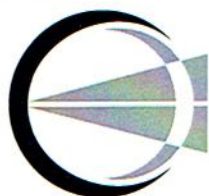


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SPRING 2016



Diagnosing the Causes of Photopsia

Photopsia—the perception of light accompanying a pathologic condition especially of the retina or brain—results from a multitude of causes. Brown et al from the Wills Eye Hospital at Jefferson Medical College, Pennsylvania, reviewed 169 patient records (217 eyes) presenting with photopsia to explore the causes and characteristic clinical features.

The median age of the patients was 63 years. Of the 50 patients with bilateral photopsia, 21 had simultaneous photopsia, suggesting a central nervous system (CNS) origin. Although the researchers identified 32 causes of photopsia, 3 causes accounted for >70% of cases.

A total of 122 eyes (56.2%) had vitreoretinal traction, including 86 eyes with posterior vitreous detachment, 19 eyes with retinal tears, 15 eyes with rhegmatogenous retinal detachment and 2 eyes with traction retinal detachment. New floaters accompanied photopsias in 85% of these eyes. Most photopsias were <1 second in length and had the morphology of a flash.

The 25 eyes (11.5%) included 18 eyes with neovascular age-related macular degeneration (AMD) and 7 eyes with neovascular macular degeneration secondary to myopic degeneration, angioid streaks or presumed ocular histoplasmosis. Unlike photopsias associated with vitreoretinal traction, photopsias in 76% of eyes with neovascular macular degeneration lasted ≥ 1 second. Flashes were centrally located in approximately 80% of these eyes, compared with 18% of eyes with vitreoretinal traction.

Six of the 8 patients with migraine-associated photopsias had bilateral symptoms; 1 patient with 1 eye had a unilateral symptom. The photopsias were constant, lasted from 1 to 30 min-

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utes (mean, 16 minutes) and traveled across the visual field.

Other causes in ≥ 4 eyes included diabetes mellitus accompanied by hypoglycemia or hyperglycemia, choroidal neovascularization not associated with AMD, vertebrobasilar insufficiency, severe cough with acute respiratory infection, retinitis pigmentosa and central serous chorioretinopathy. Posterior vitreous detachment and migraine were frequent causes of bilateral photopsias.

Variations in the morphology of photopsias aid in the diagnosis of underlying causes and symptom management. Photopsia location is a critical sign; temporal photopsias suggest vitreoretinal traction, while central photopsias suggest macular diseases, CNS abnormalities or systemic abnormalities. Bilaterality is associated with CNS abnormalities or systemic conditions.

Brown GC, Brown MM, Fischer DH. Photopsias: a key to diagnosis. Ophthalmology 2015;122:2084-2094.

Long-term Outcomes for Traumatic Macular Holes

Traumatic macular holes arising from sudden extrinsic force, often blunt trauma, create forces within the sclera and vitreous leading to retinal pathology, including commotio retinae, choroidal rupture, sclopetaria and retinal breaks. These sequelae to traumatic macular holes cause vision loss. Long-term data on the history of traumatic macular holes is scarce.

Miller et al from Harvard Medical School, Massachusetts, presented a retrospective long-term follow-up case series of traumatic macular holes treated at a tertiary referral center, including 28 patients seen from July 2007 to September 2012 and followed for a mean of 2.2 years.

Macular holes were examined using spectral-domain ocular coherence tomography for diameter, height, con-

figuration, and the presence of cystoid edema and subretinal fluid. Researchers noted the location and the type of injury, the time interval to surgery and the type of surgery. Visual acuity (VA) was measured at the initial, preoperative and final visits.

At presentation, 28 patients had eye injuries that included

- 2 open globe
- 17 hyphema
- 13 vitreous hemorrhage
- 1 posterior vitreous detachment
- 10 foveal commotio
- 3 choroidal rupture

Mean VA at presentation was the logarithm of the minimum angle of resolution (logMAR) 1.3. Spontaneous closure occurred in 11 of the macular holes at a median of 5.6 weeks; all but 2 of the closures occurred by 11 weeks (range, 1.7–67.3 weeks). The patients whose macular holes closed spontaneously were younger (mean age, 17.6 years) than those for whom spontaneous closure did not occur (mean age, 23.5 years).

Successful closure occurred for 5 of the 11 patients who opted to undergo vitrectomy. The median time to intervention for the 5 patients with a successful outcome was 11 weeks compared with 56 weeks for the 6 patients with an unsuccessful outcome, a significant difference

Table 1. Median VA (logMAR) in eyes with traumatic macular holes

	VA at baseline	VA at final follow-up	p value
All eyes (n = 28)	1.15	0.70	<.001
All eyes with hole closure (n = 16)	1.30	0.42	<.001
Eyes with spontaneous hole closure (n = 11)	1.30	0.30	.003
Eyes that underwent vitrectomy (n = 11)	1.20	0.80	.016
Eyes with surgical hole closure (n = 5)	1.60	0.60	.043

($p = .017$). VA improved for all eyes once the macular hole closed (Table 1), but not in those eyes in which the hole did not close.

Given the large number of traumatic macular holes that closed spontaneously within the first 11 weeks, the authors recommended an initial course of observation for 2 to 3 months. Delayed intervention had a lower rate of success, so surgical repair should be pursued if the hole has yet to close within 3 months of presentation.

Miller JB, Yonekawa Y, Elliott D, et al. Long-term follow-up and outcomes in traumatic macular holes. Am J Ophthalmol 2015;160:1255-1258.

Cataract Surgery in Patients with Wet AMD

Recent advances in the treatment of wet age-related macular degeneration (AMD) resulted in the improvement of vision for many patients. Ironically, this improvement in vision has created a situation in which cataracts developed in wet AMD patients. Wet AMD being a new phenomenon, little evidence exists for results of cataract surgery in patients undergoing active treatment for it.

Saraf et al from Henry Ford Health Systems, Michigan, conducted a retrospective cohort study to evaluate visual outcomes and possible complications of cataract surgery in wet AMD patients. The authors reviewed the charts of patients with wet AMD (defined as eyes that received ≥ 1 anti-vascular endothelial growth factor [VEGF] injection in the 1-year study window) who had cataract surgery between January 2008 and May 2013. They created a nonsurgical control group from patients with phakic eyes who were receiving anti-VEGF injections for active wet AMD in 2012 without having cataract surgery.

Data included the number and type of anti-VEGF injections and any changes in anti-VEGF injections during the 6 months before and after the midpoint (defined as the date of cataract surgery for the surgical cohort and July 1, 2012, for the

nonsurgical cohort). Best-corrected visual acuity (BCVA) and central optical coherence tomography (OCT) thickness were recorded at 3 months before and after the midpoint, as was the presence of macular hemorrhage.

BCVA in the 40 eyes (39 patients) in the surgical cohort improved from 20/89 at 3 months prior to midpoint to 20/53 at 3 months after midpoint, a greater improvement than in the 42 eyes (38 patients) of the control cohort (20/89 to 20/69). The average number of anti-VEGF injections remained unchanged in the surgical cohort (2.31 before midpoint and 2.30 after midpoint) but decreased in the control cohort from 3.00 to 2.57. Final central OCT thickness was 265.4 μm in the surgical eyes and 216.4 μm in the nonsurgical eyes, a significant difference, while the surgical eyes had worse cysts after midpoint.

This study suggested that cataract surgery in eyes with active wet AMD led to improved BCVA without increasing the need for anti-VEGF treatments. However, the trend toward increased central macular thickness and intraretinal cysts in eyes that underwent surgery emphasizes the need for careful preoperative patient selection and close postoperative monitoring.

Saraf SS, Ryu CL, Ober MD. The effects of cataract surgery on patients with wet macular degeneration. Am J Ophthalmol 2015;160:487-492.

Prophylaxis for Macular Edema After Cataract Surgery

Postoperative macular edema, a complication of cataract surgery, can have a negative effect on long-term vision. Evidence is lacking to show a benefit from prophylaxis. The U.S. Food and Drug Administration has yet to approve a prophylactic drug to prevent macular edema after cataract surgery.

Shorstein et al from Kaiser Permanente, California, conducted a retrospective cohort study



comparing the effectiveness of prophylaxis against macular edema after cataract surgery with topical prednisolone acetate (PA) alone or with a topical nonsteroidal anti-inflammatory drug (NSAID) added to PA and subconjunctival injection of 2 mg triamcinolone acetonide (TA). The study included 16,070 eyes after phacoemulsification for cataract from 2007 through 2013 for which optical coherence tomography (OCT) was available.

Macular edema was defined as best Snellen distance visual acuity of 20/40 or worse on the day of diagnosis and central subfield thickness of >250 μm (as measured by time-domain OCT) or >320 μm (as measured by spectral-domain OCT) at 5 to 120 days postoperatively. Analysis included the prophylaxis used, patient demographics, comorbidities and operating time. Adverse events—except macular edema—and intraocular pressure (IOP) were recorded.

Postoperative macular edema was associated with prophylaxis with PA alone, black ethnicity and a history of diabetic retinopathy, epiretinal membrane, prior iritis or uveitis, and posterior capsular rupture (Table 2). After adjusting for confounding variables, the odds ratio (OR) for patients receiving prophylaxis with PA and NSAID was 0.45 (95% confidence interval [CI], 0.21–0.95) when compared with those receiving PA alone. Removal of patients with comorbidities associated with postoperative macular edema showed that the OR remained significant at 0.35 (95% CI, 0.13–0.97). But the OR for patients receiving TA injection was 1.21 (95% CI, 0.48–3.06) when compared with patients receiving PA alone. No change resulted from removal of patients with comorbidities associated with postoperative macular edema (OR, 0.83; 95% CI, 0.20–3.37).

Prophylactic regimen is unrelated to adverse events. Spikes of ≥30 mm Hg in IOP were fre-

Table 2. Univariate analysis for characteristics of macular edema cases after phacoemulsification surgery

Characteristic	% of phacoemulsification surgeries (n = 16,070)	% of macular edema cases (n = 118)	p value
Race/ethnicity			
Black	2.6	7.6	<.001
Asian	10.5	12.7	.43
Hispanic	7.3	5.9	.57
White	78.4	70.4	.03
Other/unknown	1.2	3.4	.03
Ocular comorbidity^a			
None	56.4	44.9	.01
Glaucoma	25.7	28.0	.57
Macular degeneration	13.7	13.6	.96
Diabetic retinopathy	5.7	12.7	.001
Epiretinal membrane	5.4	13.6	<.001
Prior iritis/uveitis	3.3	6.8	.04
Posterior capsular rupture	1.1	4.2	.001
Prophylaxis			
Topical PA alone	57.5	68.7	.03
Topical PA + NSAID	29.7	18.6	
Injected TA	12.8	12.7	

^aPercentages add up to >100% because of eyes with >1 comorbidity.

quent in the PA-alone group on postoperative days 1 through 3, but were more frequent in the PA + NSAID group on postoperative days 4 through 15.

This study showed a 0.73% rate of macular edema after phacoemulsification surgery for cataract. The addition of a topical NSAID to topical PA as prophylaxis resulted in a 55% reduction in the risk of macular edema.

Shorstein NH, Liu L, Waxman MD, Herrinton LJ. Comparative effectiveness of three prophylactic strategies to prevent clinical macular edema after phacoemulsification surgery. Ophthalmology 2015;122:2450-2456.

SUMMER 2016

- Fluoroquinolones and uveitis risk
- Metamorphopsia following retinal detachment
- Hydroxychloroquine toxicity

In The Next Issue