remission and avoiding harm of ongoing, smouldering inflammation, which risks permanent visual loss.

Stuart Carter, Vipul Ramjiani, Lisa Dunkley, Sachin Salvi
Royal Hallamshire Hospital, Sheffield, United Kingdom

Correspondence to:
Stuart Carter, MBChB; s.j.carter@sheffield.ac.uk

References

1. Steenbrugge F, Raaijmaakers M, Caekebeke P, et al. Pyoderma gangrenosum following trauma of the knee: a case of pathergy and review of orthopaedic cases. *Injury*. 2011;42:421-3.
2. O'Donoghue E, Lightman S, Tuft S, et al. Surgically induced necrotising sclerokeratitis (SINS)–precipitating factors and response to treatment. *Br J Ophthalmol*. 1992;76:17-21.
3. Stone JH. Wegener's Granulomatosis Etanercept Trial Research Group. Limited versus severe Wegener's granulomatosis: baseline data on patients in the Wegener's granulomatosis etanercept trial. *Arthritis Rheum*. 2003;48:2299-309.
4. Holle JU, Voight C, Both M, et al. Orbital masses in granulomatosis with polyangiitis are associated with a refractory course and a high burden of local damage. *Rheumatology*. 2013;52:875-82.
5. Haynes BF, Fishman ML, Fauci AS, et al. The ocular manifestations of Wegener's granulomatosis. Fifteen years experience and review of the literature. *Am J Med*. 1977;63:131-41.
6. Provenzale JM, Mukherji S, Allen NB, Castillo M, Weber AW. Orbital involvement by Wegener's granulomatosis: imaging findings. *Am J Roentgenol*. 1996;166:929-34.
7. Taylor SR, Salama AD, Joshi L, et al. Rituximab is effective in the treatment of refractory ophthalmic Wegener's granulomatosis. *Arthritis Rheum*. 2009;60:1540-7.
8. Suhler EB, Lim LL, Beardsley RM, et al. Rituximab therapy for refractory orbital inflammation: results of a phase 1/2, dose-ranging, randomized clinical trial. *JAMA Ophthalmol*. 2014;132:572-8.
9. Stone JH, Merkel PA, Spiera R, et al. Rituximab versus cyclophosphamide for ANCA-associated vasculitis. *N Engl J Med*. 2010;363:221-32.

Can J Ophthalmol 2016;51:e101–e103

http://dx.doi.org/10.1016/j.jcjo.2016.02.011

Elevation of intraocular pressure after inadvertent dexamethasone implant injection into the lens

Intravitreal corticosteroids have shown beneficial effects in patients with macular edema secondary to diabetes and retinal vein occlusion and in patients with uveitis.[1] The corticosteroids are delivered through a single intravitreal injection or by implantation of a sustained-release device.[1] Biodegradable, sustained-release dexamethasone implants are inserted transconjunctivally into the vitreous cavity using preloaded 22-gauge needle delivery systems. As these implants release dexamethasone over several months, their use is increasing for indications such as macular edema followed by retinal vein occlusion, non-infectious inflammation of the uvea, and diabetic macular edema.[1–4]

Exogenous steroids, however, can cause complications, including the secondary elevation of intraocular pressure (IOP), but these risks are dependent on the route of administration, duration of treatment, type of steroids, and history of glaucoma.[1] Another complication of exogenous steroids is the progression of cataract, which generally begins to appear during the second year after starting intravitreal steroid therapy.[2,4,5] These complications have been observed in patients receiving dexamethasone intravitreal implants, although their rates were lower than those in patients treated with triamcinolone acetonide injections and fluocinolone acetonide implants.[1,2]

Additional complications related to intravitreal corticosteroid implants include migration of the implant toward the anterior chamber, usually in patients with an absent or defective lens capsule.[6] The accidental injection of a dexamethasone implant into the crystalline lens is a rare, unexpected complication also related to the procedure.[7–11] Several cases have been reported, with most showing the cataract progression and requiring cataract surgery.[7,9,12] This report describes a patient who presented with elevated IOP but without cataract progression who was inadvertently injected in the lens with a dexamethasone implant.

A 53-year-old Korean woman was referred to the Department of Ophthalmology of Ajou University Hospital (Suwon, Korea) for blurred vision in her left eye. Ten months earlier, her left eye had been intravitreally injected at another clinic with a dexamethasone implant (Ozurdex; Allergan Inc, Irvine, Calif.) to treat macular edema secondary to branch retinal vein occlusion. The patient was followed up for 2 months, but was lost to follow-up for 8 months. After 10 months, she re-presented at the other clinic with an IOP in her left eye of 30 mm Hg. She was prescribed brimonidine, latanoprost, and a fixed combination of brinzolamide and timolol for treatment of elevated IOP. She was subsequently referred to our clinic.

At initial examination, her best-corrected visual acuity was 20/200 OS and her IOP was 19 mm Hg. A conspicuous foreign body was present in the crystalline lens, but the visual axis in her left eye was not compromised (Fig. 1A). Despite the presence of an implant in the lens, there were no indications of cataract progression. Fundus examination showed sclerotic retinal vessels at the superotemporal branch, suggesting prior branch retinal vein occlusion (Fig. 2A).

As her IOP fluctuated from 19 to 40 mm Hg despite treatment with glaucoma medications, cataract surgery

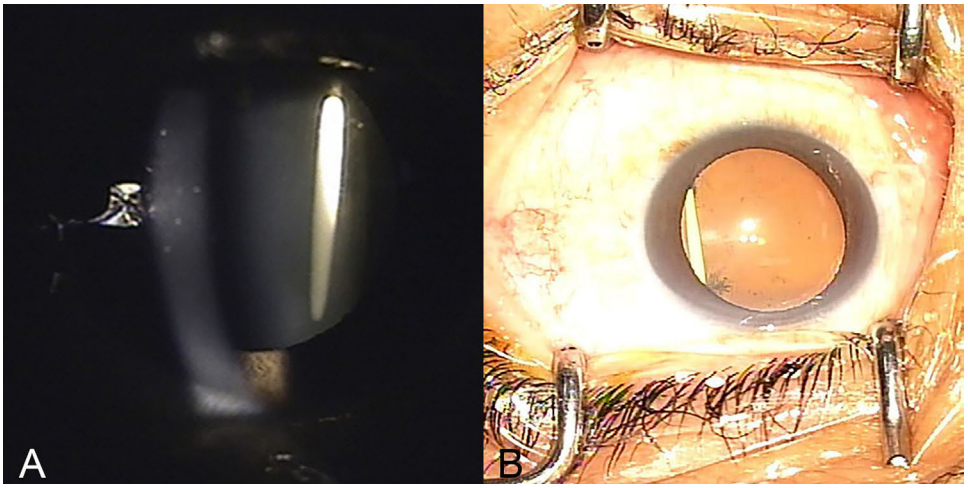

Fig. 1—Dexamethasone implant located in the crystalline lens without cataract progression at 10 months after inadvertent injection. (A) Anterior segment photograph at initial presentation. (B) Intraoperative photograph. Lens extraction, including removal of the implant, was performed by standard phacoemulsification.

was performed with standard phacoemulsification devices. The implant was absorbed with the lens nucleus during emulsification (Fig. 1B), and focal fibrosis was observed in the posterior capsule without a capsular tear. A hydrophobic acrylic 1-piece intraocular lens was inserted into the capsular bag without complications. After cataract surgery, she was prescribed a simplified glaucoma regimen, consisting of a fixed combination of brinzolamide and timolol. One week after surgery, the IOP in her left eye was 12 mm Hg and the visual acuity in that eye was 20/100 with persistent macular edema (Fig. 2B). The patient was scheduled to undergo intravitreal injection of an antivascular endothelial growth factor agent.

If the dexamethasone implant is located properly in the vitreous, subsequent IOP elevation is generally transient, peaking at approximately 6 weeks and returning to the normal range within 6 months.[13] The occurrence of cataract progression is also infrequent, as reported in the Geneva Study.[2] Indeed, that study did not report any complications related to accidental injection into the lens. In contrast, several case reports have described outcomes in patients who received inadvertent dexamethasone implants into the crystalline lens.[7–11] Three patients received inadvertent implants before cataract surgery during planned combined operations, necessitating immediate cataract.[8] Two patients underwent cataract surgery within 1 week after injection, preventing the development of cataract progression or IOP elevation.[11]

One patient presented with increased IOP 2 months after a second intracapsular dexamethasone implant injection performed for persistent macular edema.[7] Although IOP was well controlled by β-blocker monotherapy, cataract progression was observed after 10 months, leading to standard phacoemulsification and in-the-bag intraocular lens implantation after 11 months. Two additional

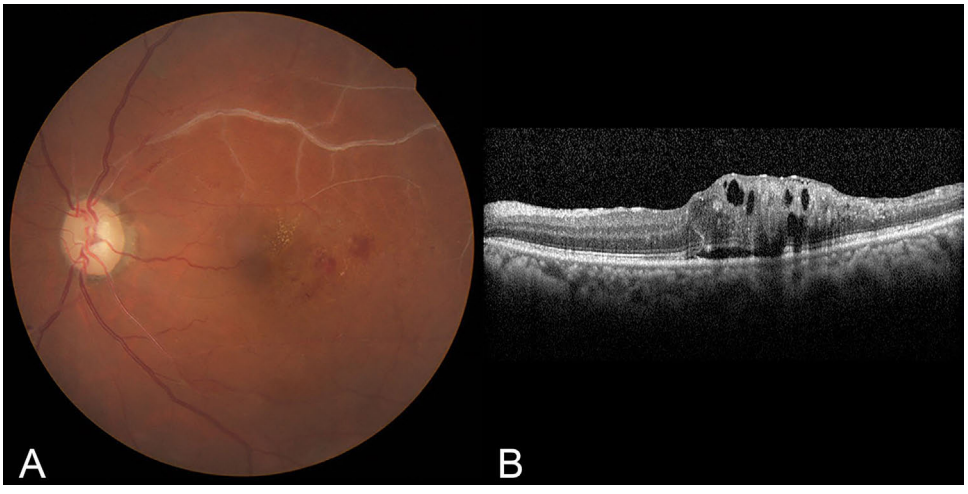

Fig. 2—Fundus findings of the left eye 10 months after inadvertent injection of the dexamethasone implant into the lens. (A) Fundus photograph showing branch retinal vein occlusion. (B) Optical coherence tomography showing persistent macular edema.

patients who experienced IOP elevation and cataract progression within 3–6 months also required cataract surgery because of decreased visual acuity secondary to cataract progression, although IOP was stabilized in both by glaucoma treatment.[10] Another patient experienced cataract progression within 10 weeks after implantation, but without IOP elevation.[9]

The findings in our patient differ from those in previous reports, in that our patient experienced delayed IOP elevation without cataract progression. As our patient did not report visual discomfort related to the intracapsular location of the dexamethasone implant, she did not present for follow-up examinations for several months. Therefore, the time at which IOP became elevated was unclear. Macular edema persisted after injection of the dexamethasone implant, as reported previously.[10] As the components of the lens (65% water and 35% protein) and vitreous (99% water) differ, the sustained release of dexamethasone through hydrolysis may have been inhibited, resulting in no change in the size of the implant in the lens and persistent macular edema.[10] This suggests that, if a dexamethasone implant is inadvertently injected into the lens, leaving the implant in place has no beneficial effects on macular edema but has risks of complications such as IOP elevation, thus indicating the need for prompt removal.

In summary, the inadvertent injection of a dexamethasone implant into the crystalline lens may lead to delayed elevation of IOP without cataract progression. Because of the possibility of delayed elevation of IOP, cataract surgery, including extraction of the implant, should be considered even if there are no immediate complications.

Disclosure: The authors have no proprietary or commercial interest in any materials discussed in this article.

Kihwang Lee, Aram Park, Seran Jang, Yoo-Ri Chung
Ajou University School of Medicine, Suwon, Korea.

Correspondence to:
Yoo-Ri Chung, MD; cyr216@hanmail.net

References

1. Kiddee W, Trope GE, Sheng L, et al. Intraocular pressure monitoring post intravitreal steroids: a systematic review. *Surv Ophthalmol*. 2013;58:291-310.
2. Haller JA, Bandello F, Belfort R Jr., et al. Dexamethasone intravitreal implant in patients with macular edema related to branch or central retinal vein occlusion twelve-month study results. *Ophthalmology*. 2011;118:2453-60.
3. Lowder C, Belfort R Jr., Lightman S, et al. Dexamethasone intravitreal implant for noninfectious intermediate or posterior uveitis. *Arch Ophthalmol*. 2011;129:545-53.
4. Gillies MC, Lim LL, Campain A, et al. A randomized clinical trial of intravitreal bevacizumab versus intravitreal dexamethasone for diabetic macular edema: the BEVORDEX study. *Ophthalmology*. 2014;121:2473-81.
5. Gillies MC, Sutter FK, Simpson JM, Larsson J, Ali H, Zhu M. Intravitreal triamcinolone for refractory diabetic macular edema: two-year results of a double-masked, placebo-controlled, randomized clinical trial. *Ophthalmology*. 2006;113:1533-8.
6. Khurana RN, Appa SN, McCannel CA, et al. Dexamethasone implant anterior chamber migration: risk factors, complications, and management strategies. *Ophthalmology*. 2014;121:67-71.
7. Koller S, Neuhann T, Neuhann I. Conspicuous crystalline lens foreign body after intravitreal injection. *Ophthalmologe*. 2012;109:1119-21.
8. Ram J, Agarwal AK, Gupta A, Gupta A. Phacoemulsification and intraocular lens implantation after inadvertent intracapsular injection of intravitreal dexamethasone implant. *BMJ Case Rep*. 2012;2012:bcr2012007494.
9. Berarducci A, Sian IS, Ling R. Inadvertent dexamethasone implant injection into the lens body management. *Eur J Ophthalmol*. 2014;24:620-2.
10. Coca-Robinot J, Casco-Silva B, Armada-Maresca F, Garcia-Martinez J. Accidental injections of dexamethasone intravitreal implant (Ozurdex) into the crystalline lens. *Eur J Ophthalmol*. 2014;24:633-6.
11. Fasce F, Battaglia Parodi M, Knutsson KA, et al. Accidental injection of dexamethasone intravitreal implant in the crystalline lens. *Acta Ophthalmol*. 2014;92:e330-1.
12. Kocak N, Ozturk T, Karahan E, Kaynak S. Anterior migration of dexamethasone implant in a pseudophakic patient with intact posterior capsule. *Indian J Ophthalmol*. 2014;62:1086-8.
13. Dugel PU, Bandello F, Loewenstein A. Dexamethasone intravitreal implant in the treatment of diabetic macular edema. *Clin Ophthalmol*. 2015;9:1321-35.

Can J Ophthalmol 2016;51:e103–e105
0008-4182/16/$-see front matter
http://dx.doi.org/10.1016/j.jcjo.2015.12.022

Malignant transformation of retinocytoma treated with intra-arterial chemptherapy

Clara Knieper was most likely the first to make the closest description of what is currently known as retinocytoma, in a child named Peter Neuburger, born on May 12, 1899, and who underwent bilateral enucleations 11 years apart.[1] Since then, the clinical features of this rare tumour have become well established and include a translucent greyish retinal mass sometimes associated with a staphyloma, calcific foci, retinal pigment epithelial alterations, cystic areas, and chorioretinal atrophy without exudation and prominent feeder vessels.[2,3] Retinocytoma is traditionally considered a benign tumour and therefore requiring no treatment. However, malignant transformation was documented in several individual case reports, and a recent study on 36 patients with retinocytoma showed that 12% of these tumours progressed into retinoblastoma.[3–5] Little or no response to systemic chemotherapy is another characteristic of retinocytoma.[6]