Severe Bilateral Ischemic Retinal Vasculitis Following Cataract Surgery

Laura B. Nicholson, BA; Brian T. Kim, MD; Javier Jardón, MD; William Townsend-Pico, MD; Carmen Santos, MD; Andrew A. Moshfeghi, MD; Thomas A. Albini, MD; Dean Elliott, MD; Lucia Sobrin, MD, MPH

Abstract: This report describes two cases of severe, bilateral ischemic retinal vasculitis following cataract surgeries at different surgical centers. In both cases, the patient underwent bilateral cataract surgeries, performed 1 week apart for each eye. In the perioperative period following the second of the two surgeries, both patients developed severe, bilateral intraocular inflammation and profound vision loss. The underlying cause of this adverse response remains unknown. The authors suggest that the severe inflammatory reaction could be related to an intraoperative intracameral vancomycin injection.


Introduction

Inflammatory syndromes occurring after cataract surgery, such as toxic anterior segment syndrome (TASS) and endophthalmitis, are usually limited to the operated eye. The purpose of this report is to describe two cases of severe bilateral inflammation and posterior segment vasculitis that occurred after the second eye underwent cataract surgery within 1 week of the first eye. To our knowledge, this clinical entity has not been previously described. We present these cases in order to increase awareness of this type of postsurgical reaction with the hope of identifying similar cases and common underlying risk factors.

Case 1

A 67-year-old woman underwent cataract surgery in the right eye with injections of lidocaine with epinephrine (1%; 1:100,000) and a vancomycin injection (1.0 mg/0.1 cc) into the anterior chamber at the end of the procedure. The postoperative drops administered were ofloxacin, prednisolone acetate 1%, and ketorolac tromethamine 4%. One week later, the patient’s visual acuity was 20/25, and clinical examination findings were unremarkable. The following day, cataract surgery was performed in the left eye with the same technique and postoperative regimen. On the first postoperative day, the patient noticed painless bilateral visual loss without any systemic symptoms. Her visual acuity was hand motion in the right eye and light perception in the left eye. Pupils were fixed and nonreactive, and IOP was 38 and 16 mm Hg in the right and left eyes, respectively. Slit lamp examination revealed bilateral corneal stromal edema, keratic precipitates in the left eye, and a severe anterior-chamber fibrinous reaction in both eyes, but no hypopyon (Figure 1). The vitreous was clear, and fundus examination revealed diffuse intraretinal hemorrhages (Figure 2). No other cases of postsurgical inflammation were reported from the surgical center from these 2 days.

Prednisolone drops and oral prednisone were initiated without any change after 1 day. Vitreous taps were performed, and intravitreal injections of vancomycin 1 mg, ceftazidime 2.25 mg, and ganciclovir 2 mg were administered. Serologic testing, including blood cultures, did not identify an underlying cause (Table, page 340). Pars plana vitrectomy was performed in the right eye, and intraoperative exam-
ination revealed severe panretinal ischemia and diffuse intraretinal hemorrhages. No retinitis was seen. Fungal and bacterial cultures from both the vitreous tap and vitrectomy specimen showed no growth. An anterior chamber tap yielded negative results for herpes viruses by polymerase chain reaction (PCR) testing (Table). She completed a course of systemic antiviral and antibiotic therapy without improvement. At a 7-month follow-up examination, the patient had no light perception in both eyes. There was bilateral corneal opacification and hypotony but no intraocular inflammation.

CASE 2

A 71-year-old man underwent cataract surgery in each eye performed 3 days apart. During both procedures, the eye was irrigated with a balanced salt solution with epinephrine (1:1000 dilution). Preservative-free lidocaine (0.5 mL of 1% solution) and epinephrine (0.5 mL of 1:100,000 dilution) were injected intracamerally, and an intracameral vancomycin injection was administered at the end of both procedures. Postoperative drops given were moxifloxacin, cyclopentolate and prednisolone 1%. He had 20/25 visual acuity in both eyes and unremarkable clinical examination findings 1 day after the second procedure. One week later, visual acuity was 20/25 in the right eye and 20/20 in the left eye, with 1+ anterior chamber cell in the left eye. Fundus examination revealed intraretinal hemorrhages in both eyes and white-centered hemorrhages in the left eye. The following day he developed bilateral, painless visual loss without systemic symptoms. Twelve days after the second surgery, his visual acuity had declined to light perception in both eyes, with 3+ anterior chamber cell, 2+ vitritis, and hemorrhagic ischemic vasculitis (Figures 3A-B, page 341) in both eyes. Optical coherence tomography (OCT) demonstrated significant retinal thickening. Fluorescein angiography showed global retinal nonperfusion and diffuse vascular leakage consistent with occlusive vasculitis (Figure 3C, page 341). There were no other reports of postsurgical inflammation at the surgical center.

The patient was treated empirically for possible acute retinal necrosis with intravitreal ganciclovir injections, oral valacyclovir, and oral and topical prednisolone. Vitreous fungal and bacterial cultures, along with aqueous viral PCR and serologic testing, did not reveal an underlying cause (Table, page 340). On further questioning, the patient revealed a remote history of acute renal failure requiring dialysis.
<table>
<thead>
<tr>
<th>Case</th>
<th>Serologic Testing</th>
<th>Fluid Sample Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Seropositivity for herpes simplex 1 IgM and IgG elevated C reactive protein. Negative anti-nuclear antibody, anti-neutrophil cytoplasmic antibodies, and syphilis serologies. Complete blood count, sedimentation rate, and comprehensive metabolic panel were all normal. No growth on blood cultures.</td>
<td>Bacterial and fungal cultures and polymerase chain reaction for herpes viruses were negative on samples from the vitreous tap and vitrectomy. Aqueous fluid sample was negative by polymerase chain reaction for herpes simplex viruses, varicella zoster virus, and cytomegalovirus.</td>
</tr>
<tr>
<td>2</td>
<td>Negative anti-nuclear antibody, anti-neutrophil cytoplasmic antibodies, rheumatoid factor, human immunodeficiency virus, rapid plasma reagin, and toxoplasmosis antibodies. Complete blood count, comprehensive metabolic panel, sedimentation rate, angiotensin-converting enzyme, and C reactive protein levels were all within normal limits. All serologies for hypercoagulability were normal. No growth on blood cultures.</td>
<td>Vitreous bacterial and fungal cultures were negative. Aqueous fluid sample was negative by polymerase chain reaction for herpes simplex viruses 1 and 2, varicella zoster virus, Epstein-Barr virus, and cytomegalovirus.</td>
</tr>
</tbody>
</table>

DISCUSSION

We report two cases in which severe, bilateral ischemic retinal vasculitis developed within the first week after the second of two uncomplicated cataract surgeries performed within 1 week’s time. The cases share a number of similarities with regard to presentation timing, type of inflammatory response, and intracameral vancomycin use during cataract surgery. The two patients underwent surgeries on their right and left eyes separately, and their sudden profound visual loss occurred bilaterally in the perioperative period (from 1 to 7 days) after the second procedure. Both patients exhibited inflammation in both the anterior and posterior chambers, although the first patient experienced more severe anterior segment inflammation. Fundus findings from both patients demonstrated hemorrhagic, ischemic retinal vasculitis. Vancomycin was injected intracamerally during the procedures on both patients. Neither surgical center reported other cases of postoperative inflammation on the days these patients underwent surgery. The differences in management between these two cases resulted from the different treatment patterns at the respective centers where the patients were seen.

The etiology of disease in these patients is not certain, and no similar cases have been presented in the literature thus far. Results from a PubMed search with the terms “post-cataract surgery,” “posterior inflammation,” “retinal vasculitis,” and “retinitis” did not yield any entities resembling those presented in this report. Infectious etiologies are unlikely given the negative intraocular fluid results and progression despite antimicrobial therapy. These cases are also unlikely to be due to a direct toxic reaction to an exposure at the time of surgery. With a toxic exposure, one would expect to see the...
effects immediately after surgery only in the most recently operated eye. In these cases, however, onset of disease began simultaneously in both eyes, at least 24 hours after the second surgery and after documentation of normal postoperative examination findings in the first operated eye (as well as the second operated eye for case 2). An atypical variant of TASS is also unlikely because there were no other reported cases of inflammation following surgery at these centers on the days these patients underwent cataract extraction.\textsuperscript{1} Simultaneous bilateral ischemic retinal disease such as central retinal vein occlusion is also unlikely given the presence of anterior segment inflammation and no evidence of a systemic disease associated with bilateral central retinal vein occlusion.

Several features of these cases could support an autoimmune reaction. The first patient had a history of autoimmune disease (vitiligo), and it is has been documented that patients with a history of one autoimmune disease have an increased chance of developing additional autoimmune diseases.\textsuperscript{3} The fact that the second patient’s inflammation has been controlled with immunosuppressive treatment also supports an autoimmune etiology. Both patients received intracameral vancomycin for endophthalmitis prophylaxis at the end of surgery. The second patient’s previous adverse response to vancomycin raises the possibility that the inflammatory response was the result of a delayed immune reaction to the medication. It is possible that exposure to the medication with the first surgery sensitized the patients to vancomycin, and then the exposure with the second surgery triggered an immune response. In the second case, in which the patient had been exposed in the past to vancomycin, it is possible that the patient’s reaction in the first operated eye was initially subclinical. We do not believe this to be a direct toxic effect of the vancomycin. Vancomycin retinal toxicity has been seen in rabbits when injected intravitreally with ceftazidime in silicone oil–filled eyes.\textsuperscript{4} However,
no cases of vancomycin-associated retinal toxicity or occlusive retinal vasculitis in humans have been reported thus far, despite the widespread use of intravitreal vancomycin (1 mg/0.1 mL) for endophthalmitis. Although we cannot definitively rule out the possibility of lidocaine with epinephrine as the cause of this response, these medications are more widely used, and neither patient had a history of allergic response to either of them.

While we are not certain of the autoimmune etiology in these two cases, we believe that it is important for other ophthalmologists to be aware of these cases. If other physicians have similar cases, joint examination of the cases could help to identify the underlying risk factors and etiology of this clinical entity that is rapidly progressive and visually devastating.

REFERENCES