

# Perioperative Pharmacology in Cataract Surgery

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Preoperative and postoperative medications for cataract surgery have the potential to prevent the development of postoperative infections including endophthalmitis; potentiate the effect of pupil-dilating drops; decrease the discomfort associated with the surgery; prevent the development of postoperative inflammation; and inhibit the development of cystoid macular edema.

Through an evidenced-based review of clinical studies, the optimum treatment with antibiotics, steroids, and nonsteroidal ophthalmic medications of patients undergoing cataract surgery can be determined. This paper reviews the key clinical studies and recommendations for the most efficacious treatment of cataract patients.

## Introduction

Today's patients expect that their surgery will be rapid and painless, that they will be able to return to normal activity immediately afterward, that they will have total recovery of visual acuity, and that there will be no complications. While cataract surgery has a very low incidence of complications, patients who develop endophthalmitis or cystoid macular edema often do not achieve the postoperative visual improvement they expected.

Advances in the instrumentation of cataract surgery have dramatically improved the outcomes of this procedure but have not eliminated all complications. Thus, cataract surgeons depend on perioperative pharmaceuticals to control inflammation and prevent infection. The judicious use of antibiotics and anti-inflammatories can reduce the incidence of these infrequent complications.

## Cystoid Macular Edema

Cystoid macular edema (CME) may develop in association with a wide variety of ocular conditions. It is the result of cystic accumulation of extracellular intraretinal fluid in the outer plexiform and inner nuclear layers of the retina, due to the breakdown of the blood-retinal barrier. In chronic CME, fluid accumulation is associated with thinning of the retina and fibrosis. It is most common following intraocular surgery and in patients with venous occlusive disease, diabetic retinopathy, and posterior segment inflammatory conditions.

While the clinical syndrome of cystoid macular edema was described by Irvine in 1953, the pathogenesis of this condition remains unclear. Five major causes of CME are:

1. photic retinopathy
2. trauma to ocular tissues
3. secondary irritation of the ciliary body
4. vitreous traction
5. pharmaceutically induced CME

Cystoid macular edema is often separated into two entities: angiographic CME, where evidence of edema is present anatomically and may be identified by fluorescein angiography, but there is no clinical effect on the patient's vision; and clinical CME, where the patient has a decrease in visual acuity and/or contrast sensitivity.<sup>1</sup>

In the past, clinical CME was the diagnosis if a patient after surgery saw only 20/40 or 20/50 as a result of cystic accumulation of fluid in the macula. With today's refined surgery, patients rarely have this degree of visual impairment. Often the visual deficit may be only a decrease in contrast sensitivity, a sense of "grayness" to the central vision, or the inability to see the letters in the center of the Snellen line as well as the ones at the ends. A patient may have 20/20 Snellen acuity and still have clinical CME. So today, we consider clinical CME to be any decrease in visual function as a result of edema of the macula.

This approach to the prophylaxis of cystoid macular edema begins with the identification of patients who are at highest risk for the development of CME. Over the years, many clinical settings have been associated with the development of cystoid macular edema, and they need to be reviewed.

## Cystoid Macular Edema Associated With Surgical Procedures and Complications

Broken posterior capsule

Two mechanisms have been offered to explain the development of CME following breakage of the posterior capsule:

1. Loss of integrity of the anterior segment. The posterior capsule may act as a barrier preventing the diffusion into the posterior segment of prostaglandins, the mediators of inflammation produced in the ciliary body. When this anatomical barrier is lost, prostaglandins produced in the anterior chamber may migrate into the posterior segment. The macula is very sensitive to prostaglandins, thus inflammation develops in the retina, resulting in retinal edema.<sup>2</sup>
2. Vitreous traction. Breakage of the posterior capsule allows vitreous to migrate anteriorly. As the vitreous gel moves forward, traction is created on the posterior attachment of the vitreous to the retina. The traction creates inflammation as well as the development of cystic spaces within the outer plexiform layer of the retina.<sup>3</sup>

## Retained lens fragments

Fragments of the nucleus of the lens are antigenic, due to the embryologic development of the nucleus of the lens prior to the development of the blood supply to the eye. Thus, when fragments of the lens nucleus are retained in the vitreous, inflammation develops, which leads to the development of macular edema.<sup>4,5</sup> Surgical removal of retained lens fragments results in decreased incidence of CME compared to eyes where the inflammation is treated medically.<sup>6,7</sup>

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### Misplaced intraocular lens

Intraocular lens implants (IOLs) that chafe the iris or are imbedded in the ciliary body, root of the iris, or trabecular meshwork may induce inflammation and the production of elevated levels of prostaglandins.<sup>8</sup>

### Secondary intraocular lens implantation

Whether placed in the anterior chamber or sutured into the posterior chamber, secondary IOLs are associated with a higher incidence of CME than primary IOL placement at the time of cataract surgery. The incidence of CME after suturing a posterior chamber IOL is reduced when a vitrectomy is performed simultaneously. Presumably, the vitrectomy clears away vitreous strands that would otherwise become imbedded in either the suture or the haptics of the IOL, resulting in traction on the macula.<sup>9-11</sup>

### Vitrectomy

Patients undergoing vitrectomy may develop cystoid macular edema. This may be due to the inherent inflammation resulting from tissue injury or from tugging and traction on the macula during and after the surgical procedure.<sup>12</sup>

### Iris incarceration

Iris incarceration was frequently used in the past during glaucoma operations, where the stroma of the iris acted as a filter to allow aqueous humor to percolate out of the anterior chamber in a controlled manner. Today iris incarceration may develop during or after routine cataract surgery. Trauma to the vascular iris is often associated with the development of inflammation and the release of elevated levels of prostaglandins.<sup>13</sup>

### YAG laser capsulotomy

While some studies report a zero incidence of cystoid macular edema following YAG laser capsulotomy,<sup>14</sup> others report a low incidence of approximately 1%.<sup>15</sup> Given the frequency of YAG laser capsulotomy in the United States—approaching one million procedures per year—even a low incidence may result in thousands of affected eyes.

### Technique of cataract surgery

Extracapsular cataract extraction (ECCE) has been associated with a lower incidence of CME than intracapsular cataract extraction.<sup>16</sup> While phacoemulsification through a small incision is associated with lower levels of postoperative inflammation than with larger incision planned ECCE, some studies have found no difference in incidence of CME between these two techniques.<sup>17</sup> The newer Erbium YAG technology of laser cataract surgery seems to be associated with incidence of postoperative CME similar to that of phacoemulsification.<sup>18</sup>

### Cystoid Macular Edema Associated With Preexisting Medical and Ocular Conditions

Posterior vitreous detachment

To the extent that the CME results from mechanical traction of the vitreous gel against the internal limiting membrane of the retina, it has been suggested that a preexisting posterior vitreous detachment (PVD) would protect the eye from the development of CME. The results of several studies are inconclusive.

### Aphakic bullous keratopathy

While corneal endothelial dysfunction and the resultant aphakic bullous keratopathy (ABK) is a localized corneal disorder, patients with ABK are associated with a high incidence of CME. Prompt surgical treatment with penetrating keratoplasty may result in a lower incidence of permanent vision-degrading CME, compared to cases where penetrating keratoplasty is delayed.

### Uveitis

Patients with a previous history of inflammatory eye disease tend to develop elevated levels of inflammation after cataract surgery. Thus, these patients are at greater risk for the development of postoperative CME.<sup>19,20</sup>

### Diabetes and diabetic retinopathy

While some studies have identified a progression of diabetic retinopathy and CME after routine cataract surgery,<sup>21-23</sup> other studies have not identified a significant difference.<sup>24</sup> Some studies have demonstrated an increase in angiographic CME with no difference in rates of clinical CME. Diabetics develop more postoperative inflammation with breakdown of the blood aqueous barrier compared to nondiabetics.<sup>25</sup>

### Branch retinal vein occlusion

As a venous occlusive disease, branch retinal vein occlusion (BRVO) often results in CME, and patients with preexisting BRVO undergoing cataract surgery are thus at a higher risk for the development of CME.<sup>26</sup>

### Ischemic heart disease

Patients with ischemic heart disease (IHD) have a higher incidence of CME than those who do not.<sup>27</sup> Since cataract surgery is performed mostly on a geriatric population with a high incidence of IHD, cataract surgery patients comprise a large group that is at risk for CME.

### Increased preoperative retinal thickness

With the development of new technologies of ultrasound and optical coherence tomography, retinal thickness can be measured with greater precision than in the past.<sup>28, 29</sup> Patients with an increased

preoperative retinal thickness have been found to have a higher incidence of postoperative CME than those with a lesser preoperative retinal thickness.<sup>30</sup>

### Drugs That May Predispose Patients to the Development of Cystoid Macular Edema

Latanoprost and other hypotensive lipids

Because these ocular hypotensives are derivatives of prostaglandins—the primary mediators of inflammation—there has been a debate as to whether the clinical introduction of these drugs for the treatment of glaucoma would predispose patients to the development of postoperative CME. While some studies have shown an increased rate of CME in latanoprost-treated patients,<sup>31-33</sup> others have failed to demonstrate a significant difference in CME rates.<sup>34-35</sup> Other studies have shown that the concomitant use of nonsteroidal anti-inflammatory drugs (NSAIDs) are protective against the adverse action of latanoprost.<sup>33</sup>

### Other chronic glaucoma drugs

The preoperative use of both epinephrine<sup>31</sup> and betaxolol<sup>36</sup> has been associated with the development of elevated rates of cystoid macular edema following subsequent cataract surgery.

### Vancomycin in the irrigating infusion during cataract surgery

As prophylaxis against the development of postoperative bacterial endophthalmitis, many ophthalmic surgeons add vancomycin, an antibiotic that is highly effective against gram-positive bacteria, to the irrigating solution. One study has demonstrated a higher incidence of postoperative CME in patients where vancomycin was added to the irrigating solution compared to patients who did not receive vancomycin in their irrigating solution.<sup>37</sup>

### The prophylaxis of postoperative cystoid macular edema with NSAIDs

While we believe that all patients benefit from preoperative prophylaxis with topical ophthalmic NSAIDs, certainly the above patient groups are at particular risk for the development of CME. Of particular concern to us are the glaucoma patients on prostaglandin derivatives, and we try to stop these medications for a week prior to cataract surgery when possible.

NSAIDs are potent inhibitors of prostaglandin synthesis. Topical ophthalmic NSAIDs are FDA-approved to prevent intraoperative miosis during cataract surgery, to reduce postoperative inflammation following cataract surgery, and to control symptoms of allergic conjunctivitis and pain and photophobia following refractive surgery. While CME is not an FDA-approved indication for the use of ophthalmic NSAIDs, the significance of postoperative inflammation and the role of prostaglandins as an offending agent is well established and suggests the benefit of NSAIDs both in the prophylaxis and treatment of CME.

Inflammation develops as a result of tissue injury. Tissue injury activates phospholipase A2, which breaks down cell membranes to arachidonic acid. Arachidonic acid is then converted to

either prostaglandins by cyclo-oxygenase or to leukotienes and hydroxy acids by 5-lipoxygenase. While corticosteroids act to inhibit the action of phospholipase A2 and stabilize cell membranes, NSAIDs act by inhibiting the action of cyclo-oxygenase, thus directly blocking the production of prostaglandins. This is why NSAIDs are believed to be so effective in both the prophylaxis and treatment of prostaglandin-mediated conditions such as CME.<sup>38-43</sup>

Prostaglandins are produced primarily in the ciliary body and the iris. They act locally to produce vasodilation and a breakdown of the blood-aqueous barrier, resulting in anterior segment inflammation. Prostaglandins may diffuse into the posterior segment of the eye to the retina, producing posterior segment inflammation and macular edema. One theory is that when retinal edema develops, it drains posteriorly to the macula. Thus the macula acts as the sink of the retina. If the macula is unable to drain all the fluid that accumulates there, the fluid will persist in the form of CME.

While both corticosteroids and NSAIDs act to decrease inflammation, NSAIDs have been shown to be more effective in the prophylaxis and treatment of CME than are steroids.<sup>44,45</sup> Also, patients with CME treated with topical NSAIDs develop less loss of visual acuity than those not treated with steroids.<sup>46</sup>

While some cataract practices begin treating their patients with NSAIDs after cataract surgery, we recommend beginning NSAID therapy preoperatively. By pretreating patients with topical NSAIDs, the action of cyclo-oxygenase can be inhibited preoperatively. The inflammatory cascade is blocked because the eye is unable to convert arachidonic acid to prostaglandins. We showed that patients with three days of preoperative NSAIDs had a lower level of initial postoperative inflammation than did those cataract patients who were not pretreated with NSAIDs.<sup>47</sup> Less inflammation leads to a lower incidence and severity of CME.

For our patients, we prescribe topical NSAIDs four times per day starting three days prior to surgery. Patients also receive NSAIDs with their dilating drops on the day of surgery. Immediately following surgery, they take NSAIDs with steroids four times per day for the first week. The steroids are very important to this regimen. While the NSAIDs are blocking the production of prostaglandins, the steroids are suppressing any inflammation that may develop. The lesser the postoperative inflammation, the lower the incidence and severity of CME.

When the patients are examined at one week, assuming there is minimal inflammation, we stop the steroids but continue the NSAIDs, now twice daily for the remainder of the month. At one month, we assess the vision and measure the thickness of the macula. If everything is fine, we stop the NSAIDs. But if we feel that there may be some persistent macular edema, we continue the topical NSAIDs for an additional month.

### Endophthalmitis

The most common mechanism for the development of endophthalmitis is that bacteria from the lids, lashes, tears, or conjunctiva are inoculated into the anterior chamber either at the time of surgery or shortly after, before the incision is sealed. These bacteria seed the anterior chamber, where they are incubated and replicate. Thus, to most effectively prevent endophthalmitis, we need to sterilize the

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ocular surface as well as deliver bactericidal levels of antibiotics into the anterior chamber.

The goal for antimicrobial prophylaxis in cataract surgery is twofold:

1. To minimize the number of bacteria in the operative field
2. To maximize the concentration of potent antibiotic in the target tissues

Antibiotics and disinfectants such as povidone iodine are complementary in achieving this goal. Preoperative prophylaxis with fluoroquinolones will preoperatively inhibit the growth of bacteria and will sterilize the cornea and conjunctiva. A skin prep of 10% povidone iodine and a conjunctival prep of 5% povidone iodine will sterilize the surgical field at the time of surgery, minimizing the number of available bacteria that could be inoculated into the eye during surgery. After surgery, since bacteria may still be drawn into the wound for several days, antibiotics are continued to keep the ocular surface sterile and the concentration of intraocular antibiotics at a high level. Thus, the protocol of preoperative antibiotics, povidone iodine, and postoperative antibiotics has become the standard of care in phacoemulsification.

### A Randomized Study

How do we know the most efficacious regimen for administering preoperative antibiotics? That is, should we begin them at the surgery center on the day of surgery? Should we begin them the day before surgery, or several days before surgery?

We performed a study to determine the most effective regimen to secure the maximum amount of antibiotic in the anterior chamber. We investigated two commercially available antibiotics, and we divided our patients into three treatment groups. One group started taking their antibiotics on the day of surgery upon arrival at the surgery center. The second group began taking their antibiotics four times a day, one day prior to the surgery, and the third group started three days prior to surgery. Within each group, half the patients took gatifloxacin (Zymar, Allergan Pharmaceuticals) and the other half took ofloxacin (Ocuflox, Allergan Pharmaceuticals).

In this randomized mass study, we drew out a paracentesis right before surgery and sent the aqueous for high-performance liquid chromatography analysis to measure the concentration of antibiotic in the anterior chamber at the time of surgery. With 600 patients in the study, our data show the following:

1. The concentration in the anterior chamber was greatest in the patients who had taken the antibiotics for three days.
2. The penetration into the anterior chamber was greater for the gatifloxacin group than for the ofloxacin group.
3. With three days of preoperative antibiotics, we exceeded the MIC-90 levels for the common pathogens that cause endophthalmitis.

### Selecting an anti-infective

Several properties or characteristics should be considered when selecting an ocular anti-infective. A preferred topical ophthalmic antimicrobial agent would have a broad spectrum of antimicrobial activity, including efficacy against resistant organisms; would demonstrate rapid eradication rates; would demonstrate effective penetration into the ocular tissues and tear fluids; would not interfere with re-establishment of epithelial organization; and would be well-tolerated with minimal risk for corneal toxicity or any adverse events.

Our study supports the study of Henry Allen, who showed in 1974 that patients with two or three days of preoperative antibiotics had a lower incidence of endophthalmitis than those who did not receive preoperative prophylaxis.<sup>48</sup> While that study was done using erythromycin, today we cannot use erythromycin for prophylaxis because many of the bacteria that cause endophthalmitis have since become resistant to erythromycin. Similarly, aminoglycoside antibiotics such as gentamicin and tobramycin are not recommended for prophylaxis, because while they are highly potent against gram-negative bacteria, they have little coverage for the gram-positive bacteria, which constitute greater than 90% of all cases of endophthalmitis.

Among the commercially available ophthalmic antibiotics, only the ophthalmic fluoroquinolones offer adequate coverage against the gram-positive bacteria. Fluoroquinolones differ in their antibiotic coverage, their efficacy against resistant bacteria, their penetration into the anterior chamber, and their epithelial toxicity.

Toxicity is an important and often overlooked property. The faster the surgical wound heals, the sooner it becomes a barrier to the entry of the bacteria that may lead to endophthalmitis. If the antibiotic retards epithelial migration and stromal repair, the surgical wound will remain porous and patent to bacterial invasion.

In our practice, all patients begin gatifloxacin drops four times per day beginning three days prior to surgery, and they again receive it along with their dilating drops in the preoperative area. With three days of preoperative use, we sterilize the lids, lashes, and ocular surface preoperatively in such a way that drops started on the day of surgery alone cannot accomplish; and at the same time, we load the anterior segment of the eye with therapeutic levels of antibiotics.

In order to minimize the number of bugs bacteria at the operative site at the time of surgery, we use 10% povidone-iodine detergent solution for the skin prep. Per the advice of Robert Snyder, MD (personal communication), we never use 10% povidone-iodine in the conjunctiva, because the detergent base is caustic to conjunctival goblet cells. Instead, we use only the 5% ophthalmic solution when administering it into the conjunctiva.

## Conclusion

Remember that the use of antibiotics for preoperative prophylaxis is an off-label indication for these drugs. Although no fluoroquinolone or antibiotic is indicated for surgical prophylaxis, the literature supports the use of preoperative antibiotics for this indication. Make sure that the antibiotic you use is supported by the greatest amount of literature showing its efficacy.

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After completing this independent study activity, the participant should be able to:

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- Discuss postoperative complications of cataract surgery.
- Describe the prophylaxis of postoperative cystoid macular edema with non-steroidal anti-inflammatory drugs (NSAIDs).

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**General Evaluation:** Please use the scale below to evaluate this educational activity and objectives. Circle your response. As a result of completing this offering, I am able to:

	4 Very well	3 Moderately well	2 Fairly well	1 Not at all
1. Compare clinical studies evaluating anti-infective and anti-inflammatory regimens for patients undergoing cataract surgery.	4	3	2	1
2. Discuss postoperative complications of cataract surgery.	4	3	2	1
3. Describe the prophylaxis of postoperative cystoid macular edema with non-steroidal anti-inflammatory drugs (NSAIDs).	4	3	2	1
4. The content matches the objectives.	4	3	2	1
5. Independent study was an effective teaching method.	4	3	2	1
6. This course helped me achieve personal objectives.	4	3	2	1
7. The time required to complete this offering (in minutes) and take the test was:	60	75	90	>90

**CNE Test****Questions**

1. Cystoid macular edema is a result of the breakdown of the
  - a. blood-retinal barrier
  - b. choroidal vascular bed
  - c. retinal pigment epithelium
  - d. vitreo-retinal attachment
2. CME is common following intraocular surgery in patients with
  - a. glaucoma
  - b. multiple sclerosis
  - c. diabetic retinopathy
  - d. choroidal nevi
3. CME is often separated into which two entities?
  - a. clinical and postoperative
  - b. clinical and angiographic
  - c. angiographic and postoperative
  - d. preoperative and postoperative
4. Which of the following surgical procedures is *not* associated with the development of CME?
  - a. secondary IOL implantation
  - b. vitrectomy
  - c. scleral buckling
  - d. Yag laser capsulotomy
5. Which of the following preexisting ocular disorders is *not* associated with increased risk of CME?
  - a. aphakic bullous keratopathy
  - b. uveitis
  - c. branch retinal vein occlusion
  - d. narrow-angle glaucoma
6. Which antibiotic used in irrigating solution has been shown to increase the incidence of postoperative CME?
  - a. vancomycin
  - b. Ocuflox
  - c. Quixin
  - d. Zymar
7. In the eye, prostaglandins are primarily produced in the
  - a. iris and choroid
  - b. ciliary body and choroid
  - c. iris and ciliary body
  - d. iris, ciliary body, and choroid
8. In this paper's study, NSAIDs are given preoperatively
  - a. twice daily for two days preoperatively
  - b. three times daily, three days preoperatively
  - c. four times daily, three days preoperatively
  - d. four times daily, four days preoperatively
9. CME is the result of cystic accumulation of
  - a. intracellular fluid
  - b. extracellular fluid
  - c. subretinal fluid
  - d. subfoveal fluid
10. Medical conditions associated with increased risk of CME after cataract surgery include
  - a. diabetes and ischemic heart disease
  - b. diabetes and lupus
  - c. multiple sclerosis and ischemic heart disease
  - d. atherosclerosis and rheumatoid arthritis