Precis: Steroid response after cataract surgery was more frequent in glaucoma patients than nonglaucoma patients. Longer axial length and more preoperative medications were risk factors for steroid response in glaucoma patients.

Purpose: The aim was to evaluate incidence and risk factors for topical steroid response after uneventful cataract surgery in patients with and without glaucoma.

Setting: Academic glaucoma clinics.

Design: This was a retrospective review.

Participants: Consecutive patients with and without glaucoma and no prior incisional glaucoma surgery undergoing cataract surgery between March 2007 and September 2016. All patients routinely received topical prednisolone acetate 1% postoperatively.

Methods: Pertinent clinical information was recorded. Steroid response was defined as intraocular pressure >50% above the baseline intraocular pressure measurement, occurring at or after the second postoperative week.

Results: We included 472 eyes of 472 nonglaucoma patients and 191 eyes of 191 glaucoma patients. Ten (2.1%) nonglaucoma eyes and 16 (8.4%) glaucoma eyes were diagnosed as steroid responders (relative risk = 3.72; 95% confidence interval: 1.71-8.07; \( P < 0.001 \)). Logistic regression showed that for nonglaucoma, longer axial length (AL) and younger age were associated with a higher incidence of steroid response (\( P \leq 0.003 \)), while for glaucoma patients, longer AL and more preoperative medications were associated with steroid response (\( P \leq 0.030 \)). An AL \( \geq 26 \) mm was associated with steroid response for both groups (\( P \leq 0.024 \)).

Conclusion: Although glaucoma patients were 3.72 times more likely to have steroid response after uneventful cataract surgery, the incidence of steroid response with prednisolone acetate 1% was relatively low after phacoemulsification in both nonglaucoma and glaucoma eyes. Steroid response was associated with longer AL in both groups and with more preoperative medications in glaucoma patients.

Key Words: cataract surgery, steroid response, glaucoma, IOP (J Glaucoma 2021;30:e159–e163)

Age-related cataract is the leading cause of visual impairment,1 and cataract surgery is one of the most frequently performed operations worldwide.2–4 Postoperative inflammation is commonly experienced after cataract surgery and is treated to prevent events related to severe inflammation, including corneal edema, secondary glaucoma, anterior or posterior synechia, and macular edema.5 Several different corticosteroids are available, but prednisolone acetate 1% is the most widely prescribed after cataract surgery.6

The phenomenon of glucocorticosteroid-induced intraocular pressure (IOP) elevation has been recognized for decades, and previous studies have demonstrated a significant increase in IOP in normal eyes and in glaucoma patients after steroid use.7 Armaly8 indicated that approximately one-third of normal eyes and >90% of patients with primary open-angle glaucoma (POAG) respond with >6 mm Hg of IOP elevation after receiving a 4-week course of topical dexamethasone 0.1%. Becker and Mills9 reported that 100% of POAG patients showed IOP rise (from a mean value of 16.9 to 32.1 mm Hg) after 2 to 4 weeks use of topical betamethasone 0.1%. A more recent publication noted a steroid response incidence of 2.4% in patients receiving topical prednisolone acetate 1% after routine cataract surgery, but this report included an unknown number of eyes with glaucoma.10 We studied a series of patients with and without glaucoma who had phacoemulsification and evaluated the characteristics of patients who had a postoperative steroid response to prednisolone acetate 1%.

METHODS

The Human Subjects Division of the University of Washington approved this study. This study followed the tenets of the Declaration of Helsinki and was conducted in compliance with the Health Insurance Portability and Accountability Act. We conducted a retrospective review of consecutive patients with and without glaucoma with no prior incisional surgery undergoing phacoemulsification as a sole procedure between March 2007 and September 2016 at the University of Washington. The diagnosis of glaucoma was based on characteristic optic nerve findings and/or visual field loss. Patients were excluded if they had a prior trabeculectomy or other incisional glaucoma surgery, did not have at least 3 prior IOP measurements within 1 year before surgery, had IOP measured by any other method other than Goldmann application, or had a complication during phacoemulsification. When both eyes of one patient were eligible, the eye undergoing surgery first was chosen for the study.

For the entire cohort, we recorded pertinent clinical information before phacoemulsification, including baseline demographics, diagnosis subtype, disease severity indices, ophthalmic biometry obtained by IOL Master (Carl Zeiss Meditec Inc., Dublin, CA) or ultrasound A-scan (Innovative Imaging Inc., Ellex, Minneapolis, MN), preoperative IOP, postoperative IOP at day 1, week 1, and at least 1 additional time between week 3 and week 5, and treatment measures.
All Snellen VA measurements were converted to logarithm of the minimum angle of resolution (logMAR) for statistical analysis. Cataract density was graded 0–4 with the primary grade based on the nuclear cataract, and 0.5 added for any element of cortical or subcapsular cataract.

Cataract surgery was performed using topical plus intracameral anesthesia and standard phacoemulsification technique; pupil expansion techniques and devices were used to enlarge the pupil as necessary, and incisions that were not watertight were sutured. Oral acetazolamide was routinely prescribed postoperatively (250 to 500 mg to be taken 3 to 6 hours postsurgery and the following morning) for patients with a history of glaucoma. After surgery, all patients were started on topical prednisolone acetate 1% at a frequency of 4 times daily. The cumulative prednisolone dose was defined as the sum of all doses received starting immediately after surgery until the visit that a steroid response was diagnosed during the postoperative period, or until the final postoperative visit if no steroid response was noted. Postoperative complications were recorded as noted in the chart. Persistent anterior chamber inflammation was defined as any anterior chamber cells at the 1-month postoperative visit.

All IOP measurements were taken using Goldmann applanation by the ophthalmic technician or surgeon. For glaucoma patients, the baseline IOP was the average of the 3 IOP readings within 1 year before phacoemulsification. For nonglaucoma patients, the baseline IOP was the average of up to 3 prior IOP measurements within 1 year before surgery. A steroid response was defined as an IOP > 50% greater than baseline while using topical prednisolone acetate 1% drops and occurring at or after the second postoperative week, with the first postoperative week IOP not meeting the criteria for steroid response. Patients with eyes that did not meet the steroid response criteria were considered nonresponders. For steroid responders, we recorded the postoperative IOP at 6 months, which was the single visit reading obtained at the first routine visit at least 6 months postoperatively.

Univariate analysis was performed using a 2-tailed t test for continuous variables and the Pearson $r^2$ and the Fisher exact test for categorical variables. Logistic regression was used to evaluate the relationship between variables considered potential risk factors ($P < 0.450$) and steroid response risk. No IOP variables were used in the multivariate regression. A P-value < 0.05 was considered significant.

**RESULTS**

All results are in mean ± SD where applicable. The study population included 472 eyes of 472 nonglaucoma patients [age: 68.4 ± 11.3 yrs; axial length (AL) 24.2 ± 1.6 mm; baseline IOP: 15.3 ± 2.6 mm Hg] and 191 eyes of 191 glaucoma patients [age: 71.8 ± 9.9 yrs; AL 24.1 ± 1.6 mm; baseline IOP 16.3 ± 4.4 mm Hg on 2.1 ± 1.2 medications; visual field mean deviation $–7.0 ± 7.3$ (median $–4.7$)] dB. Ten nonglaucomatous eyes (2.1%) and 16 glaucomatous eyes (8.4%) were identified as having a steroid response during follow-up (relative risk $= 3.72$; 95% confidence interval: 1.71–8.07; $P < 0.001$). Figure 1 shows the distribution of surgical cases and steroid response over time. Nonglaucoma patients who had a steroid response were more likely to be younger, high myopes, and to have a longer AL than those who did not have a steroid response (Table 1). An AL ≥ 26 mm was associated with steroid response ($P < 0.001$). No differences in other demographic or preoperative characteristics, including cataract density, or cumulative prednisolone dose were found to reach statistical significance (Table 1). Axial length was highly correlated with refractive error ($r = –0.744$, $P < 0.001$). Male patients had a longer mean AL than female patients ($24.54 ± 1.41$ vs. $23.83 ± 1.63$ mm; $P < 0.001$). The mean IOP at month 1 was $23.6 ± 5.7$ for the steroid response group and $14.2 ± 3.1$ for the no steroid response group ($P < 0.001$). Postoperative visual acuity was not different between the 2 groups (logMAR $0.074 ± 0.115$ vs. $0.079 ± 0.224$; $P = 0.943$).

Glaucoma patients who had a steroid response had a longer AL than those who did not have a steroid response and were using more preoperative glaucoma medications (Table 2). An AL ≥ 26 mm was associated with steroid response ($P = 0.024$). No significant differences were found in other demographics or preoperative characteristics, indices of glaucoma severity, laser treatment, or cumulative prednisolone dose ($P ≥ 0.099$, Table 2). Male patients had a longer mean AL than female patients ($24.51 ± 1.50$ vs. $23.79 ± 1.60$ mm) ($P = 0.001$).

Logistic regression analysis included risk factors with $P < 0.450$ and did not include IOP variables. Some variables were highly correlated (eg, male sex, refraction, and AC depth, with AL), and only the variable with the lowest P-value was used in the regression. Among nonglaucoma patients, steroid response was associated with each mm increase in AL [odds ratio (OR): 1.826; 95% confidence interval (CI): 1.374–2.427; $P < 0.001$], and younger age at time of surgery (per year) (OR: 0.914; 95% CI: 0.862–0.970; $P = 0.003$) (Table 3). Among glaucoma patients, steroid response was associated with each mm increase in AL (OR: 1.813; 95% CI: 1.313–2.505; $P < 0.001$) and each additional preoperative medication (OR: 1.774; 95% CI: 1.058–2.973; $P = 0.030$) (Table 3).

For glaucoma patients with steroid response, the mean IOP at the time of diagnosis of steroid response was $26.5 ± 9.3$ (range: 14 to 45 mm Hg), compared with $15.3 ± 3.9$ in nonsteroid responders at postoperative month 1 ($P < 0.001$). Eight glaucoma patients (50%) with steroid response required additional medications to control IOP, including 5 who received oral acetazolamide, and 1 had selective laser trabeculoplasty within 90 days of the cataract surgery. All patients that had steroid response had resolution of their IOP increase (mean weeks to resolution $6.0 ± 4.7$). Among steroid responder glaucoma eyes with at least 6 months of follow-up ($N = 15$), the mean preoperative IOP was $13.7 ± 3.0$ on $2.6 ± 0.9$ medications, and the IOP at

**FIGURE 1.** Distribution of surgical cases and 122 steroid response over time.
TABLE 1. Clinical Characteristics of Nonglaucoma Patients Studied, Including Risk Factors for Postoperative Steroid Response After Phacoemulsification (N=472 Eyes)

| Risk Factor | Steroid Response (N = 10, 2.1%) | No Steroid Response (N = 462, 97.9%) | P |
|-------------|-------------------------------|----------------------------------|---|
| Age (y)     | 57.0 ± 8.5                    | 68.7 ± 11.3                     | 0.001* |
| Sex (male), n (%) | 8 (80.0)                 | 201 (43.5)                      | 0.026‡ |
| Race, n (%) |                               |                                 |     |
| Caucasian   | 9 (83.3)                      | 353 (76.4)                      | 0.626† |
| Asian       | 0                             | 53 (11.5)                       |     |
| Black       | 0                             | 16 (3.5)                        |     |
| Other       | 1 (10.0)                      | 40 (8.6)                        |     |
| Preoperative VA (LogMAR) | 0.264 ± 0.201               | 0.356 ± 0.400                   | 0.471* |
| Preoperative spherical equivalent (D) | −8.2 ± 5.3               | −1.5 ± 3.9                      | <0.001* |
| Cataract density (0-4) | 2.15 ± 1.05               | 2.51 ± 0.74                     | 0.308* |
| Average preoperative IOP (mm Hg) | 13.6 ± 2.8                | 15.4 ± 2.6                      | 0.051§ |
| AL (mm)     | 27.0 ± 2.2                    | 24.1 ± 1.5                      | <0.001* |
| AL ≥ 26.0 mm, n (%) | 7 (70.0)                | 52 (11.2)                       | <0.001† |
| ACD (mm)    | 3.21 ± 0.52                   | 3.12 ± 0.46                     | 0.553* |
| Pupil enlargement (yes), n (%) | 0                        | 21 (4.5)                        | 1.000‡ |
| Postop CME (yes), n (%) | 1 (10.0)                 | 26 (5.6)                        | 0.448‡ |
| Persistent AC reaction | 12 (2.6)                | 1.000‡                          |     |
| Cumulative prednisolone dose (drops) | 100.4 ± 15.9             | 101.7 ± 15.7                    | 0.972‡ |
| Postop VA (LogMAR) | 0.074 ± 0.115               | 0.079 ± 0.224                   | 0.943* |

Bold values indicate statistically significant (P < 0.05).

*Two-tailed independent sample t test.
†The Pearson χ² test.
‡The Fisher exact test.
§Mann-Whitney U test.

ACD indicates anterior chamber depth; AL, axial length; CME, cystoid macular edema; IOP, intraocular pressure; steroid response, > 50% increase in IOP from baseline at postoperative month 1; VA, visual acuity.

the 6-month visit was 14.4 ± 4.1 (P = 0.450, paired t test) on 2.7 ± 1.1 medications (P = 0.433, paired t test).

**DISCUSSION**

Topical ophthalmic corticosteroids carry an inherent risk of side effects, including IOP elevation, a risk factor for the development or progression of glaucoma. The criteria for steroid responsiveness vary widely between studies in the literature. In 1984, Stewart et al13 conducted a study comparing the ocular pressure effects of fluorometholone acetate and dexamethasone sodium phosphate in steroid responders and proposed that an increase in IOP of ≥ 10 mm Hg over baseline should be considered clinically significant. Other studies in the literature have also used the same definition.12,13 More recently, Chang et al10 arbitrarily defined a steroid response as being an elevation to at least 28 mm Hg as they believed that at this IOP level, many clinicians would be concerned enough to stop the topical corticosteroid, prescribe a topical IOP-lowering medication, or increase the frequency of follow-up visits. They reported among 1613 patients, including an unknown number with glaucoma, 39 (2.4%) had a steroid response.10

In our study, we found the incidence of a steroid response after uneventful phacoemulsification with topical prednisolone acetate 1% use postoperatively to be low among nonglaucoma eyes, at 2.1%. The incidence was higher at 8.4% in glaucoma eyes, though much lower than historical values.8,9 We elected to use a percentage change based on our prior work on IOP spike after cataract surgery14,15 and because we believe this is more consistent with the glaucoma literature in which treatment goals are frequently defined as percentage changes from baseline. It also accounts for the severity of the patient’s glaucoma. Previously we have shown that among 4 IOP spike definitions (IOP > 50% greater than baseline IOP, IOP ≥ 30 mm Hg, IOP elevation ≥ 10 mm Hg above baseline, IOP elevation ≥ 5 mm Hg above baseline) the percentage change criteria included > 70% of patients that had fulfilled one of the IOP spike definitions, primarily omitting some eyes with IOP elevation ≥ 5 mm Hg, which is the most conservative definition.15

Glaucoma is a known risk factor for a steroid response,7,9,16 and in our study, glaucoma patients were 3.721 times more likely to have a steroid response compared with nonglaucoma patients (P < 0.001). Corticosteroid-induced IOP elevation involves reduced trabecular aqueous humor outflow associated with morphologic and biochemical changes in the trabecular meshwork (TM).9,17 Several genes are upregulated in dexamethasone-treated TM, with the myocilin gene being the most extensively studied.17 In addition, the prevalence of myocilin mutation is associated with a variety of glaucoma phenotypes in both juvenile and adult-onset POAG.18 Myocilin is highly expressed in trabecular cells exposed to glucocorticoids, and the delay in its expression is similar to the delay in the IOP elevation in steroid responder eyes. In addition, the dose required to cause the protein expression is similar to that needed to raise IOP.19 In our study, the cumulative dose of prednisolone acetate 1% was similar between steroid responders and nonresponders (P = 0.168), indicating that patient-specific factors such as genetic susceptibility may be important in development of steroid response. Interestingly, among steroid responders, the cumulative dose was similar for nonglaucoma and for glaucoma (102 ± 40 vs. 112 ± 37 drops, respectively; P = 0.401). Although the number of affected eyes was small, we did not find effects of steroid response on IOP control at 6 months after surgery in glaucoma patients.

Chang et al10 found in their cohort that long AL was a risk factor for steroid response. They theorized the possibility of predisposing genes linked to high myopia and steroid response, or molecular and structural changes that lead to abnormal globe elongation might also be associated with
TABLE 2. Clinical Characteristics of Glaucoma Patients Studied, Including Risk Factors for Postoperative Steroid Response After Phacoemulsification (N=191 Eyes)

| Risk Factor             | Steroid Response (N = 16, 8.4%) | No Steroid Response (N = 175, 91.6%) | P |
|-------------------------|---------------------------------|-------------------------------------|---|
| Age (y)                 | 69.9 ± 11.2                     | 72.0 ± 9.8                          | 0.425*|
| Sex (male), n (%)       | 7 (43.7)                        | 76 (43.4)                           | 1.000‡|
| Race, n (%)             |                                 |                                     |   |
| Caucasian               | 13 (81.2)                       | 122 (69.7)                          | 0.550†|
| Asian                   | 1 (6.3)                         | 24 (13.7)                           |   |
| Black                   | 0                               | 12 (6.9)                            |   |
| Other                   | 2 (12.5)                        | 17 (9.7)                            |   |
| Glaucoma type (POAG)    | 7 (43.7)                        | 76 (43.4)                           | 0.302†|
| Visual Field            |                                 |                                     |   |
| MD (dB)                 | −7.46 ± 6.47                    | −6.94 ± 7.40                        | 0.789*|
| PSD (dB)                | 4.92 ± 3.71                     | 5.13 ± 3.74                         | 0.835*|
| Preoperative VA (LogMAR)| 0.296 ± 0.213                   | 0.317 ± 0.371                       | 0.821*|
| Cup:disc ratio          | 0.76 ± 0.15                     | 0.67 ± 0.20                         | 0.099*|
| Preoperative spherical equivalent | −3.3 ± 4.81 | −0.92 ± 3.34 | 0.020*|
| Cataract density (0-4)  | 2.28 ± 0.41                     | 2.41 ± 0.73                         | 0.490*|
| Average preoperative IOP (mm Hg) | 14.2 ± 4.0 | 16.5 ± 4.3 | 0.048*|
| Preoperative medications | 2.62 ± 0.88                    | 2.00 ± 1.66                         | 0.028∥|
| Any preoperative laser‡ | 4 (25.0)                       | 61 (34.9)                           | 0.426‡|
| LPI                     | 2 (12.5)                        | 41 (23.4)                           | 0.531‡|
| LTP                     | 3 (18.8)                        | 28 (16.0)                           | 0.728†|
| LPI and LTP, n (%)      | 5 (6.3)                         | 8 (4.6)                             | 0.553‡|
| CCT (µm)                | 535.5 ± 31.7                    | 542.9 ± 40.8                        | 0.499*|
| AL (mm)                 | 25.6 ± 2.2                      | 24.0 ± 1.5                          | 0.011*|
| AL > 26.0 (mm), n (%)   | 4 (25.0)                        | 18 (10.3)                           | 0.024*|
| ACD (mm)                | 3.07 ± 0.38                     | 2.98 ± 0.44                         | 0.436*|
| Pupil enlargement (yes), n (%) | 3 (18.7) | 27 (15.4) | 0.721‡|
| CME (yes), n (%)        | 0                               | 4 (2.3)                             | 1.000‡|
| Persistent AC reaction, n (%) | 3 (18.7) | 15 (8.6) | 0.180‡|
| Cumulative prednisolone dose (drops) | 111.5 ± 36.9 | 98.0 ± 19.7 | 0.168*|
| Postop VA (LogMAR)      | 0.073 ± 0.100                   | 0.135 ± 0.321                       | 0.451*|

Bold values indicate statistical significance (P < 0.05).
*Two-tailed independent sample t test.
†The Fisher exact test.
‡The Pearson χ² test.
∥Patients having both LPI and LTP are also counted in the LPI and LTP rows.
ACD indicates anterior chamber depth; AL, axial length; CCT, central corneal thickness; CME, cystoid macular edema; IOP, intraocular pressure; LPI, laser peripheral iridotomy; LTP, laser trabeculoplasty; POAG, primary open-angle glaucoma; PSD, pattern standard deviation; steroid response, >50% increase in IOP from baseline at postoperative month 1; VA, visual acuity.

TABLE 3. Logistic Regression of Risk Factors for Steroid Response After Phacoemulsification

| No Glaucoma (N = 462) | Odds Ratio | 95% CI | P   |
|-----------------------|------------|--------|-----|
| Axial length, per mm  | 1.826      | 1.374-2.427 | <0.001|
| Age, per year         | 0.914      | 0.862-0.970 | 0.003|
| Glaucoma (N = 191)    |            |        |     |
| Axial length, per mm  | 1.813      | 1.313-2.505 | <0.001|
| Preoperative medications, per medication | 1.774 | 1.058-2.973 | 0.030|

CI indicates confidence interval.

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morphologic alterations in the TM. In our study, logistic regression showed that longer AL was a risk factor in both nonglaucoma and glaucoma patients (P = 0.001). Some authors have proposed that intrinsic differences in the biomechanical properties of patients with longer ALs might contribute to the ability of the aqueous outflow pathway to accommodate stressors such as cataract surgery.20 Prior ultrasound biomicroscopy study has shown that TM thickness is smaller in POAG eyes compared with normal eyes.21 In addition, prior optical coherence tomography studies have shown that (1) the area and perimeter of Schlemm canal in POAG is smaller than in normal eyes,22 (2) highly myopic eyes without glaucoma have smaller TM thickness and width compared with nonmyopic eyes,23 (3) highly myopic eyes with early postoperative IOP elevation after cataract surgery have smaller Schlemm canal vertical diameter and area compared with nonhighly myopic eyes.24 In addition, corticosteroids have been shown to cause stiffening of the TM.25 We postulate that a thinner and potentially less cellular TM in high myopia has a more limited ability to maintain normal function with exposure to glucocorticoids, leading to relatively rapid IOP elevation.

Previous studies14,26,27 indicate that male sex is associated with longer AL, and this finding was reproducible in our population (P < 0.001). While a higher proportion of steroid responders in our study were male, this was not significant in regression analysis.

Chang et al41 reported younger age as a risk factor for steroid responsiveness. In their cohort, patients 40 to 54 years old with an AL ≥ 29.0 mm had a 46-fold increase in the risk of steroid response compared with their reference group (patients > 65 y with AL <25.0 mm). Other studies have also reported younger age as a risk factor for steroid response after intra-vitreal triamcinolone injection.16,28 In the pediatric population, an IOP increase related to topical steroids has been reported to occur rapidly and to higher levels than in adults.9,30 and carries a higher risk of developing steroid-induced glaucoma.31,32 Our study findings in nonglaucoma patients were consistent with these prior studies; younger age at the time of surgery was associated with steroid response (P = 0.003). We did not find this in glaucoma patients; therefore, our findings suggest the presence of glaucomatous changes in the TM is a more prominent risk factor for steroid response than age.

Our study has some limitations. The retrospective nature of our study may have resulted in errors related to inaccurate and incompleteness of data recording, and variability of postoperative regimens and follow-up. Although the phacoemulsification technique did not change substantially during the span of the study, changes in equipment and management philosophy likely occurred. Lastly, most (>70%) of the patients in our study were Caucasian, which may limit the generalizability of our findings to other populations.

In summary, we found that, although glaucoma patients were 3.72 times more likely to have a steroid response after phacoemulsification prednisolone acetate 1% use compared with nonglaucoma patients, the incidence of steroid response was relatively low in both groups. In addition, glaucoma and nonglaucoma patients with longer ALs were at elevated risk for a steroid response after phacoemulsification. Better identification of patients at risk for steroid-induced IOP elevation can aid in the clinical management of glaucoma patients. Further research is warranted.
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