Müller cell cone-associated foveal detachment as a risk factor for visual acuity loss after glaucoma filtering surgery

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This study was conducted at Sensho-kai Eye Institute.

Abbreviated title: Foveal detachment & VA loss in hypotonia

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Brief summary statement:
Enhanced retinal thickness in eyes with intraocular hypotension may cause traction on the Müller cell cone. Combined intraocular hypotension-associated retinal edema and epiretinal membrane may exacerbate the photoreceptor detachment and the damage to the ellipsoid zone, resulting in long-lasting visual acuity loss after glaucoma filtering surgery.
Abstract

**Purpose:** To examine hypotony-associated foveal lesions (FovLs) utilizing optical coherence tomography and to assess risk factors of visual deterioration after glaucoma filtering surgery.

**Methods:** Parameters that may be associated with post-surgical deterioration of visual acuity were retrospectively studied in 44 eyes of 44 patients who experienced post-surgical intraocular hypotension ≤6 mmHg between 2015 and 2019.

**Results:** Six eyes (14%) had FovLs, such as detachment of photoreceptors (five eyes, 11%) and acquired vitelliform lesions (one eye, 2%) at 3 months after trabeculectomy.

Logistic regression analysis revealed that hypotony maculopathy (P=0.0141 at 3
months) and FovLs (P=0.0486 and 0.0296 at 3 and 12 months, respectively) were significant risk factors for VA loss after trabeculectomy. The FovLs were located just behind the Müller cell cone (MCC). Visual acuity at 3 and 12 months after surgery in patients with FovLs was significantly lower than in those without FovLs (P=0.0013 and P=0.006, respectively). Epiretinal membrane was more common in eyes with FovLs (5 of 6 eyes, 83%) than in eyes without FovLs (7 of 38 eyes, 18%; P=0.0037).

**Conclusion:** MCC-associated FovLs lead to long-lasting visual acuity loss after filtering surgery.

**Key words:** 1) Acquired vitelliform lesion; 2) ellipsoid zone; 3) epiretinal membrane; 4) foveal detachment; 5) glaucoma filtering surgery; 6) hyperreflective foci; 7) hypotony maculopathy; 8) Müller cell cone; 9) optical coherence tomography; 10) visual acuity

**Introduction**

Visual acuity (VA) loss after trabeculectomy is common. VA loss of four or more lines is found in 14.6% of patients, and postsurgical hypotension and hypotony maculopathy (HM) in 42.3% and 8.9% of patients, respectively, after trabeculectomy
with adjunctive mitomycin C (MMC). This type of VA loss has been observed in both trabeculectomy and tube surgery cases.

There is a controversy concerning VA recovery after filtering surgery. Some researchers reported good recovery, while others did not. Kashiwagi et al. reported that VA loss in 28.3% of cases was permanent. Anterior segment complications such as hypotensive corneal striae, astigmatism, edema, deformation of the globe, and cataract may recover spontaneously with restoration of the intraocular pressure (IOP) or may be successfully treated by surgery. In contrast, posterior segment complications, such as HM and microscopic abnormalities of the outer retina, may persist and lead to long-lasting VA loss. Delayed normalization of IOP may result in permanent structural and functional damage. HM occurred in 0–20% of patients and was associated with the duration of hypotension and hypotony score. While maculopathy is a risk factor for postsurgical visual dysfunction after filtering surgery, many patients may experience VA loss without signs of macroscopic HM. In such cases, tiny structural changes in the retina that could be revealed exclusively by optical coherence tomography (OCT) may be responsible for the VA loss. In hypotensive eyes, a 40-μm increase in central retinal thickness (CRT) has been reported. An increase in CRT may lead to traction on the Müller cell cone (MCC) and affect the integrity of the outer retina. Subclinical macular
abnormalities were detected using OCT in over half of the eyes with postoperative hypotony. Another factor that may affect post-surgical VA is the effect of epiretinal membrane (ERM). The power to lift the MCC by the ERM may be transmitted to the outer retina and lead to deterioration of the VA. If the prevalence of ERM increases after glaucoma surgery, it may affect the configuration of the outer retina and VA. Studies on the outer retina may be useful to understand VA loss after filtering surgery; however, this has not been well-studied. In this study, we assessed postsurgical changes in the outer retina utilizing OCT and the effects of ERM and other parameters on postsurgical VA loss after filtering surgery.

**Patients and methods**

This study was a retrospective interventional case series. The inclusion criteria were patients who underwent trabeculectomy with adjunctive MMC at Sensho-kai Eye Institute between 2015 and 2019, experienced post-surgical IOP ≤6 mmHg, and had pre-surgical VA ≥20/40 and available OCT images before and 3- and 12-months after surgery. All surgeries were performed by one surgeon (EC). The exclusion criteria were eyes with vascular diseases, macular degeneration or active iridocyclitis at the time of
surgery, combined cataract and glaucoma surgery, incomplete data, postsurgical IOP exceeding 21 mmHg, next glaucoma surgery performed within 12 months, poor OCT images, and patients who did not agree to participate in this study. The study design was approved by the internal review board of the Sensho-kai Eye Institute. Informed consent was obtained at the time of surgery. The study was conducted in accordance with the tenets of the Declaration of Helsinki.

We recruited 334 eyes that underwent trabeculectomy between 2015 and 2019. Sixty-three eyes with VA<20/40, 160 that underwent combined cataract and trabeculectomy surgery, 18 with postsurgical hypertension >21 mmHg, 21 without an IOP ≤6 mmHg, and 11 with poor or no OCT images were excluded. In 17 patients who underwent surgery in both eyes, the second eye was excluded from the study. Finally, one eye each of 44 patients who underwent trabeculectomy alone were enrolled. Six patients dropped out after 6 months of follow-up, and the data for these eyes at 12 months were treated as missing statistical data.
Baseline data

Age, sex, type of glaucoma, right/left eye treated, best-corrected visual acuity (BCVA), refractive error, mean deviation (MD) using the Humphrey visual field analyzer, IOP at the first visit, presurgical mean IOP at three consecutive visits, number of medications, surgical procedure undergone, history of diabetes mellitus, and central corneal thickness were recorded.

Surgical procedures

After a sub-Tenon injection of 2% lidocaine, subconjunctival 0.02% MMC was applied at the superotemporal meridian. Next, following radial conjunctival incisions at the 10 o’clock position, a 2-clock-hour fornix-based peritomy at the superior meridian was created. A 6-0 nylon traction suture was placed at the tendon of the superior rectus muscle and a 5×4 mm parabolic one-third full-thickness scleral flap was created and extended 1 mm into the clear cornea. Beneath the first scleral flap, a second scleral flap with a size of 4×3 mm and a half the scleral thickness was created, extended to the limbus, and removed by excision at the limbus. The anterior chamber was entered from the surgical limbus, and peripheral iridectomy was performed. A small amount of
viscoelastic material was injected from the side-port into the anterior chamber and the first flap was repositioned and secured by three 10-0 nylon sutures. The conjunctival wound was closed using absorbable 10-0 polyglactin sutures. Thereafter, 0.8 mg of dexamethasone was subconjunctivally injected and the wound was dressed with 0.3% ofloxacin ointment. Topical 0.3% gatifloxacin and 0.1% betamethasone eye drops were applied for 1 month. Postsurgical laser suture lysis was performed following the decision of the attending doctors.

Post-surgical data acquisition

The post-surgical IOP within 1 week was recorded. The IOP, number of medications, and BCVA at 1, 3, 6, and 12 months after surgery, and postsurgical interventions such as laser suture lysis and goniopuncture were recorded. The postsurgical complications studied included HM, cataract, shallow chamber, iridocyclitis, decompression retinopathy, serous choroidal detachment, FovLs such as foveal detachment and acquired vitelliform lesion, hyphema, presence of bleb, bleb leaks, and malignant glaucoma. The progression of lens opacity was assessed using the Emery-Little classification. The definition of HM was a low IOP, ophthalmoscopic radial retinal folds,
choroidal folds, tortuous vessels, optic disc edema, and/or presence of intra-retinal fluid and choroidal folds using OCT.

The macular retina was studied using spectral-domain OCT (AngioVue XR, Optovue, Fremont, CA, USA). Details of the device and our technique of image acquisition have been reported previously. The mean retinal thickness at the central area (within a 1-mm circle of the fovea) and at the superior, temporal, inferior, and nasal meridians of the inner paramacular area (between a 1- and 3-mm circle) and outer paramacular areas (between a 3- and 5-mm circle) were measured mimicking the ETDRS standard.

Subjects with a history of ocular hypotension ≤6 mmHg were subclassified into four groups: 1. eyes without signs of ERM or HM at 3 months (N=24); 2. eyes with ERM but without signs of HM at 3 months (N=7); 3. eyes with signs of HM but without ERM or foveal detachment at 3 months (N=7); and 4. eyes with foveal detachment at 3 months (N=5). One eye had both ERM and a history of HM but did not have foveal detachment at 3 months and was excluded from group 2–4. Thereafter, retinal thickness at the center and paramacular areas was studied.

The presence of ERM, choroidal folds, and foveal detachment was assessed by two independent researchers (EC, TC) using cross-line and retinal map images from the device. Lesions were judged as positive when both researchers agreed on the findings.
**Statistical analysis**

A multiple regression analysis, logistic regression analysis, Fisher’s exact test, Wilcoxon signed rank test, Spearman rank correlation analysis, and Mann–Whitney U-test were performed using the Bell Curve for Excel (Social Survey Research Information Co., Ltd, Tokyo).

**Results**

One eye each of 44 patients who underwent trabeculectomy alone were reviewed. The types of glaucoma included 18 primary open angle glaucoma, eight exfoliation glaucoma, three normal tension glaucoma, four primary angle closure glaucoma, two developmental glaucoma, eight secondary glaucoma, and one ocular hypertension. The demographic data and follow-up changes in IOP and VA are shown in Table 1.

There was a significant reduction in VA at 3 months (P<0.001) and 12 months (P=0.0046, Wilcoxon signed rank test). After a large drop in the VA at 3 months, there was a mild recovery of the ETDRS letter score from 77.1±14.7 letter to 80.7±11.4 letters at 12 months; however, this difference was not statistically significant (P=0.155, Wilcoxon signed rank test).
In this study, 26 of the 44 eyes were pseudophakic, and cataract progression by Emery-Little classification was noted in 4 of 18 phakic eyes. The ETDRS letter score in the 18 phakic and 26 pseudophakic eyes deteriorated to 76.3±15.6 and 77.6±13.0 letters (P=0.723) at 3 months and 80.5±9.0 and 80.8±7.4 letters (P=0.853, Mann–Whitney U test) at 12 months, respectively. The difference in deterioration of the VA between the phakic and pseudophakic eyes was not statistically significant.

Spearman rank correlation analysis revealed a significant association between best corrected VA loss at 3 months and IOP at 1 week (r=−0.349, P=0.020) and pre-surgical IOP (r=−0.328, P=0.030). However, the association between the deterioration of VA at 3 months or 12 months and numerical variables such as age, presurgical MD, pre-surgical BCVA, refractory error, pre-surgical IOP, number of presurgical medications, lowest post-surgical IOP, and postsurgical IOP at 1 week were not statistically significant in a forward-backward stepwise multiple regression analysis (data not shown).

Five of 44 patients had foveal detachment (Figure 1, 2) and 1 of 44 patients had an acquired vitelliform lesion at 3 months after surgery (Figure 3). HM was noted in 13 of 44 eyes. ERM was found in 5 of 6 eyes (83%) with FovLs and in 7 of 38 eyes (18%) without FovLs (P=0.0037, Fisher’s exact test). Five of 12 eyes (42%) with ERM
developed FovLs; however, only 1 of 32 eyes (3%) without ERM developed FovLs (P=0.013). Hard exudation or bleeding were not found in hypotony-associated FovLs.  

An example of foveal detachment and consequent damage to the ellipsoid zone (EZ) in a 71-year-old woman is shown in Figure 1. Her right pre-surgical IOP and BCVA were 29 mmHg and 20/20 and decreased to 3 mmHg and 20/100 at 3 months. At that time, she had choroidal folds and foveal detachment (Figure 1a).  

Six months after surgery, her IOP and BCVA were 7 mmHg and 20/50, respectively. The choroidal folds and retinal edema were diminishing and there was no detachment of the photoreceptors; however, disruption of the EZ was apparent (Figure 1b).  

The detachment of the photoreceptors occurred exclusively at the fovea in all the 6 eyes with hypotony-associated FovLs. FovLs were common in eyes with ERM combined with ocular hypotonia. Foveal detachment in eyes without ERM was a relatively rare event, but occurred when the paramacular retina was thick (Figure 2).  

The retinal thickness at 3 months post-surgery was significantly greater in group 2 than in group 1 by 41.4 µm at the central retina (P=0.010) and by 36.0–43.6 µm at the inner paramacular areas (P=0.017–0.024) except for inferior paramacular area. The outer paramacular retinal thickness at the superior and temporal meridian was significantly

The central and inner paramacular retinal thickness in group 3 was greater than that in group 1 by 72.4 µm (P=0.003) and by 36.8–70.9 µm (P≤0.001–0.004). The outer paramacular retinal thickness also was significantly greater in group 3 than in group 1.

As the Humphrey visual field mean deviation did not differ between group 1 and 3 (P = 0.860), this difference was not attributed to glaucomatous retinal atrophy.

This increase was further exacerbated in group 4.

The central and both the inner and outer paramacular retinal thickness in group 4 was significantly greater than those in group 1 by 211.9 µm (P=0.001), 97.0–121.5 µm (P=0.001) and 43.6–84.4 µm (P=0.004–0.013), respectively (supplemental Table 1, 2).

The central, inferior inner paramacular, and nasal inner paramacular retinal thickness in group 4 was greater than that in group 2 by 170.5 µm (P=0.003), 74.9 µm (P=0.005), and 69.3 µm (P=0.008), respectively (supplemental Table 1, 2). Even if one eye without ERM was excluded from group 4, the inferior and nasal inner paramacular retinal thickness of the remaining four eyes was greater than that in group 2 eyes (P=0.004 and 0.006, respectively).
Similarly, the retinal thickness in group 4 was greater than that in group 3 (supplemental Table 1 and 2).

Occasionally, hypotension triggered development of hyperreflective foci (Figure 3a), which suggests the development of acquired vitelliform lesion. After resolution of the hyperreflective foci, reduction in the intensity of the EZ and structural changes in the inner retina persisted; however, no neovascularization was found (Figure 3b). Thus, VA loss after a hypotonic event cannot be attributed to other diseases such as age-related maculopathy or myopic maculopathy.

The Mann–Whitney U-test showed a significant difference in deterioration of VA at 3 and 12 months in eyes with and without HM and FovLs (supplemental Table 3, http://links.lww.com/IAE/B471). In contrast, the presence of ERM and choroidal effusion caused marginal but non-significant differences in deterioration of VA at 3 and 12 months (supplemental Table 3).

In the binominal logistic regression analysis, the dependent variable was set as yes or no in 5 ETDRS letter score loss (0.1 logMAR visual acuity loss) at 3 months, and nine explanatory parameters such as HM, FovLs, IOP at 1 week, presurgical BCVA, presurgical MD, age, diabetes mellitus, number of medications, and myopia exceeding -6D were set as independent parameters. When cut-off P values of <0.2 were used for
inclusion in the regression equation, presence of HM (P=0.0141) and FovLs (P=0.0486) were significant risk factors for 5 ETDRS letter score loss at 3 months, and post-surgical low pressure at 1 week (P=0.103) was a marginal risk factor. Presence of FovLs was the only significant risk factor for the 5 ETDRS letter score loss at 12 months (P=0.0292; supplemental Table 4, http://links.lww.com/IAE/B472). Thus, HM was a risk factor for mid-term VA loss but not for VA loss at 12 months.

**Discussion**

There may be some reasons for VA loss after trabeculectomy. Besides anterior segment complications, choroidal folds may lead to changes in the orientation of photoreceptors (Stiles-Crawford effect) and affect VA. Concerning the effects of retinal edema, some people reported VA loss in cases of long-lasting structural changes in the internal retina. However, effects of these factors may be minor, because hypotony itself was not a risk factor for VA loss in others. In contrast, presence of HM and FovLs were significant risk factors for VA loss after filtering surgery. Thus, a key point may be the development of the HM and/or
FovLs. In other diseases such as diabetic maculopathy, the extent of EZ disruption was a predictor of visual outcome in diabetic macular edema.\textsuperscript{15}

Paramacular retina was thicker in group 4 than in groups 1, 2, and 3. These findings suggest that the increase in paramacular retinal thickness served as a prop and lifted the internal limiting membrane and MCC, causing impairment of the EZ and detachment of photoreceptors.\textsuperscript{16} The central location of the MCC may be the reason why the detachment was confined at the fovea. After resolution of foveal detachment, damage to the EZ may persist for months (Figure 1b) and may explain the long-lasting VA loss after resolution of foveal detachment.

Foveal detachment may occur in other diseases such as diabetic maculopathy, but the pathogenesis of macular lesions differs among diseases. In cases of diabetic maculopathy, disruption of the blood-retinal barrier, activated cytokine and vascular endothelial growth factor, accumulation of exudate proteins, increased interstitial pressure, and reduced function of the retinal pigment epithelium co-work with retinal Müller glial cells.\textsuperscript{17} The disruption of the blood-retinal barrier and accumulation of interstitial lipoprotein seen in diabetic eyes do not occur in hypotony eyes. This difference may lead to differences in clinical features. In diabetic maculopathy, the first sign of macular lesions is fluid accumulation in the inner retina and detachment of the
photoreceptor occurs in an advanced stage of the disease. In contrast, detachment of the photoreceptor occurs in the early stage of hypotony eyes and tends to diminish over time. This suggests that the main cause of FovLs in hypotonia is not the accumulation of exudates, but choroidal effusion and mechanical traction on the photoreceptor.

Another factor that may affect foveal detachment is ERM. ERM in the normal population is approximately 6%, but may reach 18% after trabeculectomy. When ERM combines with post-surgical hypotension, traction on the MCC by the ERM may combine with traction by retinal edema and lead to long-lasting foveal detachment. The thicker paramacular retina observed in eyes with foveal detachment and ERM than in eyes with only ERM or HM (supplemental Table 1, 2) suggests that ERM exacerbates the tractional detachment of the fovea.

**Limitations**

This is a single site and retrospective study, which may include a bias by the surgeon and during data acquisition. Risk factors for VA loss such as corneal astigmatism and opacity and cataract were not evaluated. The subretinal fluid is drained by active transport by the retinal pigment epithelium, oncotic pressure of the uvea, and the
IOP. However, the power imbalance between absorption, effusion, and traction to the retina in hypotensive eyes has not been proven yet and is a subject for future studies.

**Conclusion**

VA loss was significant at 3 and 12 months after trabeculectomy. Post-surgical FovLs, such as foveal detachment and appearance of hyperreflective foci, were significant risk factors for post-surgical VA loss at 3 and 12 months. HM was a significant risk factor for the post-surgical VA loss at 3 months. The prevalence of ERM in eyes with post-surgical FovLs was higher than that in eyes without FovLs. The combination of post-surgical hypotension and ERM exacerbates VA loss at 12 months after trabeculectomy.

**References**


Figure legends

Figure 1: Hypotony-associated foveal detachment and choroidal folds 3 months after trabeculectomy in a 71-year-old woman with pseudophakic open angle glaucoma and ERM.

Figure 1a: Her pre-surgical IOP under four topical medications, pre-surgical BCVA, axial length, refractive error, central corneal thickness, mean deviation (MD), and MD slope by the Humphrey visual field analyzer in her right eye were 29 mmHg, 20/20, 26.14 mm, −4.25 diopters, 589 µm, −13.7 dB, and −0.56 dB/year, respectively.

One-week post-surgical IOP was 2 mmHg and hypotony maculopathy developed 2 weeks after trabeculectomy. At three months, intra-retinal fluid accumulated at the paramacular inner nuclear layer, and her IOP, central retinal thickness, and BCVA were 3 mmHg, 519 µm, and 20/100, respectively. The foveal detachment developed only at the central retina, where traction on the Müller cell cone is loaded.

Figure 1b: Hypotony associated disruption of the ellipsoid zone (6 months after surgery).

Six months after trabeculectomy, her IOP and central retinal thickness were 7 mmHg and 341 µm, respectively and her BCVA improved to 20/50 but had not recovered to the presurgical level of 20/15. Choroidal folds and the accumulation of
intraretinal fluid at inner nuclear layer were diminishing but persisted. Disruption and low intensity of the ellipsoid zone is apparent at 6 months (arrow). BCVA, best corrected visual acuity; ERM, epiretinal membrane; IOP, intraocular pressure; OCT, optical coherence tomography.

Fig 2: A case of foveal detachment without a sign of foveal traction by the ERM on the Müller cell cone

This is a 3-month post-surgical OCT image of a 77-year-old woman with primary open-angle glaucoma who developed foveal detachment without a sign of traction by the ERM. Her pre-surgical BCVA, refractive error, CCT, IOP, and MD by HFA were 20/25, −1.675 diopters, 566 µm, 22 mmHg, and −14.42 dB, respectively. Her lowest IOP recorded was 3 mmHg and her IOP, BCVA, and CRT at 3 months were 12 mmHg, 20/40, and 329 µm, respectively. No choroidal fold was found at 3 months. Swelling of the paramacular retina (nasal and temporal inner paramacular retinal thickness were 387 and 416 µm, respectively) might have caused elevation of the internal limiting membrane and Müller cell cone and foveal detachment (arrow). CCT, central corneal thickness; MD, mean deviation; HFA, Humphrey visual field analyzer; CRT, central retinal thickness.
Figure 3: Acquired vitelliform lesion in a case of severe traction by the ERM combined with ocular hypotony (Figure 3a) and partial regeneration of EZ 4 years after surgery (Figure 3b)

Figure 3a: Development of acquired vitelliform lesions (hyperreflective foci) is a rare event in cases of ocular hypotension; however, it may occur when the traction by ERM is strong. This is a 3-month post-surgical OCT image of a 62-year-old man with primary open-angle glaucoma. His pre-surgical BCVA, refractive error, CCT, IOP, and MD were 20/15, −12 diopters, 461 µm, 21 mmHg, and −9.15 dB, respectively. His lowest post-surgical IOP was 3 mmHg and accompanied choroidal folds. At 3 months his IOP was 11 mmHg, tight ERM stretched the neural retina, and the external limiting membrane and ellipsoid zone (EZ) were largely disrupted. Hyperreflective foci (arrow) are visible in front of the EZ. His BCVA and CRT at 3 months after surgery were 20/100 and 281 µm, respectively.

Figure 3b: The hyperreflective foci persisted for more than 2 years and then disappeared. At four years after the surgery, his BCVA, CRT, and IOP were 20/30, 284 µm, and 17 mmHg, respectively. He did not recover his pre-surgical acuity of 20/15. Wrinkling of the inner nuclear and Henle fiber layers persisted. The EZ largely regenerated except for partial faintness at the inferior parafovea. Because of high
myopia, his choroid was thin; however, no myopia-associated macular neovascularization was noted.
Table 1: Demographic data and time course of the IOP of the study group.

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<th>Age</th>
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<th>Follow up period (M)</th>
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<th>Myopia &lt;-6 D yes/no</th>
<th>Hypotony maculopathy yes/no</th>
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ETDRS, early treatment diabetic retinopathy study; CCT, central corneal thickness; IOP, intraocular pressure; W, week; M, month;
BCVA, best corrected visual acuity; ERM, epiretinal membrane.