
RETINA TODAY

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Clinical Cases: NSAIDs for Uveitic CME

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This continuing medical education activity is supported by an unrestricted educational grant from Alcon Laboratories.

TARGET AUDIENCE

This activity is designed for retina specialists and other ophthalmologists.

LEARNING OBJECTIVES

Upon successfully completing this learning program, participants should be able to:

- identify the currently available pharmaceutical agents used to treat CME
- discuss the mechanism of action of NSAIDs
- discuss the tolerability of steroids and NSAIDs
- discuss the data that support the treatment described in the cases presented.

FACULTY

Seenu M. Hariprasad, MD

METHOD OF INSTRUCTION

Participants should read the continuing medical education (CME) activity in its entirety. After reviewing the material, please complete the self-assessment test, which consists of a series of multiple-choice questions. To answer these questions online and receive real-time results, please visit <http://www.dulaneyfoundation.org> and click "Online Courses."

Upon completing the activity and achieving a passing score of over 70% on the self-assessment test, you may print out a CME credit letter awarding 1 *AMA PRA Category 1 Credit*.™ The estimated time to complete this activity is 1 hour.

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commensurate with the extent of their participation in the activity.

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CONTENT VALIDATION

In compliance with ACCME standards for commercial support and the Dulaney Foundation's policy and procedure for resolving conflicts of interest, this CME activity was peer reviewed for clinical content validity to ensure the activity's materials are fair, balanced and free of bias; the activity materials represent a standard of practice within the medical profession; and any studies cited in the materials upon which recommendations are based are scientifically objective and conform to research principles generally accepted by the scientific community.

FACULTY CREDENTIALS

Seenu M. Hariprasad, MD, is Associate Professor of Ophthalmology and Visual Science, Director of Clinical Research, and Chief of Vitreoretinal Service at the University of Chicago School of Medicine Department of Ophthalmology. He can be reached via e-mail at retina@uchicago.edu.

FACULTY/STAFF DISCLOSURE DECLARATIONS

Dr. Hariprasad states that he serves on the speakers bureaus of Genentech, Alcon, and Novartis and is a consultant to Pfizer.

All those involved in the planning, editing, and peer review of this educational activity have indicated that they have no financial relationships to disclose.

Clinical Cases: NSAIDs for Uveitic CME

STATEMENT OF NEED

Cystoid macular edema (CME) is a condition characterized by swelling of the retina due to leakage from the small blood vessels within the fovea, the central part of the retina responsible for detailed vision.¹ It is a general condition caused by a wide range of retinal diseases, which may include:

- diabetic retinopathy
- wet age-related macular degeneration or other causes of bleeding under the retina
- retinal vein occlusions
- epiretinal membranes (or macular pucker)
- uveitis
- other causes of inflammation within the eye such as

recent eye surgery (eg, cataract surgery)

Because many factors can lead to CME, effective treatment varies. Retinal inflammation is usually treated with anti-inflammatory medications.²⁻⁷ These are usually given as eye drops, although occasionally they must be administered as an injection or by mouth.

Topical NSAIDs have been shown to be effective in reducing postoperative cell and flare in cataract patients.⁸⁻¹⁴ A small but significant portion of patients, however, will not have complete control of postoperative inflammation with a topical NSAID alone. Therefore, using a combination of topical corticosteroids and topical NSAIDs is often most effective in ensuring excellent control of inflammatory responses.¹⁵⁻¹⁸

Many studies have suggested that topical NSAIDs are effective at preventing CME.^{19,20} In some studies, topical NSAIDs appear to be more efficacious than corticosteroids at preventing macular edema.²¹⁻²⁵

In light of increasing evidence for adequate, and sometimes improved, efficacy of NSAID monotherapy compared with corticosteroids, a postoperative regimen consisting solely of an NSAID may replace combination therapy as the primary regimen for CME prophylaxis.

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Three cases suggest efficacy for recalcitrant uveitic CME.

By Seenu M. Hariprasad, MD

Off-label use of topical NSAIDs is discussed in this article.

Cystoid macular edema (CME) is a condition that is often associated with decreased visual acuity and is a frequent cause of visual impairment for patients with uveitis.¹ Inflammation is often associated with CME and so it is often treated with corticosteroid therapy.² Steroids, although effective,³⁻⁵ can cause untoward effects, such as increased intraocular pressure (IOP) and resulting steroid-induced glaucoma and posterior subcapsular cataract.⁶ Thus, an alternative therapy would be advantageous to the patient and the clinician.

The mechanism of action of steroids against CME is via both mediation of inflammatory factors and stabilization of the blood-retinal barrier, thus reducing fluid buildup in the layers of the retina. Nonsteroidal anti-inflammatory drugs (NSAIDs) also have inflammatory mediation and blood-retinal-barrier stabilization capabilities and are not associated with increased IOP and cataract; thus, these agents may prove useful in the treatment of CME. Several studies show that NSAIDs may be effective in the treatment or prevention of CME.⁷⁻¹⁰

Nepafenac 0.1% (Nevanac, Alcon Laboratories, Inc.) is a prodrug that converts to amfenac in the eye. It is currently approved by the US Food and Drug Administration for pain and inflammation in cataract surgery procedures.

There are in vitro and animal data demonstrating nepafenac's efficacy for treating inflammation in the posterior segment of the eye.^{11,12}

In this article, we present three cases of uveitic CME that demonstrate the efficacy of topical nepafenac for treatment. Two of these cases have been previously reported in *Retinal Cases & Brief Reports*.¹³

CASE #1

Presentation. A 42-year old man presented with 20/80 visual acuity in his right eye and 20/100 visual acuity in the left eye. The patient had chronic bilateral CME that was secondary to pars planitis. He had received initial treatment in both eyes with prednisolone 1% and ketorolac 0.4% (Acular LS, Allergan, Inc.) for 6 to 8 months with good resolution of the inflammation, but no effect on the CME. The CME in the left eye was treated with 4 mg intravitreal triamcinolone acetonide

(Kenalog, Bristol-Myers Squibb). Three weeks post-treatment, the patient's left eye had an intraocular pressure (IOP) rise to 56 mm Hg. After treatment with three IOP-lowering agents, IOP was reduced to 22 mm Hg, and the visual acuity improved to 20/25 in that eye.

Treatment. Because of the complications that the patient experienced in his left eye due to treatment with intravitreal steroid, it was decided that we would treat the CME in the right eye with nepafenac three times daily for 3 months.

Result. Optical coherence tomography (OCT) at 3-month follow-up showed significantly reduced CME in the right eye as a result of treatment with nepafenac. Additionally, the patient's visual acuity improved to 20/25 in his right eye.

CASE #2

Presentation. A 26-year-old man presented with bilateral intermediate uveitis that had persisted for 3 years despite systemic treatment with 10 mg oral prednisone and 100 mg cyclosporine daily. The patient had a visual acuity of 20/25 in his right eye and 20/40 in his left eye. CME was visibly present in the right eye. Retinal thickness measured 358 μ m in the right eye and 243 μ m in the left eye.

Treatment. We treated the patient bilaterally with topical nepafenac twice daily for 6 weeks.

Result. After 6 weeks of treatment, the patient reported subjective improvement in visual acuity, and there was clinical evidence of improvement in CME, with retinal thickness measuring 220 μ m in the right eye and 224 μ m in the left eye.

CASE #3

Presentation. A 66-year old black woman with a history of bilateral nongranulomatous anterior uveitis presented with recalcitrant chronic uveitic CME with vision of 20/60 in both eyes and IOP of 23 mm Hg in her right eye and 24 mm Hg in her left eye due to mild steroid response. She had been treated previously with ketorolac 0.4% (Acular LS, Allergan), prednisolone acetate 1%, and loteprednol etabonate 0.5% (Lotemax, Bausch & Lomb) in both eyes for 6 months. Although the patient responded well in terms of decreased inflammation in both eyes, there was no improvement in her vision or the CME (Figure 1).

Treatment. This patient had been taking bri-

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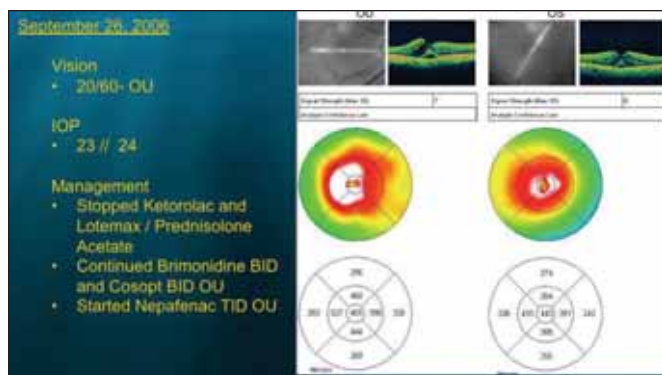


Figure 1. Case #3 presentation.

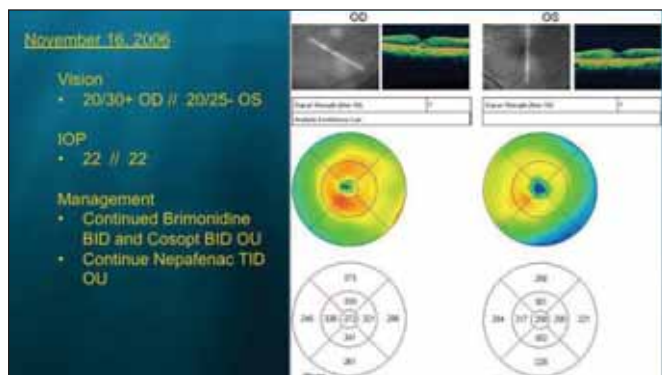


Figure 2. Case #3 after 7 weeks of treatment with nepafenac.

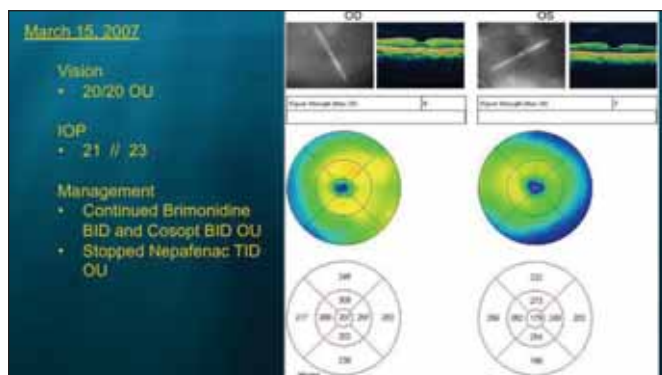


Figure 3. Case #3 after 3 more months of treatment with nepafenac.

monidone tartrate 0.1% (Alphagan P, Allergan) and dorzolamide hydrochloride and timolol maleate (Cosopt, Merck) twice daily for her IOP, and this treatment was continued. We stopped the ketorolac, prednisolone, and loteprednol and started her on nepafenac in both eyes three times daily.

Result. After approximately 7 weeks of treatment, the patient's vision improved to 20/30 in the right eye and 20/25 in the left eye. Her IOP was 22 mm Hg in both eyes (Figure 2). We continued treatment with the glaucoma drops and the NSAID for the next 3 months, and the patient's vision further improved

to 20/20 in both eyes, and IOP remained stable at 21 mm Hg in the right eye and 23 mm Hg in the left eye (Figure 3). The glaucoma therapy was continued, and NSAID treatment was discontinued.

DISCUSSION

Increased prostaglandin-mediated vascular permeability has been cited as a factor in the pathogenesis of CME. The mechanism of action of NSAIDs in CME is to prevent prostaglandin synthesis from arachidonic acid through the inhibition of cyclooxygenase (COX). Prophylaxis against CME has been shown with diclofenac 0.1% (Voltaren, Merck),^{14,15} ketorolac 0.5% (Acular, Allergan),¹⁶⁻¹⁸ flurbiprofen,^{19,20} and indomethacin.^{8-10,18-21}

PENETRATION AND EFFICACY OF NSAIDS

The penetration of NSAIDs is an important factor in their efficacy. All the available topical NSAIDs have the ability to penetrate the cornea and thus have some biologic effect against CME. To date, there are no head-to-head studies that have found that one NSAID provides a superior degree of protection against CME following cataract surgery. There are a number of differences, however, among the NSAIDs. One area of difference is their relative efficacy against COX 1 and COX 2.

NSAIDs are effective against CME via their ability to reduce the production of prostaglandins through cyclooxygenase inhibition. The two major forms of COX are COX 1 and COX 2. COX 1, which ketorolac has shown superior efficacy against,²² is an enzyme that promotes the production of prostaglandins and that is normally present throughout the body. COX 2, which nepafenac has shown superiority efficacy against,²² is similar to COX 1 in that it promotes the production of prostaglandins; however, COX 2 is present only at the site of injury or inflammation, making it a more effective target in the presence of a condition such as CME.²³

Nepafenac, a COX-2 inhibitor, is a prodrug, converted to amfenac by amidases in both the iris/ciliary body and the choroid/retina.^{24,25}

Walters and colleagues²² evaluated the aqueous humor concentrations of various NSAIDs following instillation of a single drop. They found the mean peak aqueous humor concentrations (ng/mL) to be 70.1 for amfenac, 205.3 for nepafenac, 57.5 for ketorolac, and 25.9 for bromfenac (Xibrom, Ista Pharmaceuticals).

This information is helpful in determining the level of

these agents available to suppress inflammation, considering that potency of a medication plays a role in its ability to control inflammation.

Nepafenac has been proven to be effective for reducing pain and inflammation in cataract surgery.²⁶ The cases presented in this article show that nepafenac can be of significant benefit to certain patients with uveitic CME. Each patient in this case series had marked improvement in retinal edema and subsequent visual acuity improvement after nepafenac treatment, suggesting that nepafenac may be effective for recalcitrant uveitic CME. Although there are no head-to-head published studies comparing NSAIDs for the treatment of CME, nepafenac has recently been shown to reduce the level of recalcitrant CME in patients who had previously been treated with ketorolac.^{13,27,28} Further study, however, is required to evaluate efficacy for posterior segment inflammation. ■

Seenu M. Hariprasad, MD, is Associate Professor of Ophthalmology and Visual Science, Director of Clinical Research and Chief of Vitreoretinal Service at the University of Chicago School of Medicine Department of Ophthalmology. Dr. Hariprasad is on the speakers bureaus of Genentech, Alcon, and Novartis and is a consultant to Pfizer. He can be reached via e-mail at retina@uchicago.edu.

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Name _____ MD participant non-MD participant

Phone (required) _____

I would like my certificate sent via fax _____

I would like my certificate sent via e-mail _____

CME QUESTIONS

1. Ocular corticosteroids are associated with what risks:

- a. increased IOP
- b. cataracts
- c. glaucoma
- d. all of the above

2. In vitro and animal data suggest that nepafenac is effective for posterior-segment inflammation.

- a. True
- b. False

3. A cited factor in the pathogenesis of CME is:

- a. systemic infection
- b. prostaglandin-mediated vascular permeability
- c. presence of subcapsular cataract
- d. none of the above

4. Nepafenac converts to amfenac by amidases in the:

- a. iris/ciliary body
- b. choroid
- c. retina
- d. all of the above

5. In vivo pharmacodynamic studies comparing nepafenac, ketorolac, and bromfenac show that nepafenac achieves an anterior chamber concentration ___ times higher than bromfenac.

- a. 8
- b. 10
- c. 5
- d. 3.6

6. In case #2, retinal thickness improved in the right eye with nepafenac treatment by:

- a. 58 μm
- b. 138 μm
- c. 19 μm
- d. 142 μm

7. The mechanism of action of NSAIDs in CME is to prevent prostaglandin synthesis from arachidonic acid through the inhibition of cyclooxygenase.

- a. True
- b. False

8. NSAIDs are approved for pain and inflammation by the US Food and Drug Administration for:

- a. retinal surgery
- b. refractive surgery
- c. cataract surgery
- d. none of the above

(Continued on page 7)

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ACTIVITY EVALUATION

Your responses to the questions below will help us evaluate this CME activity. This will provide us with evidence that improvements were made in patient care as a result of this activity as required by the Accreditation Council for Continuing Medical Education (ACCME). Please complete the following course evaluation and send back to the Dulaney Foundation via fax at +1 610-771-4443.

Name _____

Do you feel the program was educationally sound and commercially balanced? Yes No

Comments regarding commercial bias:

Rate your knowledge/skill level prior to attending this course: 5 = High, 1 = Low _____

Rate your knowledge/skill level after attending this course: 5 = High, 1 = Low _____

Would you recommend this activity to a colleague? Yes No

Do you feel the information presented will change your patient care? Yes No

If yes, please specify. We will contact you by E-mail in 2-3 months to see if you have made this change.

If no, please identify barriers to change.

List any additional topics you would like to see offered at future Dulaney Foundation programs or other suggestions or comments:

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