and severity of diabetic retinopathy on macular thickness.

Materials and Methods

We reviewed the records of the patients with type 2 diabetes mellitus who underwent phacoemulsification with intraocular lens insertion between January 2008 and December 2008. A total of 104 eyes from 104 subjects were enrolled. Patients were excluded from the study if they had any of the following conditions: prior or concomitant surgery to the eye scheduled for cataract extraction such as vitreous surgery or glaucoma surgery, intraoperative complications, a history of macular edema within 6 months before the preoperative exam, a history of ocular and systemic conditions known to be associated with potentially irreversible significant vision loss, presence of any retinal or choroidal disease other than diabetes that could affect retinal thickness, and less than 6 months of follow-up care. Meanwhile, we included patients who had undergone laser photocoagulation for macular edema or for proliferative diabetic retinopathy (PDR) more than 6 months prior to surgery. Cataract surgery was performed by one single surgeon (IWP). A limbal incision or clear corneal incision was made, and continuous curvilinear capsulorrhexis was performed. For cataract surgery, phacoemulsification equipment (Legacy; Alcon Laboratories Inc., Fort Worth, TX, USA) was used. The nucleus was divided and phacoemulsification and aspiration were performed after cortical aspiration, an acrylic foldable IOL (Sensar AR40e; AMO, Santa Ana, CA, USA) was inserted in the capsular bag. Preoperative and postoperative examinations including best-corrected visual acuity using standard Korean visual acuity chart (Han's chart), which was translated into a logarithm of the minimal angle of resolution (logMAR) scale for analysis purposes, slit-lamp examination, fundus photography and intraocular lens implantation were checked at every follow-up visit. All patients were followed at 1 week, 1-, 2-, 3- and 6-months after surgery.

OCT was performed using commercially available equipment (software 3.0; Carl Zeiss Meditec, Dublin, CA, USA). After pupil dilation, a macular thickness map was generated. This standard OCT protocol uses six radially oriented scan lines to produce a topographical map of the macula and the mean value of 128 thickness values obtained in the central subfield, which is circular area of diameter 1 mm centered around the center point (central subfield mean thickness), was measured [7]. Central subfield mean thickness (CSMT) was evaluated every follow-up and we manually calculated the difference in the thickness (absolute change in thickness; ΔCSMT) from the baseline. We used the ΔCSMT to analyze the course of the macular edema because of the wide range of normal macular thickness. Macular edema was defined as 30% or more increase of CSMT from preoperative thickness determined by OCT [2]. In addition, the influence of prior laser treatment and severity of diabetic retinopathy on macular thickness was also analyzed. Study participants were divided based on the presence or absence of a history of photocoagulation (panretinal photocoagulation, focal or grid laser) and divided into 5 groups based on the severity of diabetic retinopathy (no diabetic retinopathy, mild-, moderate-, severe non-proliferative diabetic retinopathy [NPDR], and PDR). The absolute change in macular thickness and the change of visual acuity were calculated at every follow-up after surgery and analyzed among comparative groups.

Statistical analysis was performed by SPSS ver. 12.0 (SPSS Inc., Chicago, IL, USA). Descriptive statistics, including mean and standard deviation were calculated for each group. Group comparisons were performed using the student *t*-test. Fisher's exact, two-tailed test was used for analysis of data on a nominal scale. A p-value less than 0.05 was considered statistically significant.

Results

The 104 subjects in this series were made up of 36 men and 68 women and their mean age was 68.21 ± 9.09 years. Mean preoperative best-corrected visual acuity in logMAR units was 0.57 ± 0.47 and mean preoperative CSMT was 164.42 ± 25.02 μm. The number of patients without diabetic retinopathy were 61 (58.65%), with mild to moderate NPDR were 27 (25.96%), with severe NPDR or PDR were 16 (15.36%). Postoperative complications occurred in 39 eyes (37.5%).

Table 1. Demographics

Demographics	
Eyes (n)	104
Sex (male:female)	36:68
Age (yr)	68.21 ± 9.09
SE (diopter)	1.25 ± 0.68
Preoperative BCVA (logMAR)	0.57 ± 0.47
Preoperative CSMT	164.42 ± 25.02
Diabetic retinopathy stage	
No diabetic retinopathy	61 (58.65%)
Mild to moderate NPDR	27 (25.96%)
Severe NPDR or PDR	16 (15.38%)
Postoperative complications	
Macular edema	19 (18.27%)
IOP elevation	5 (4.81%)
Vitreous hemorrhage	2 (1.92%)
Posterior capsular opacity	1 (0.96%)
Retinopathy progression	12 (11.54%)

BCVA = best corrected visual acuity; logMAR = logarithm of the minimal angle of resolution; CSMT = central subfield macular thickness; NPDR = non proliferative diabetic retinopathy; PDR = proliferative diabetic retinopathy; IOP = intraocular pressure.

The most common complication was macular edema, which were occurred in 19 eyes (18.27%), followed by retinopathy progression (11.54%), elevation of intraocular pressure, vitreous hemorrhage, and posterior capsular opacity (Table 1).

Among the 19 eyes with macular edema, 12 eyes (63%) developed macular edema at 1 month after surgery, and 13 eyes (68%) showed improvement of macular edema by 6 months after surgery (Fig. 1). The remaining 6 eyes did not improve by 6 months and were followed for more than 1 year. Three eyes improved macular edema at 7, 8 and 12 months, 2 eyes were treated with intravitreal triamcinolone acetonide at 12 months, and 1 patient did not visit at 1 year after surgery.

We explored the changes of macular thickness (*Δ*CSMT) using OCT. Because the laser photocoagulation could affect on macular thickness, the diabetic patients were divided into a laser group, which were patients who had a history of any photocoagulation, and a non-laser group, which were patients who had not had photocoagulation. Of the 104 diabetic patients, 14 had a history of photocoagulation (12 with panretinal photocoagulation, 2 with focal laser). There were no statistical differences in baseline characteristics except the severity of diabetic retinopathy (Table 2). After cataract surgery, it seemed that the incidence of macular edema and progression of retinopathy increased in the non-laser group compared to the laser group, although it was not statistically significant. Compared to the laser group, *Δ*CSMT of the non-laser group was statistically significant at 1 and 2 months after surgery. *Δ*CSMT was greatest at 1 month post surgery and gradually decreased in both groups thereafter (Fig. 2A). Similarly, *Δ*CSMT of the eye with macular edema (19 eyes) was greatest at 1 month after surgery and gradually decreased by 6 months, but was still significantly increased compared to the eyes without macular edema (Fig. 2B).

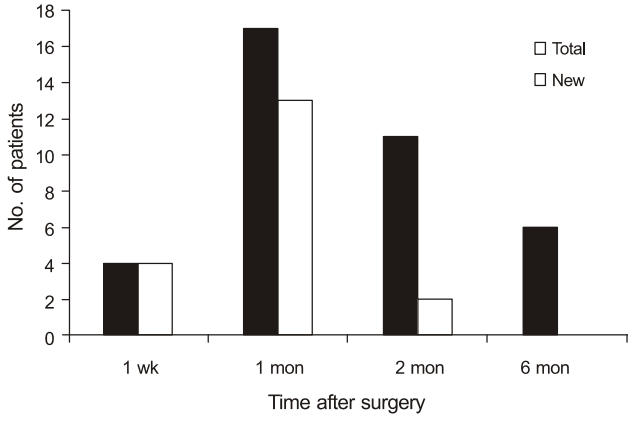

Fig. 1. The total and newly developed number of patients with macula edema after cataract surgery.

The changes of visual acuity (*Δ*logMAR), which was not statistically significant between laser and non-laser groups, constantly improved in both groups (Fig. 3A). The visual acuity of the eyes without macular edema constantly improved for 6 months after surgery. However, the visual acuity of the eyes with macular edema showed little improvement until 1 month after surgery and only improved after 2 months post surgery (Fig. 3B). Fifteen (79%) out of 19 patients with macular edema improved more than 0.1 logMAR, and 10 patients (53%) achieved logMAR of 0.1 or less (≥20 / 20) at 6 months after surgery.

Severity of diabetic retinopathy was not significantly correlated to macular edema ($p = 0.116$). Since the laser treatment could affect macular thickness and confuse the interpretation of the results, we re-evaluated the correlation between severity of diabetic retinopathy and changes of macular thickness after patients who had prior laser treatment were excluded. We found that there was statistical difference in macular thickness through severity of diabetic retinopathy after exclusion of these patients ($p = 0.017$).

Table 2. Characteristics of diabetic patients according to the history of laser treatment

	Non-laser group	Laser group	*p*-value
Demographics			
Eyes (n)	90	14	
Sex (male:female)	31:59	5:9	
Age (yr)	69.46 ± 8.41	60.21 ± 9.54	>0.05*
SE (diopter)	1.25 ± 0.76	1.22 ± 0.52	>0.05*
Preop BCVA	0.56 ± 0.47	0.66 ± 0.4	>0.05*
Preop CSMT	163.16 ± 26.09	172.50 ± 23.43	>0.05*
Incidence of macular edema	18 (20%)	1 (7.14%)	0.457†
Diabetic retinopathy stage			
No diabetic retinopathy	61 (67.78%)	0 (0%)	
Mild to moderate NPDR	25 (27.78%)	2 (14.29%)	
Severe NPDR or PDR	4 (4.44%)	12 (85.71%)	
Retinopathy progression	11 (12.22%)	1 (7.14%)	0.699†

BCVA = best corrected visual acuity; CSMT = central subfield macular thickness; NPDR = non proliferative diabetic retinopathy; PDR = proliferative diabetic retinopathy.
*Student *t*-test ($p < 0.05$).
†Fisher's exact test ($p < 0.05$).

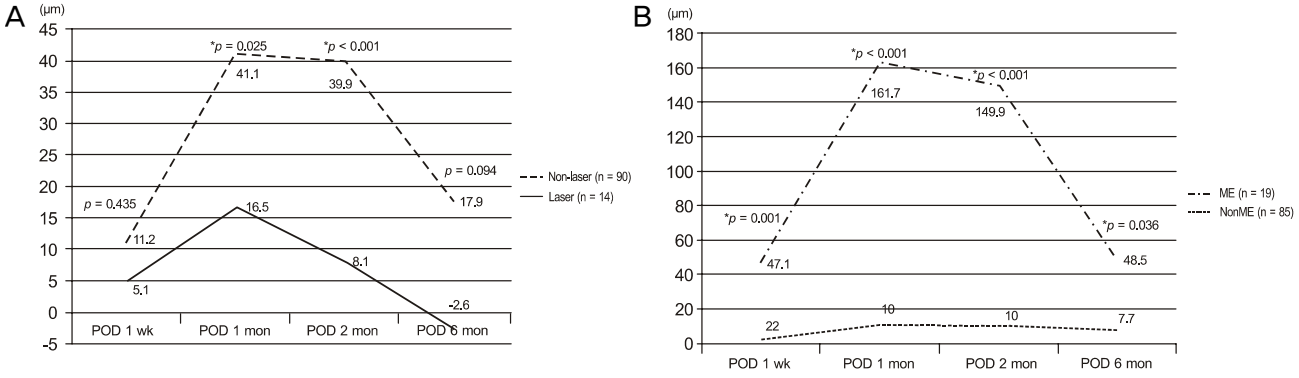

Fig. 2. The changes of central subfield macular thickness in eyes with and without prior laser treatment (A) and in eyes with and without macular edema (B) after cataract surgery. POD = postoperative day; ME = macular edema. *Student *t*-test ($p < 0.05$).

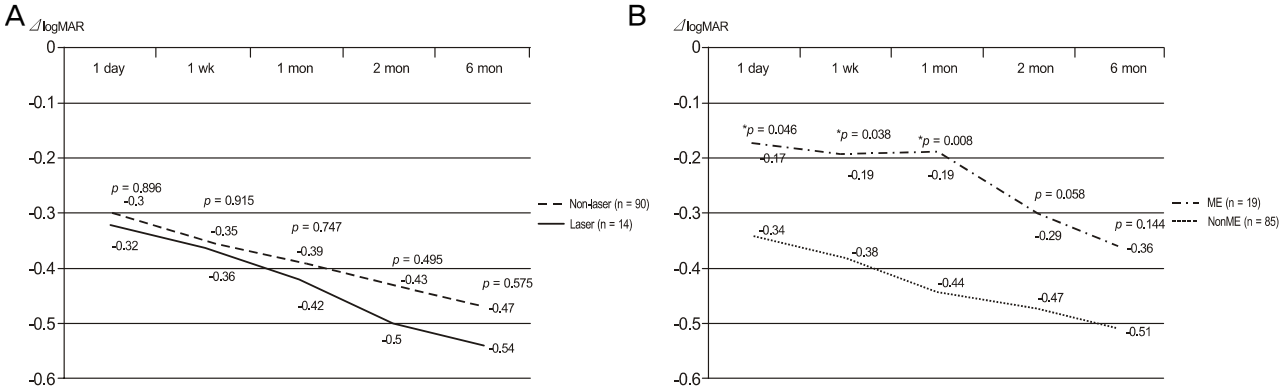

Fig. 3. The changes of best corrected visual acuity in eyes with and without prior laser treatment (A) and in eyes with and without macular edema (B) after cataract surgery. logMAR = logarithm of the minimal angle of resolution; ME = macular edema. *Student *t*-test ($p < 0.05$).

Discussion

Macular edema after cataract surgery in diabetic patients could be caused by the cataract surgery (Irvine-Gass syndrome) or diabetes itself. Most of the macular edema after cataract surgery resolves spontaneously by 6 months, but diabetic macular edema tends to persist [8,9]. Therefore, it is recommended that laser therapy for macular edema be deferred until 6 months after surgery to permit resolution of the Irvine-Gass element [9]. Dowler et al. [6] reported that CSME arising after surgery commonly resolved, particularly if retinopathy was mild. CSME occurred in half of the eyes in which CSME was absent at the time of surgery, with a peak incidence at 6 weeks. It resolved spontaneously by 6 months in half of the eyes affected and by 1 year in three quarters of the affected eyes. In our study, macular edema occurred in 19 eyes (18%) and improved spontaneously by 6 months in 68% of eyes. Its peak incidence was at 1 month post surgery, which is similar compared to previous reports. However, the incidence of macular edema appears to be rather low compared to previous studies, which reported incidence rates of 29% to 56% and the proportion of improvement is slightly higher [6,10,11]. One explanation for this result might be that patients with previous laser treatment such as panretinal photocoagulation or laser treatment for macular edema were not excluded in our study. Early Treatment Diabetic Retinopathy Study (ETDRS) [12] reported that prior photocoagulation might be the reason for relatively good visual results and photocoagulation in patients with severe NPDR or CSME should be considered before lens surgery. They also recommended that photocoagulation be considered before lens surgery in patients with severe NPDR or CSME when possible [13].

The eyes without a history of laser therapy showed a statistically significant increase in CSMT at 1 and 2 months after surgery compared to eyes with previous laser therapy. However, there was no statistical difference between the two groups at 6 months post surgery. From these results we could assume that prior laser treatment could lower the risk for early postoperative macular edema until 2 months after surgery, but there would be little influence of the previous laser therapy on macular thickness 6 months post surgery. ETDRS [10] reported that they did not observe an increased rate of macular edema after lens surgery. It is possible that the photocoagulation performed before surgery may have had a protective effect on the development of postoperative macular edema [14]. However, they assessed the rates of macular edema at 4-months after surgery. Thus, an accelerated rate of

macular edema development immediately after surgery cannot be ruled out. We presumed that early postoperative macular edema is due mostly to the Irvine-Gass component.

On the contrary, the improvement of visual acuity was not related to the history of laser treatment and the visual acuity improved even in eyes with macular edema at 6 months after surgery. There was no significant difference in visual acuity between the eyes with and without macular edema after 2 months post surgery, although macular edema still remained as seen in the OCT. This result suggests that macular edema could be managed conservatively in diabetic patients.

In conclusion, this study demonstrated that 18% of diabetic patients developed increases in CSMT > 30% after cataract surgery. Its peak incidence was at 1 month post surgery and it resolved spontaneously in 68% of patients by 6 months post surgery. This suggests that macular edema after cataract surgery in diabetic patients could be managed conservatively because it is likely to improve without treatment by 6 months post surgery, although prior laser treatment could lower the risk of early postoperative macular edema.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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