

Reply: We appreciate the interest and comments of Dr. Kamal. To respond to the first comment, we agree that measurement of foveal thickness after 4 weeks is too early to detect all cases of macular edema. In our study, the postoperative follow-up visits were scheduled in accordance with our standard postoperative evaluations; therefore, the measurements were taken 4 weeks after cataract extraction. We tried to account for this by reviewing patients' records after 6 months to determine the number of visits for post-cataract clinical macular edema after the last study visit. We agree with Dr. Kamal that we certainly could have missed some cases, but this applies to both groups. If we had measured 6 weeks after cataract extraction or at another fixed time point, we also could have missed some cases. Moreover, the percentage of patients with post-cataract macular edema depends on the definition used. Currently, there is no validated or universally accepted method for reporting post-cataract (clinical) macular edema, as discussed in our article.

To respond to the second comment, we should have been more specific by describing our exclusion criteria. We listed only the exclusion criteria mentioned in the study protocol; in addition to these, we excluded patients with systemic steroids and pseudoexfoliation. We also excluded patients with complicated cataract surgery, but this applied to only a few patients with posterior capsule rupture or iris manipulation. No patient with intraoperative hemorrhage was included.

As for the third comment, we may not have been clear that in the table we wanted to show that our flare values were not specifically better than but at least comparable to the values in other studies. We wanted to discuss the clinical relevance of the result that our postoperative flare values in the subconjunctival betamethasone acetate 5.7 mg/mL group were significantly higher than in the dexamethasone group.

Although treatment with subconjunctival betamethasone is relatively safe, it can produce unwanted side effects, as mentioned by Dr. Kamal. We agree that stopping the steroid is difficult when a subconjunctival steroid is used. In our study, no systemic or ocular side effects could be attributed to the use of a betamethasone injection and we did not see any patient with corneal necrosis or significant increase in intraocular pressure. However, we did not include patients with glaucoma or other ocular pathology. Our intention was to investigate the possibility of using a subconjunctival injection as an alternative treatment to steroid eyedrops in standard cataract cases, especially in patients with compliance problems.
—Myrthe Dieleman, MD, Peter de Waard, MD

Prevention of post cataract–surgery cystoid macular edema with nepafenac

The recent article by Miyake et al.¹ on post cataract–surgery cystoid macular edema left me with questions about study design and financial disclosures. The results are not surprising given that fluorometholone 0.1% is known to have poor corneal penetration² and would not be expected to have significant effects on the posterior segment. Why not compare nepafenac with a steroid with better penetration, one that is more commonly used as part of the post cataract–surgery regimen (eg, prednisolone acetate 1.0%), another nonsteroidal agent with proven penetration and efficacy in treatment or prevention of postoperative cystoid macular edema, or an inert control?

The cynic might conclude that there was a financial bias behind these study design decisions, as 2 of the authors are paid consultants to Alcon, nepafenac's manufacturer. The casual reader might never consider this possibility, as the abstract states that "[n]o author has a financial or proprietary interest in any material or method mentioned." This statement may be technically correct, and the abstract does indicate that additional disclosures can be found in the footnotes, where the authors' consultant status is acknowledged. However, many people do not read beyond the abstract and would not realize that Drs. Miyake and Numaga are consultants to the manufacturer of the drug being studied.

I am a fervent supporter of a strong partnership between industry, academia, and clinicians. Collaboration with industry toward development of new drugs, devices, and techniques, participation in clinical research, and physician education offers the best hope for continued innovation in medicine. However, this sort of article and the misleading disclosure statement only gives ammunition to those who want to place barriers between industry and physicians. If financial disclosures are listed in the abstract, they should err on the side of inclusion, leaving no room for misinterpretation.

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REFERENCES

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