LETTERS

Comment on: Anterior chamber cytokine production and postoperative macular edema in patients with diabetes undergoing FLACS

Our attention was drawn to the recent article by Cioana et al. that explores the effect of femtosecond laser–assisted cataract surgery and manual cataract surgery on cytokine expression in patients with and without diabetes.[1] The study identified several cytokines with potential proinflammatory properties, and although no notable increase in clinical inflammatory biomarkers such as postoperative cystoid macular edema was observed, we believe it is crucial to elucidate the sources of these cytokines to further minimize clinical biomarkers and ensure a less traumatic cataract surgery.

During cataract surgery, cytokines may originate from various sources, including lens epithelial cells (LECs), iris epithelial cells, ciliary body epithelial cells, and corneal endothelial cells. Previous reports have demonstrated that LECs produce a range of cytokines in vitro, including interleukin (IL)-1, IL-6, IL-8, basic-fibroblast growth factor, epidermal growth factor, transforming growth factor β, and prostaglandin E_2.[2–4] It is plausible that the additional cytokines detected by the authors also originate from LECs. We posit that LECs are likely the primary source of cytokine production, given that they are subjected to considerable surgical trauma during procedures such as continuous curvilinear capsulorhexis, cortical removal, and phacoemulsification by ultrasound. In essence, it is imperative to exercise caution during surgery to minimize unnecessary trauma to LECs.

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Disclosures: *The author has no financial or proprietary interest in any material or method mentioned.*

Reply: Anterior chamber cytokine production and postoperative macular edema in patients with diabetes undergoing FLACS.

We would like to thank Nishi et al. for his interest in our paper and his insightful comments.

We agree that it is important to elucidate the sources of cytokines released during cataract surgery and to try and minimize them to ensure safer surgical outcomes. As Nishi et al. point out, cytokines released during cataract surgery may originate from several ocular tissues, including but not limited to lens epithelial cells (LECs), iris epithelial cells, ciliary body epithelial cells, and corneal endothelial cells.[1,2] Another important source is the mechanical disruption of the blood–aqueous barrier during surgery, which is especially compromised in diabetes.[3]

Although we appreciate the importance of elucidating the source of cytokine production, ultimately it is the impact of these inflammatory cytokines (regardless of source) on downstream tissue effects that is of clinical significance (ie, driving cystoid macular edema, anterior chamber inflammation, and retinal microvascular changes). For that reason, our paper did not explore the sources of cytokines. Future studies are encouraged in this area, perhaps using laboratory-based models of cataract surgery to elucidate release of cytokines from different tissues.

It is interesting to ponder on the impact of cytokines released from LECs about our dataset. In our femtosecond-laser–assisted cataract surgery (FLACS) group, the cytokine draw happened after femtosecond laser-based capsulotomy, whereas in our manual cataract surgery (MCS) group, the cytokine draw happened only after corneal incision, without any capsulotomy being done. Interleukin (IL)-7, IL-13, and interferon-induced protein-10 (IP-10) were significantly higher in diabetic patients receiving FLACS than those diabetic patients undergoing MCS. Granulocyte colony stimulating factor, interferon-γ, IL-7, IL-8, IL-13, IP-10, monocyte chemoattractant protein (MCP)-1, and macrophage inflammatory protein-1β were significantly higher in the FLACS nondiabetic cohort than in the MCS nondiabetic cohort. As the difference between the surgical procedure of these 2 cohorts includes a capsulotomy, it is possible that LECs contributed to the production of these cytokines. This is an important question that deserves further exploration.

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Comment on: Feeling relaxed about your anesthesia routine?

As with all of Dr. Osher's publications, we enjoyed his Guest Editorial on "Feeling relaxed about your anesthesia routine?"[1]

The authors appreciated Dr. Osher's initial difficulties with anesthetic techniques in the 1970s. Although all surgeons wish the very best outcomes for the patient, both surgical outcomes and visual outcomes, the publication by Francis et al. on assisted local anesthesia (ALA) was a step forward, rendering cataract surgery a more relaxed, controlled, safe, and relatively rapid event.[2,3]

At the time, propofol, first manufactured in 1973, turned out to be very effective in achieving patient cooperation in the operating room. By contrast, although fentanyl could be administered judiciously by the anesthetist in small doses, aiming to settle the patient and minimize nociceptive events during cataract surgery, large doses of fentanyl, with its well-recognized side effects of nausea and vomiting, needed to be avoided.

Successful surgery depends on the surgical, anesthetic, and nursing teams working cooperatively in the patient's best interests. In cataract surgery, the aim is to optimize the surgical and visual outcomes, seeking to provide a technically superb operation (the surgical outcome), and aiming for patients to achieve corrected distance visual acuity (CDVA) of 20/12 in more than 92% of cases 1 month after cataract surgery (the visual outcome).[2]

When small incision cataract surgery through clear corneal incisions with assisted topical anesthesia (ATA) commenced, the one of the authors of the letter (Dr Francis) enthusiastically followed the trend. However, his anesthetists were overjoyed when the rigors of ATA no longer had to be faced, when their surgeon switched back to ALA. The anesthetists, therefore, had much better control of patient movement and co-operation during the procedure, especially if it turned out to be prolonged.

The senior author of the letter, Dr Francis attempted to determine preoperatively whether the patient could be operated under ATA or whether ALA was required. Accordingly, the Lanindar (light and nociceptive interaction noting distress and response) test was developed, utilized, and published by Figueira et al.[4]

The senior author of the letter, Dr Francis experienced and reported 3 cases of incomplete expulsive choroidal hemorrhage in the early days of extracapsular cataract extraction with ALA, all patients fortunately achieving CDVA 20/20 or better at 1 month.[5] The authors suspect that Dr. Osher would agree that having an expulsive choroidal hemorrhage is a defining experience for the surgeon, demanding rapid and definitive intervention, aiming to avoid losing the contents of the eye onto the drapes, let alone the floor of the operating room. These cases could only have been dealt with effectively by high-quality intervention by the anesthetist. Cataract surgeons should be grateful for the efforts of their anesthetist in managing this relatively terrifying medical situation.

Intrinsically, core anesthesia practice involves assessing preoperative patients thoroughly, and applying both physiological and pharmacological knowledge to enable best care for them during surgery, as documented by the Australian and New Zealand College of Anaesthetists.

Dr. Osher's contribution to anesthesia in cataract surgery has not only been massive but also extremely positive. The authors are, and those who have followed in Dr. Osher's footsteps should be, very grateful for his contribution.

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