Original Article

Quantitative Analysis of Macular Thickness following Open Globe Injury in Subjects with Clear Media and no Retinal Damage

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Abstract

Purpose: To evaluate the change in macular thickness after open globe injury (OGI) in patients with clear media and without retinal damage using optical coherence tomography (OCT).

Methods: In this longitudinal observational pilot study, 17 patients with clear media and without retinal damage who underwent repair of OGI for corneal, corneoscleral, or scleral laceration were studied. In addition to routine follow-up, all patients were examined at the first and third postoperative months and best corrected visual acuity (BCVA), slit-lamp examination, applanation tonometry, dilated fundus examinations, and macular OCT scans were documented.

Results: In all patients, no signs of macular edema, macular thickening, cystic changes, or other signs of cystoid macular edema were present in OCT images and examinations. The Early Treatment Diabetic Retinopathy Study map indicated that there were no significant differences in macular thickness between the first and third months in all patients ($P > 0.05$). There was no significant relationship between macular thickness and uveal or vitreous prolapse and the size or site of laceration ($P > 0.05$). BCVA in the first and third months also showed no significant change ($P > 0.05$). There were no cases of intraocular pressure increase in any of the patients.

Conclusion: Macular thickness had no significant change following OGI repair in eyes with clear media and without retinal damage; thus, it seems that OGI of this extent and its surgical repair have no effect on macular thickness.

Keywords: Corneal Laceration Repair; Cystoid Macular Edema; Macular Thickness; Open Globe Injury; Optical Coherence Tomography; Scleral Laceration Repair

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INTRODUCTION

Open globe injury (OGI) is defined as full thickness injury to the cornea, sclera, or both that ranges from small, penetrating, self-sealing pin pricks to globe rupture with total disorganization and prolapse of the intraocular contents.[1] OGI has an incidence of 200,000 per year worldwide and is a major cause of permanent visual impairment and blindness.[2]

Cystoid macular edema (CME) is characterized by thickening of the macula associated with the accumulation of fluid in the extracellular space of the neurosensory

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retina.[3-6] Normally, the blood-retinal barrier (BRB) prevents the accumulation of fluid in the retinal extracellular space, but intraretinal fluid can accumulate when the BRB is damaged.[3-5] Inflammatory mediators are hypothesized to trigger the breakdown of the BRB.[7,8] Muller cells are thought to play an important role in acting as metabolic pumps that keep the macula dehydrated[9] and their dysfunction leads to accumulation of fluid in the outer plexiform and inner nuclear layers of the retina.[7,10]

CME occurs in a wide variety of ocular diseases, such as intraocular inflammation, central or branch retinal vein occlusion, diabetic retinopathy, and retinitis pigmentosa, and can occur secondary to the use of several drugs such as epinephrine, latanoprost, and nicotinic acid, and following intraocular surgery.[3-7,9,10] CME is most commonly observed 1 to 3 months postoperatively.[7] Risk factors of postoperative CME include intraocular inflammation with release of prostaglandins, vitreomacular traction, excessive ultraviolet (UV) light exposure, posterior capsule rupture, vitreous loss or incarceration, iris prolapse, transient or prolonged hypotony, use of an intracapsular cataract extraction technique, and use of iris-fixed intraocular lenses.[7,8,11] Fluorescein angiography has been the gold standard test for the evaluation of CME[7] and OCT is a noninvasive imaging technique that can be used to identify and quantify macular edema.[12] OCT findings of CME include diffuse retinal thickening with cystic areas of low reflectivity more prominently in the inner nuclear and outer plexiform layers.[13] Because CME is frequently observed after elective intraocular surgery, especially cataract surgery, the question arises whether OGI and its repair can lead to CME. This study was designed to detect CME and changes in macular thickness in patients with OGI. Most patients with OGI have additional damage and complications such as corneal opacity, traumatic cataract, and vitreous hemorrhage that precludes performing OCT; thus, this study was limited to a small subgroup of patients with OGI who had clear media and no retinal damage.

METHODS

This prospective longitudinal observational pilot study enrolled OGI patients who required surgical repair. This study was approved by the Ahvaz Jundishapur University of Medical Sciences Ethics Committee and informed consent was obtained from all participants.

Patients with a history of previous ocular surgery, significant previous eye trauma, or coexisting ocular pathology that could affect macular thickness, e.g., age-related macular degeneration, glaucoma, diabetic retinopathy and history of uveitis were excluded from the study. Media opacity obscuring the fundus details precluding OCT, presence of retinal damage, and the need for additional surgery were additional exclusion criteria. Between May 2011 and May 2012, among patients with OGI referred to Imam Khomeini Hospital in Ahvaz, Iran, 17 patients who met the inclusion criteria were enrolled.

All patients underwent repair surgery under general anesthesia without intraocular injection at the end of surgery. Vitreous prolapse was managed with cleaning of the wound by superficial vitrectomy using the Alcon Accurus system (Alcon Laboratories, Inc, Fort Worth, TX). In case of iris prolapse, as much of the iris as possible was preserved. For cases with choroidal prolapse, repositioning was performed.

Postoperatively, all patients received standard follow-up and medication, including chloramphenicol and betamethasone drops (Sina Darou, Tehran, Iran) four times per day for 3 weeks. Oral or systemic non-steroidal anti-inflammatory drugs (NSAIDs) and steroids were not used. All patients were followed routinely such that they were admitted for 3-5 days postoperatively, visited 2 days and 1 week after discharge from the hospital, and then 1, 3, 6, and 12 months postoperatively. Additional visits were performed as needed. Follow-up appointments at the first and third postoperative months were considered for this study. At each visit, best-corrected visual acuity (BCVA) and intraocular pressure were measured, and a vitreoretinal specialist performed slit-lamp biomicroscopy and dilated fundus examination, then macular OCT scans were obtained. BCVA was measured at each visit with a Snellen chart and converted to the logarithm of the minimum angle of resolution (Log MAR) equivalent for statistical analysis.

OCT (3D OCT-1000, Mark II; Topcon Corporation, Tokyo, Japan) was performed 1 and 3 months after the operation. Patient follow-up continued from that point, and any observed changes in vision or other examinations were included in the study. In the Early Treatment Diabetic Retinopathy Study (ETDRS) map, macula is divided into 9 regions with 3 concentric rings measuring 1 mm (innermost ring), 3 mm (inner ring), and 6 mm (outer ring) in diameter, centered on the fovea. The innermost 1 mm ring is the fovea while the 3 mm inner ring and 6 mm outer ring are further divided into four equal regions.[14] Software identifies the layers of the retina and determines macular thickness by measuring the distance between the inner limiting membrane (ILM) and the inner boundary of the retinal pigment epithelium (RPE) in each of the 9 regions.[8]

Data are presented as mean ± standard deviation and were analyzed using SPSS version 16.0 (SPSS, Chicago, IL) with independent and paired *t*-tests. *P* values less than 0.05 were considered significant.

RESULTS

Seventeen patients with a mean age of 27.5 ± 9.5 years, ranging 8 to 45 years, were studied. Fifteen patients (88%)

were males and 2 patients (12%) were females. In 7 patients the injured eye was the right eye and in 10 it was the left eye. This difference was not statistically significant (P = 0.12). The laceration was corneal in 10 eyes, scleral in 4 patients, and involved both cornea and sclera in 3 patients. Uveal prolapse was observed in 5 patients, and 3 patients had vitreous prolapse. The laceration average size was 3.47 ± 1.69 mm, ranging from 1 to 7 mm.

Average BCVA in the first and third months was 0.23 ± 0.18 and 0.19 ± 0.18 LogMAR, respectively. This difference was not statistically significant (P = 0.30). There were no cases of intraocular pressure (IOP) rise in any patient; the mean postoperative IOP was 14.4 ± 2.3 and 14.1 ± 2.6 mmHg at 1 and 3 months after operation, respectively, without any statistically significant difference.

None of the studied eyes showed signs of macular edema in the examinations. No cystic changes or other signs of CME were observed in the OCT images.

The average central subfield macular thickness was 235.82 ± 26.35 μm at the first month and 232.76 ± 20.43 μm at the third month. This difference was not statistically significant (P = 0.40). The average central subfield macular thickness at the first and third month were not statistically different from the normal central subfield macular thickness measurements (229 ± 20.46 μm)[15] (P = 0.30 for the first month and P = 0.45 for the third month). The average macular thickness was 273.74 ± 16.30 μm at the first month and 276.16 ± 15.24 μm at the third month. This difference was not statistically significant (P = 0.12).

As determined using the ETDRS map, there was no significant difference in average macular thickness,[14] central thickness, central subfield, total volume, or parafoveal and perifoveal thicknesses between the first and second OCT images of the patients [Table 1]. There was no significant difference in the macular thickness map between eyes with and without uveal prolapse, vitreous prolapse, or scleral lacerations [Table 1]. Macular thickness map was not significantly different between eyes with lacerations less than 4 mm and lacerations equal to or larger than 4 mm [Table 1]. No posterior vitreous detachment, vitromacular traction, or macular holes were observed in any of the eyes.

Table 1. Comparison of variables of patients with open globe injury at the first and third postoperative months

Group	Average macular thickness First month	Average macular thickness Third month
No uveal or vitreous prolapse (9 patients)	272.11±16.15	274.95±15.87
Uveal prolapse (5 patients)	277.64±17.85	279.06±14.91
P value	0.54	0.62
No uveal or vitreous prolapse (9 patients)	272.11±16.15	274.95±15.87
Vitreous prolapse (3 patients)	282.60±21.81	284.84±16.05
P value	0.31	0.30
Laceration size <4 mm (9 patients)	273.14±12.49	277.31±11.53
Laceration size ≥4 mm (8 patients)	274.40±20.69	274.85±19.38
P value	0.84	0.92
Scleral involvement (7 patients)	278.71±17.97	279.65±15.99
No scleral involvement (10 patients)	270.25±14.97	273.72±15.05
P value	0.30	0.44

DISCUSSION

CME occurs 1 to 3 months after intraocular surgery and in a wide variety of ocular diseases, such as intraocular inflammation.[3] Eyes with OGI are subjected to surgical repair that involves intraocular surgery, and frequently experience intraocular inflammation. This study utilized OCT for quantitative evaluation of the macula and detection of possible CME following surgical repair of OGI in patients with clear media and intact retina. OCT was performed 1 and 3 months after the operation because CME is more common 1-3 months after intraocular surgery.[9]

In comparison to normal macular thickness, our patients' average macular thickness in the first and third months did not show any significant differences. The central subfield macular thicknesses at the first and third months were not significantly different from normal central subfield macular thickness. We also found that the presence or absence of vitreous or uveal prolapse, and size and site of laceration did not affect macular thickness. To justify these results, it should be mentioned that patients included in this study were otherwise healthy children, teenagers, and young adults that had no pre-existing vascular abnormalities. The healthy vasculature and BRB may have prevented leakage in the macular region. Also surgeries performed in these eyes were corneal, scleral, and corneoscleral laceration repairs without significant intraocular manipulation. Perhaps, manipulation of the eye is a main trigger for CME, and opening of the globe is not an important factor. On the other hand, traumatized eyes with significant posterior segment trauma, inflammation, corneal opacity, or traumatic cataract that prevents OCT scanning were excluded from the study. These eyes may have had more propensity to develop post-repair macular edema.

Several potentially confounding factors in this study deserve further attention. The results cannot

be generalized to all traumatized eyes because not all patients with OGI were included. This study had a limited follow-up period of 3 months and may not present the late macular thickness changes found in OGI. In addition, small sample size and lack of data pertaining to normal macular thickness in the Iranian population are other limitations of this study.

To the best of our knowledge, there have been no other evaluations of macular thickness after OGI repair. We believe that similar studies with more cases and longer follow-ups would be beneficial.

In conclusion, macular thickness did not change significantly following OGI repair in cases with clear media and without retinal damage; thus, it seems that OGI of this extent and its surgical repair have no effect on macular thickness.

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Conflicts of Interest

There are no conflicts of interest.

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