CME from the Anterior Segment Surgeon’s Perspective

What the posterior segment specialist should know.

BY ERIC D. DONNENFELD, MD

The definition of cystoid macular edema (CME) has evolved since the condition was first described by Irvine in the 1950s and further explicated by Gass in the 1960s. In those days of intracapsular cataract extraction, CME was the most common cause of poor visual outcome after cataract surgery. Despite many ensuing advances in technique and technology, it can still be the cause of disappointing visual results today. Increased patient expectations and the advent of advanced refractive IOLs now make the prevention and management of CME crucial to improving surgical outcomes.

The traditional description of CME included a cystic, yellow appearance in the fovea on ophthalmoscopy, leakage in the macula in a petaloid formation on fluorescein angiography (Figure 1), and decrease in visual acuity to 20/40 or worse.

Gass reported that angiographically detected CME reached its peak at about 6 weeks following cataract surgery. However, not all CME that was evident on angiography caused visual loss. This led to a distinction between angiographic CME and clinically significant CME, in which decreased visual acuity is noted.

As patients’ and surgeons’ expectations have risen in recent years, the degree of visual acuity reduction that is tolerated after cataract surgery has diminished. There has been a shift of focus from quantity of vision, as measured by 100% contrast Snellen acuity, to quality of vision, including measures such as contrast sensitivity with and without glare and mesopic visual acuity. With...
the advent of multifocal IOLs, which inherently decrease contrast sensitivity, these types of measures have become yet more important. Even subtle changes in macular thickness can affect these real-world measures of vision.

The current definition of CME for anterior segment surgeons incorporates all characteristics of the traditional definition but in addition now includes any visual deficit, no matter how small—20/25 or even a hesitation on the 20/20 line. Any metamorphopsia or image distortion is also considered a clue to the presence of CME, along with other parameters such as decreased contrast sensitivity.

In suspicious cases, optical coherence tomography (OCT) may detect cystoid formations in the macula and evidence of subretinal fluid earlier than ophthalmoscopy or fluorescein angiography (Figure 2), even before visual acuity or contrast sensitivity is affected.

The sensitive measurement possible with OCT suggests that the incidence of CME after routine cataract surgery is greater than previously thought. Lobo and colleagues measured retinal leakage with scanning laser ophthalmoscopy (CSLO, Carl Zeiss Meditec, Jena, Germany) and OCT (Humphrey Instruments) in 32 eyes at 3, 6, 12, and 30 weeks after uneventful cataract surgery. Almost all eyes (97%) showed evidence of retinal thickening compared with controls at some point during follow-up. Increase in retinal thickness reached a peak at 6 weeks postoperative, when 41% of eyes showed increased retinal thickness, even with visual acuities better than 20/25. During the first 12 weeks, macular leakage was seen in 88% of eyes; this percentage decreased to 68% at 30 weeks, suggesting a trend toward recovery. At 30 weeks, visual acuity was good in all eyes, but retinal thickening was still seen in 22% of eyes.

HOW TO TREAT

Luckily, as ophthalmic diagnostic instrumentation has improved, it has become possible to detect accumulation of fluid at subclinical levels on OCT, before visual acuity is affected, so that treatment can be initiated earlier in the course of disease. Still, visual deficits due to CME can persist in some cases permanently, even when the edema is successfully treated. Therefore, prevention of CME is paramount for the cataract surgeon.

Several studies suggest that use of a nonsteroidal antiinflammatory drug (NSAID) can reduce the incidence of CME after cataract surgery. McColgin and Raizman reported that 12% of patients with no relevant risk factors developed visual changes in association with macular edema after routine cataract surgery. In patients treated prophylactically with an NSAID, however, no patients developed CME.

More recently, Henderson and colleagues reported a 2.4% incidence of CME with use of prophylactic NSAIDs in more than 1,600 consecutive cases of cataract surgery performed by residents over 5 years. When patients with diabetes were excluded, the rate of CME was 2.1%.

I participated in a prospective study to compare the efficacy of several prophylactic regimens with the topical ophthalmic NSAID ketorolac tromethamine 0.4% (Acular LS, Allergan). One-hundred patients were randomly assigned to one of four groups; three groups received pre- and postoperative NSAID and the fourth received placebo. In the three treatment groups, patients received NSAID either for 3 days preoperatively or 1 day preoperatively or for 1 hour preoperatively. All patients in the treatment groups also received the NSAID postoperatively four times daily for 2 weeks.

Pretreatment with NSAID four times daily for 1 or 3 days improved visual acuity outcomes significantly in the immediate postoperative period (at 1 day and 2 weeks) compared with pretreatment for 1 hour or placebo (P<.05 for all). Eyes pretreated for 1 or 3 days had 0% incidence of CME, vs 12% in the placebo group and 4% in the 1-hour group. These differences did not reach statistical significance.

Wittpenn and colleagues demonstrated a statistically significant difference in the incidence of CME in patients treated prophylactically for 3 days before cataract surgery with ketorolac tromethamine plus steroids compared with patients receiving steroids only. Those investigators also showed an association between retinal thickening and postoperative quality of vision as measured by contrast sensitivity.

NSAIDs inhibit the production of prostaglandins; they do not affect prostaglandins that have already been formed. Therefore, it is important to begin NSAID
treatment sufficiently in advance of surgery to inhibit the formation of prostaglandins to inhibit the formation of prostaglandins in response to surgical insult. While postoperative NSAID use is important to control pain and inflammation, preoperative treatment is essential to improve the results of surgery.

WHEN WE REFER

Patients with CME after cataract surgery who are seen by retina subspecialists, for the most part, are refractory or challenging cases—patients that the anterior segment surgeon has tried initially to manage or who have complex histories that we suspect will not respond to typical management strategies.

Like most cataract surgeons, I treat most patients with CME myself. For patients with macular thickening on OCT after surgery, I prescribe a topical NSAID and a topical steroid four times daily for 1 month, and if the patient demonstrates no or little improvement on OCT, at that point I will refer him or her to a retina subspecialist.

If, however, the patient’s first OCT shows frank cyst formation or other structural changes to the retinal architecture, or if the patient has significant visual loss, I tend to refer that patient to the retinal specialist rather than treating him myself for a month. In addition, patients with complicated medical histories, such as those with diabetes, epiretinal membranes, venous occlusive disease, or other high-risk characteristics, are referred early.

I also sometimes refer patients for psychosocial reasons, such as those who have extraordinarily high expectations. I feel they may respond better in the hands of the appropriate subspecialist.

Retina subspecialists report a number of approaches to management of patients referred with refractory CME after cataract surgery. Management strategies may include sub-Tenon’s or intravitreal injection of triamcinolone acetonide (Triesence, Alcon Laboratories, Inc.), focal laser treatment in patients with existing diabetic retinopathy, or intravitreal injection of a vascular endothelial growth factor inhibitor in patients with concomitant choroidal neovascularization.

If CME is refractory to medical management, or if there is an anatomical problem such as vitreous incarceration in the wound or IOL-iris chafe, pars plana vitrectomy may be necessary.

CONCLUSIONS

As noted above, McColgin reported visual changes associated with macular edema in 12% of patients with no relevant risk factors after routine cataract surgery. The incidence is higher, obviously, for patients with risk factors for CME. Given that approximately 3 million cataract surgeries are performed in the United States each year, there is a potential for reduced vision due to CME in more than 350,000 patients annually.

It is vital that posterior segment subspecialists understand how the definition of CME has changed in recent years, and the importance of proper management of CME to ensure good visual outcomes in patients after modern cataract surgery. More research into the best methods for prophylaxis and treatment of CME is necessary, especially as patients’ expectations for excellent quality vision after cataract surgery with premium IOL implantation increase.

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