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Visually Significant Cystoid Macula Edema following Glaucoma Drainage Implant Surgery

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Abstract

Purpose—This study examines the incidence of visually significant cystoid macular edema (CME) following glaucoma drainage implant (GDI) surgery and analyses risk factors associated with developing CME and prognosis with treatment.

Materials and Methods—185 eyes from 185 glaucoma patients (mean age 72.46 ± 13.94 years) who underwent GDI surgery at a tertiary eye institute were recruited. Patients were classified based on the presence (CME) or absence (No-CME) of CME. Pre and post GDI best corrected visual acuity (BCVA), number of IOP lowering medications, IOP, standard automated perimetry (SAP) and post GDI complications, were recorded and compared between the two groups. Optical Coherence Tomography (OCT) was used to quantify retinal thickness and monitor CME.

Results—41 (22.2%) eyes developed visually significant CME following GDI surgery. Patients with CME had a higher incidence of pre-GDI NSAID ($p < 0.01$) use and higher number of prior glaucoma surgeries ($p < 0.01$). CME patients had a higher ($p < 0.01$) incidence of iritis, epi-retinal membrane and hypotony. CME eyes responded well to steroids, with resolving macular edema (458.4 ± 151.9 vs 322.0 ± 92.0 μm ; $p < 0.01$) and improving visual acuity (0.73 ± 0.48 vs 0.56 ± 0.56 Log MAR; $p < 0.01$). Both CME and non-CME groups had equivalent lowering of IOP and post-GDI glaucoma medications; with no significant elevation in IOP in the steroid treated CME group.

Conclusions—Post-GDI surgery visually significant CME rates are potentially higher in a real hospital scenario compared to controlled clinical trials. With diligent treatment, CME resolves effectively restoring visual acuity and central macular thickness.

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Introduction

Glaucoma Drainage Implants (GDI) aim at lowering intraocular pressure (IOP) by surgical placement of an artificial filtering device. Initially, after their first inception in clinical trials almost forty-five years ago¹ these devices were used in complicated and refractory glaucoma, but their use as a primary surgical procedure has increased in popularity. According to Medicare data between 1994 and 2012, the number of GDIs increased 410% from 2,356 in 1994 to 12,021 in 2012.² Currently, GDIs are utilized in eyes with high risk of trabeculectomy failure, increased risk of fibroblast proliferation including neovascular glaucoma, uveitic glaucoma and iridocorneal endothelial syndrome,^{3,4} as well as eyes with previous conjunctival incisional surgeries, conjunctival scarring, and cicatricial diseases.⁵⁻⁸ The prospective randomized clinical trial comparing GDI and trabeculectomy, the Tube Versus Trabeculectomy (TVT) study completed in 2006 compared the safety and efficacy of tube shunt surgery in eyes which had undergone previous surgery. After three years of follow-up, GDI surgery was demonstrated to have higher success rates in lowering IOP and lower complication rates compared to trabeculectomy.⁶ A comparison of primary GDIs versus primary trabeculectomy is currently underway and will provide further elucidation to the safety and success of GDIs as a primary surgical procedure.

While GDIs share common post-operative complications as trabeculectomies^{9,10} there are other unique complications associated with GDIs.^{11,12} The rate of endophthalmitis is higher following GDI (2%) compared to trabeculectomy (0.35-0.7%); additionally choroidal hemorrhage, rhegmatogenous retinal detachments, and tractional retinal detachments post-GDI were reported complications found using Medicare claim data.¹³ However, these findings were reported by a single, large, multi-surgeon institute study.¹⁴

Cystoid Macular Edema (CME) is a common cause of reversible vision loss following ophthalmic surgery.¹⁵ Although the exact pathogenesis of CME remains uncertain, there is an accumulation of fluid in the outer plexiform and inner nuclear layers of the retina which can manifest clinically as a decrease in visual acuity.¹⁶ Risk factors of CME include diabetes, vein-occlusive diseases, inflammatory diseases, age-related macular degeneration, Coats disease, radiation retinopathy, inherited dystrophies, tumors, drug-induced macular edema, epiretinal membrane, vitreomacular traction syndrome, or immunodeficiency, as well as others¹⁷, which can alter the hydrostatic or osmotic balance that maintains fluid within the perifoveolar capillaries.

While the TVT study identified a higher post-operative CME in the GDI group compared to the trabeculectomy group,⁶ to the best of our knowledge there is no systemic study of visually significant CME incidence following GDI surgery. The objectives of this study is to examine the incidence of visually significant CME post-GDI surgery, to analyze risk factors associated with developing CME post-GDI surgery, and to explore the therapeutic regimen and outcome during the follow up period.

Materials and Methods

A retrospective chart review was conducted on all patients who underwent GDI surgery at Bascom Palmer Eye Institute, Palm Beach Gardens (Palm Beach Gardens, FL, USA) between December 2007 and August 2014. Glaucoma surgery was performed by three glaucoma specialists and the GDI was chosen based on clinical judgment. The majority of GDIs were Baerveldt™, while a few Ahmed™ and Molteno™ tube shunts were used. The research was conducted in accordance with the Declaration of Helsinki for research involving humans, and Institutional Review Board approval was obtained from the office of Human Research at the University of Miami. The study complied with the requirements of the United States Health Insurance Portability and Accountability Act. Inclusion criteria consisted of a diagnosis of glaucoma, age >18 years, and uncontrolled IOP on maximal medical therapy necessitating surgical intervention. Glaucoma patients had glaucomatous optic nerve damage and a corresponding repeatable abnormal visual field (VF), defined as an abnormal glaucoma hemifield test or pattern standard deviation (PSD) outside 95 % normal limits. Glaucomatous Optic Neuropathy (GON) was defined as neuroretinal rim narrowing, notching, excavation, or retinal nerve fiber layer (RNFL) defect. All eyes were stable from an inflammatory standpoint and had no or trace cells prior to surgery. If both eyes of a patient were eligible, the left eye was excluded. Exclusion criteria consisted of any eyes with nonglaucomatous optic neuropathy, vitreoretinal disease, retinal or macular pathology and preoperative macular edema.

Baerveldt glaucoma implant (Abbott Medical Optics Inc., Abbott Park, IL, USA) GDI surgery was performed in a standardized fashion using either a 350-mm² or 250-mm² Baerveldt glaucoma implant. The surgical technique has been described previously.¹⁸ Briefly, a fornix-based conjunctival flap was dissected, and the implant was sutured to underlying sclera 10 mm posterior to the limbus. The implant was completely occluded to temporarily restrict flow through the device until encapsulation of the plate occurred. The surgeon was given the option of fenestrating the tube for early IOP reduction. The GDI was inserted into the anterior chamber through a 23-gauge needle track or into the pars plana through a 20-gauge microvitrectomy track. Of the 41 patients, 2 patients had pars plana placement, 1 had a sulcus placement and all others had anterior chamber placement above iris plane. Eyes that had the GDI placed into the pars plana also underwent pars plana vitrectomy by a vitreoretinal surgeon to prevent incarceration of the tube lumen. A donor patch graft (either cadaver sclera or cornea) was used to cover the limbal portion of the tube, and the conjunctiva was reapproximated to the limbus with absorbable sutures. All non-valved tubes were ligated near the plate with a 7-0 vicryl suture.

Patients were required to have a minimum follow up time of 3 months post GDI for inclusion in the study. Patient demographics, medical condition, glaucoma type, method of surgical intervention, pre and post GDI best corrected visual acuity (BCVA), number of IOP lowering medications, IOP (Goldman applanation tonometry), standard automated perimetry (SAP) using Swedish Interactive Threshold Algorithm (SITA) standard 24-2 strategy (Humphrey Field Analyzer 750 II-I; Carl Zeiss Meditec Inc., Dublin, CA) and post GDI complications, were recorded. Patients with decreased BCVA following GDI were scanned by Optical Coherence Tomography (OCT) (Zeiss Cirrus™ HD-OCT Zeiss Meditec Inc.,

Dublin CA) to evaluate CME. CME was defined as the presence of intraretinal fluid on macula OCT. Figure 1, shows intraretinal fluid and thickening of the macula associated with marked vision loss, which resolved 3 months later following topical steroid and NSAID therapy. Patients with CME were further evaluated for CME treatment, OCT derived macula thickness, SAP and BCVA during follow up visits. Hypotony was defined as IOP ≤ 5 on one visit. The primary outcome measures were CME response to GDI treatment and risk factor associated with development of CME.

Statistical analysis was performed using SPSS 24 (IBM® SPSS® Inc, Chicago, IL). Clinical characteristics of the study population and univariate analysis were performed using Chi square or Fishers's exact test for categorical variable and 2-tail independent samples t-test for continuous variables. Snellen visual acuity measurements were converted to logMAR equivalents for the purpose of data analysis. Multivariate linear regression model was used to characterize the predictor factors for CME formation and risk ratio analysis. 2-tailed pair t-test was used to quantify macula thickness and visual outcome during follow up visits. A p value of ≤ 0.05 was considered statistically significant.

Results

Two hundred and fifty one patients that underwent GDI surgery during December 2007 and August 2014 at Bascom Palmer Palm Beach Gardens. Fifty-one patients were excluded due to pre-existing macular pathology. Fifteen patients were excluded due to having less than 3 months of post-surgical follow up time. A total of 185 eyes of 185 patients were included in the study; 41 (22.2%) eyes were documented to have post-GDI surgery CME as determined by OCT macula scans and 144 (77.8%) eyes did not have any clinical evidence of CME. The mean age of all 185 subjects was 72.46 ± 13.94 years, of which 91 (49.2%) were males and 94 (50.8%) were females. Self-identified racial breakdown of individuals (152 patients) included 109 (58.9%) Caucasian, 40 (21.6%) African Americans (AA) and 3 (1.6%) Asians.

There was no statistical difference ($p > 0.05$) across age, race, gender and choice of eye between the CME group and no-CME group (Table 1). Glaucoma subtypes were similar between the CME and non-CME group and included primary open angle (POAG; 25 (61.0%) vs 82 (56.9%)), pseudo-exfoliative (XFG; 5 (12.2%) vs 22 (15.3%)) and uveitic (8 (19.5%) vs 17 (11.8%)). Lens status (phakia, pseudophakia and aphakia) were statistically similar ($p = 0.65$) between the CME and no-CME group. Patients in the CME group had higher number of pre-GDI glaucoma surgeries (1.32 ± 0.47 vs 0.45 ± 0.51 ; $p < 0.01$), a higher incidence of NSAID use (5 (12.2%) vs 3 (2.1%); $p < 0.01$) and a thicker pre-GDI central macula thickness ($288.1 \pm 60.9 \mu\text{m}$ vs $245.2 \pm 49.4 \mu\text{m}$; $p = 0.01$) when compared to the no-CME group. Because eyes with retinal vascular pathology were excluded from this study, the non-CME group had a higher incident of diabetes (without retinopathy) (35 (24.3%)) compared to the CME group (2(4.9%)). The number of glaucoma medications, IOP, BCVA (LogMAR), steroid use, visual field SAP Mean deviation (MD) and visual field SAP pattern standard deviation (PSD) were statistically similar between the CME eye and No-CME eye (Table 1).

Post-GDI surgery patients were followed for an average of 15.83 ± 12.55 months. Overall, patients in the CME group were followed for a longer duration than the no-CME group (21.0 ± 13.6 vs 14.4 ± 11.9), which was significantly longer ($p < 0.01$). At the end of study period 18/41 patients in the CME group had central foveal thickness less than pre-GDI central macular thickness mean of $288.1 \mu\text{m}$. The CME group patients had a higher incidence of iritis (14 (34.1%) vs 14 (9.7%); $p < 0.01$), epi-retinal membrane (24 (58.5%) vs 31 (21.5%); $p < 0.01$) and hypotony (13 (31.7%) vs 11 (7.6%); $p < 0.01$) compared to No-CME group. Consequently the use of anti-inflammatory agents NSAID (12 (29.3%) vs 6 (4.2%); $p < 0.01$) and intraocular steroid (27 (65.9%) vs 58 (40.3%); $p < 0.01$) post-surgery were also higher in CME group compared to no-CME group. OCT derived central macula thickness post-GDI surgery was higher in CME group both immediately after surgery (458.4 ± 151.9 vs $260.2 \pm 57.2 \mu\text{m}$; $p < 0.01$) and at the final visit ($322.0 \pm 92.0 \mu\text{m}$; $p < 0.01$) than the no-CME group. All other measured parameters namely; IOP, number of glaucoma medications, BCVA, visual field SAP MD and visual field SAP PSD were similar post-GDI surgery between CME and No-CME group (Table 2).

A multivariate linear regression analysis with post-GDI CME as an outcome variable with age, the number of prior glaucoma surgeries, pre-GDI ocular steroid use, pre-GDI ocular NSAID, pre-GDI central macula thickness as independent variables revealed that the number of pre-GDI glaucoma surgery ($p < 0.01$) and pre-GDI NSAID use ($p < 0.01$) were significant predictors for post-GDI CME. The risk for developing post-GDI CME was 3.08 times in patients who had an exposure of NSAID pre-GDI surgery. All patients in CME group has at least one prior glaucoma surgery before the present surgery. Also, the odds of having post-GDI CME with post-GDI iritis, epi-retinal membrane and hypotony was 4.81, 5.16 and 5.60 respectively.

Within the CME group, immediate post-GDI surgery BCVA (LogMAR) was lower (0.73 ± 0.48 vs 0.55 ± 0.56 ; $p < 0.01$, $n = 33$) and OCT derived central macula thickness was higher (458.4 ± 151.9 vs $322.0 \pm 92.0 \mu\text{m}$; $p < 0.01$) when compared to their final visit. There were 16 (39.0%) eyes with CME recurrence; of which 10 (62.5%) eyes were treated with topical steroid and 6 (37.5%) eyes were treated with injectable steroid prior to CME recurrence.

An analysis of IOP, number of glaucoma medications before and after GDI surgery within the CME and no-CME group separately, revealed that IOP (25.68 ± 8.59 vs 12.90 ± 5.05 mmHg and 26.46 ± 10.94 vs 12.63 ± 5.10 mmHg) and the number of glaucoma medications (3.41 ± 0.97 vs 1.76 ± 1.34 and 3.33 ± 1.25 vs 2.20 ± 1.38) were reduced significantly ($p < 0.01$) for both groups after GDI surgery. In the no-CME group there was a reduction in visual field SAP MD (-14.23 ± 9.54 vs -15.92 ± 9.66 ; $p < 0.01$) following GDI; but no significant difference in central macula thickness ($p = 0.08$) and visual field SAP PSD ($p = 0.81$). The CME group did not show any difference in BCVA, central macular thickness, visual field MD and PSD pre and post GDI surgery (Table 3).

Given the potential for IOP elevation with intraocular steroid use, an analysis of IOPs of patients who received topical and systemic steroids for treatment of the post-GDI CME was performed. There were a total of 27 patients in this group. The mean follow up time for the

group was 22.9 ± 13.4 months. The mean IOP for one year post-GDI follow up visits among these patients were 15.32 ± 2.15 mm Hg, the mean IOP for post-op month one, three, six and one year were 17.33 ± 9.78 , 16.73 ± 8.02 , 14.56 ± 8.19 and 12.64 ± 5.78 mm Hg respectively. The BCVA (in LogMAR) for the same time points were 0.56 ± 0.39 , 0.50 ± 0.36 , 0.50 ± 0.34 and 0.54 ± 0.43 respectively.

Discussion

With the increase use of GDIs both as a primary treatment modality and in refractory glaucoma, there is necessity to have a closer scrutiny on its side-effects outside the realm of controlled clinical trials. The motivation for this study is to evaluate a common ophthalmic post-surgical complication and its association with GDI surgery. We analyzed the records of all patients who underwent GDI surgery at a single academic center. Forty one (22.2%) out of 185 patients enrolled in the study developed visually significant CME following GDI surgery. Patients who developed CME had a higher incidence of pre-GDI NSAID use and a significantly higher number of prior glaucoma surgeries. CME patients also had a higher incidence of iritis, epi-retinal membrane and hypotony. However, both CME and non-CME groups had equivalent IOP lowering and post-GDI glaucoma medications.

The randomized multicenter controlled trial TVT study reported 5 % patients in the GDI group to have developed CME after 1 month or more following the GDI surgery in 5 years follow up time period¹⁹, which is much lower than findings in our study. In our study we also observed a higher incidence of CME, which had a high association with iritis and hypotony. A potential reason for this difference could be that the percentage of patients with POAG in TVT study is much higher (82% vs 57%) than ours; our study included more patients with pseudo-exfoliative, uveitic and other complicated glaucoma undergoing GDI surgery, this also supports the findings that several patients using topical NSAID prior to surgery and the pre-GDI OCT derived central retinal thickness at baseline was higher (288.1 ± 60.9 vs 244.9 ± 48.7 μ m; $p < 0.01$) in CME group than the no-CME group. The patients who developed CME had a higher mean age (75.7 ± 12.2 years) compared with patients who did not develop CME (71.5 ± 14.3 years) or patients in TVT study (70.9 ± 11.0 years). In the TVT study, CME was not reported based on OCT scans, which could explain the lower percentage of CME compared to our study; fundusoscopic examination is not as sensitive as using OCT. The baseline LogMAR visual acuity of patients in the tube group for the TVT study also had a better visual acuity (0.42 ± 0.54 vs 0.51 ± 0.55) than our study population.²⁰ The three year follow up of the TVT study showed more degradation of LogMAR visual acuity in their GDI group (0.42 ± 0.54 to 0.61 ± 0.73 vs 0.51 ± 0.55 to 0.56 ± 0.54) following GDI surgery compared to our group of patients.⁶ IOP lowering (25.1 ± 5.3 to 13.4 ± 4.8 and 26.4 ± 10.5 to 12.9 ± 5.5) outcome at 2 years' time point for the TVT study patient were very similar to the present study.⁶

Even though nearly 20 % of our GDI surgery developed post-surgical visually significant CME, the overall prognosis of CME treatment with topical and injectable steroids were very effective. CME patients showed significant ($p < 0.01$) recovery of their visual acuity and achieved reduction of OCT derived central retinal thickness. Moreover, patients ($n = 27$) who received steroid treatment for CME following GDI surgery had a mean IOP of 15.54

± 3.09 mm Hg for all post-surgery follow up visits; and their IOPs for the first three months were in mid-teens, suggesting that the presence of a GDI is protective against an steroid related IOP spike.

Potential limitations to this study include the setting of a tertiary eye care facility in which patients present with relatively severe and complicated glaucoma, making these findings less generalizable. In our study, we excluded patients with pre-existing retinal vascular disease; therefore the rate of diabetes in the CME group was actually lower than the group without CME, as patients with diabetic retinopathy tend to have a higher rate of macula edema. The current study is retrospective in nature with the purpose to evaluate the incidences of CME post-GDI surgery in a real world hospital scenario. OCT macula scan before surgery is still not a standard clinical practice; thus we do not have OCT macula scans of each eye prior to the GDI surgery. However, all patients had a careful funduscopy examination prior to surgery and any clinical evidence of CME or other vasculopathic retinopathy was excluded from the study. Also, it is possible that there might be a higher incidence of CME following GDI surgery as OCTs were performed only on patients with deterioration of visual acuity; therefore, CME in patients with stable vision would not be clinically significant. In regards to poor pre-operative vision (-0.50 LogMAR) the average severity of glaucoma was a MD -15 , which would account for limitation of pre-operative vision. A prospective study is warranted, in which a pre-operative OCT would be attained in all patients as well as post-operative OCTs in everyone. It is likely that the rate of total CME would be higher as some subclinical CME would be revealed with routine OCTs.

In conclusion, GDI is a highly effective glaucoma surgery and can be applied to a wide variety of glaucoma types both as a primary treatment modality and under refractory glaucoma conditions. Post-GDI surgery visually significant CME rates are potentially higher in a real hospital scenario compared to controlled clinical trials. However, with diligent treatment, CME resolves effectively restoring visual acuity and central macular thickness. A randomized clinical trial to treat at-risk patients prophylactically, similar to at-risk cataract patients, is warranted.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

OCT scans of the right eye from an 80 year old male following GDI surgery. Pre-op BCVA was 20/50; 5 months after surgery was noted to have BCVA of 20/250 with IOP 1; marked intra retinal fold (IRF) was noted on macula OCT (figure 1A; central macula thickness (CMT) 617 μ m). Patient was treated with topical prednisolone acetate 1% qid, ketorolac qid and atropine 1% bid. The vision gradually improved with resolution of CME noted 3 months later (figure 1B; CMT 339 μ) with BCVA 20/30. Note patient has an epi-retinal membrane (ERM).

Table 1

Baseline parameters of the study population

	Cystoid macula edema (CME) N = 41	No cystoid macula edema (No-CME) N = 144	p value
Age (years)	75.71 ± 12.16	71.53 ± 14.31	0.09
Race	Caucasian AA Asian	84 (70.6%) 33 (27.7%) 2 (1.7%)	0.69
Gender	Male Female	71 (49.3%) 73 (50.7%)	0.95
Eye	OD OS	73 (50.7%) 71 (49.3%)	0.74
Diagnosis	POAG XFG Uveitic Others	82 (56.9%) 22 (15.3%) 17 (11.8%) 23 (16.0%)	0.41
Lens Status	Phakic Pseudophakic Aphakic	24 (16.7%) 117 (81.3%) 3 (2.1%)	0.65
Pre-GDI number of glaucoma surgery	1.32 ± 0.47	0.45 ± 0.51	< 0.01
Pre-GDI number of glaucoma medication	3.41 ± 0.97	3.33 ± 1.25	0.70
Pre-GDI IOP (mmHg)	25.68 ± 8.59	26.46 ± 10.94	0.68
Pre-GDI BCVA (LogMAR)	0.52 ± 0.56	0.50 ± 0.54	0.84
Pre-GDI Steroid use	9 (22.0%)	32 (22.2%)	0.97
Pre-GDI NSAID use	5 (12.2%)	3 (2.1%)	<0.01

	Cystoid macula edema (CME) N = 41	No cystoid macula edema (No-CME) N = 144	<i>p</i> value
Pre- GDI center macula thickness (µm)	288.1 ± 60.9	245.2 ± 49.4	0.01
Pre- GDI Visual Field MD (dB)	-16.53 ± 9.51	-15.80 ± 9.20	0.68
Pre- GDI Visual Field PSD (dB)	7.14 ± 3.07	7.17 ± 3.40	0.97

AA, African American; OD, Right eye; OS, Left eye; POAG, Primary Open Angle Glaucoma; XFG, Pseudo-exfoliative Glaucoma; GDI, Glaucoma Drainage Implants; IOP, Intraocular Pressure; BCVA, Best Corrected Visual Acuity; MAR, Minimum Angle of Resolution; NSAID, Nonsteroidal Anti-inflammatory Drugs; MD, Mean Deviation; PSD, Pattern Standard Deviation.

Statistically significant *p* values are in bold. Chi-Square test for categorical variables and Independent Student's *t*-test for continuous variables.

Table 2

Post-GDI surgery findings

		Cystoid macula edema (CME) N = 41	No cystoid macula edema (No-CME) N = 144	<i>p</i> value
IOP (mmHg)		12.90 ± 5.05	12.63 ± 5.10	0.76
Iritis		14 (34.1%)	14 (9.7%)	< 0.01
Epi-retinal membrane		24 (58.5%)	31 (21.5%)	< 0.01
Hypotony		13 (31.7%)	11 (7.6%)	< 0.01
Number of glaucoma medication		1.76 ± 1.34	2.20 ± 1.38	0.07
Follow-up time (months)		21.0 ± 13.6	14.4 ± 11.9	< 0.01
Steroid use		27 (65.9%)	58 (40.3%)	< 0.01
NSAID use		12 (29.3%)	6 (4.2%)	< 0.01
Central macula thickness (µm)	Post-GDI surgery	458.4 ± 151.9	260.2 ± 57.2	< 0.01
	Last visit	322.0 ± 92.0	260.2 ± 57.2	< 0.01
BCVA (LogMAR)	Post-GDI surgery	0.73 ± 0.48	0.56 ± 0.55	0.10
	Last visit	0.56 ± 0.56	0.56 ± 0.55	0.99
Visual field MD (dB)		-12.48 ± 10.16	-15.68 ± 9.59	0.23
Visual field PSD (dB)		7.61 ± 3.02	7.18 ± 3.38	0.64

IOP, Intraocular Pressure; NSAID, Nonsteroidal Anti-inflammatory Drugs; GDI, Glaucoma Drainage Implants; BCVA, Best Corrected Visual Acuity; MAR, Minimum Angle of Resolution; MD, Mean Deviation; PSD, Pattern Standard Deviation.

Statistically significant *p* values are in bold. Chi-Square test for categorical variables and Independent Student's *t*-test for continuous variables.

Table 3

Pre-GDI and post-GDI final comparison

	Cystoid macula edema (CME)			No cystoid macula edema (No-CME)		
	Pre-GDI	Post-GDI	<i>p</i> value	Pre-GDI	Post-GDI	<i>p</i> value
IOP (mmHg)	25.68 ± 8.59	12.90 ± 5.05	<0.01	26.46 ± 10.94	12.63 ± 5.10	<0.01
Number of glaucoma medications	3.41 ± 0.97	1.76 ± 1.34	<0.01	3.33 ± 1.25	2.20 ± 1.38	<0.01
BCVA (LogMAR)	0.52 ± 0.56	0.56 ± 0.56	0.60	0.50 ± 0.55	0.56 ± 0.55	0.07
Central macula thickness (µm)	288.1 ± 60.9	316.8 ± 94.6	0.08	243.7 ± 45.0	258.6 ± 58.8	0.08
Visual Field MD	-11.40 ± 7.29	-12.17 ± 10.41	0.77	-14.23 ± 9.54	-15.92 ± 9.66	<0.01
Visual Field PSD	7.60 ± 3.28	7.38 ± 2.97	0.81	7.32 ± 3.45	7.25 ± 3.41	0.81

IOP, Intraocular Pressure; BCVA, Best Corrected Visual Acuity; MAR, Minimum Angle of Resolution; MD, Mean Deviation; PSD, Pattern Standard Deviation.

Statistically significant *p* values are in bold. Chi-Square test for categorical variables and Independent Student's *t*-test for continuous variables.