swelling, numbness, and linear whitish wheals.[3] It is generally accepted that these adverse events are temporary and do not affect the cosmetic outcome or induce significant sequelae. However, no reports have included adverse effects to the cornea, although the procedure is performed near the eyes.

It was thought that the corneal opacity in this case was caused by inaccurate targeting with IFUS and IFUS treatment that was applied close to the delicate eyelid skin. IFUS can create a focal thermal injury to the SMAS. Eyelid skin is the thinnest skin on the human body and has little connective tissue.[4] The average thickness of eyelid skin is 521 ± 115.8 μm in the upper eyelid of Korean individuals and varies from 0.013 to 0.014 μm in white individuals. Therefore, exact targeting of IFUS is difficult, and IFUS can injure adjacent tissue. The eyeball and eyelids are very sensitive organs, so a small injury can cause distinct discomfort. Therefore, a protective device for the eyeball, including the cornea or conjunctivae, is necessary.

We hypothesized that wound contraction results from 2 mechanisms: first, collagen shrinkage due to coagulation of collagen; and second, inflammatory shrinkage occurring due to myofibroblasts. First, collagen in the corneal stroma is mostly type I, with smaller amounts of types III, V, and VI. These collagen molecules are distinguished from other extracellular matrix components by their triple-helical conformation with alpha and beta cross-links. The thermal effect of IFUS shrinks the intermolecular alpha cross-links of collagen in the cornea, and collagen shrinkage in the cornea due to IFUS results in a density change of the cornea. That effect could induce unexpected scattering or refraction of light and create corneal astigmatism. The corneal wound healing process begins after thermal damage has occurred. Second, an inflammatory response occurs due to myofibroblasts. Corneal haze is associated with activation, migration, and differentiation of stromal keratocytes to corneal myofibroblasts. Myofibroblasts have alpha smooth muscle actin, which contracts wounds, resulting in corneal haze and astigmatism.

The astigmatism-induced corneal opacity improved after topical steroid application in our case, as topical steroid agents inhibit collagen synthesis, enhance collagen remodeling, and reduce corneal wound contracture.[5] Therefore, topical steroid eye drops may relieve wound contracture due to collagen shrinkage because of collagen remodeling. Second, steroids are anti-inflammatory agents that inhibit the immune response, collagen synthesis, and neovascularization. Topical steroidal agents such as fluorometholone delay the inflammatory process and the appearance of myofibroblasts.[5] We prescribed topical steroid for 1 month to increase collagen remodeling and reduce the inflammatory response caused by the IFUS thermal effect. Finally, we observed a decrease in astigmatism, which was caused by corneal opacity after IFUS: 0.75 D in the right eye and 0.55 D in the left eye.

Our case report highlights the possibility of corneal opacity developing after IFUS. Dermatologists and cosmetic surgeons should pay attention to this side effect. We recommend applying a blocking device to protect the ocular surface organs during the IFUS procedure.

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Vision loss and vascular compromise with facial and periocular injections

Facial and periorbital regions are rich in vascular supply given the extensive internal-external carotid branching and anastomoses.[1] As a result, any foreign material introduced (either accidentally or iatrogenically) around the eye and facial region has the potential of introducing emboli into the ophthalmic circulation, which could lead to devastating consequences. Severe and often permanent vision loss after injections in the facial areas is a rare, but known complication that has been reported within nasolabial folds, glabella, intranasal, periocular, and forehead

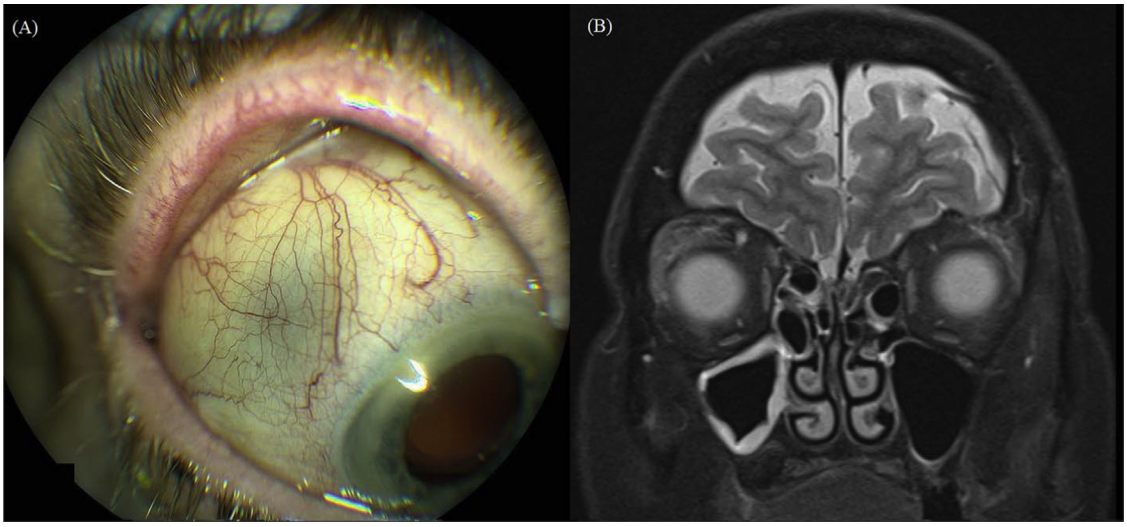

Fig. 1—Patient with complete vision loss after subtenon Kenalog injection over the superotemporal quadrant. (A) Unremarkable anterior segment examination other than mild tenderness and prominence over the lacrimal gland. (B) Magnetic resonance orbits demonstrating enlargement of the right lacrimal gland and lateral rectus muscle.

regions.[2–8] We review the current literature on the topic of visual and vascular compromise caused by facial and periorbital injections, as well as present a case of transient vision loss to no light perception (NLP) after subtenon triamcinolone acetonide (Kenalog; Bristol-Myers Squibb) injection because of ophthalmic artery occlusion (OAO) with full visual recovery.

A 77-year-old female with persistent bilateral cystoid macular edema (CME) after cataract surgery refractory to topical corticosteroid and nonsteroidal anti-inflammatory drug was referred for subtenon Kenalog injection. The patient received 2 uneventful subtenon Kenalog injections bilaterally because of the persistent CME.

On her third visit for subtenon Kenalog injection, 15 seconds after injection of the right eye over the superotemporal quadrant, the patient suffered vision loss from 20/80 to NLP with associated nausea and right orbital pain. Immediate anterior segment examination was unremarkable other than localized tenderness and fullness over the lacrimal gland (Fig. 1A). Posterior segment examination demonstrated deep white lesions within the choroidal vasculature, as well as white crystal-like lesions within small terminal retinal arterioles (Fig. 2). There were no signs of central retinal artery occlusion (CRAO), but retinal perfusion was occludable with moderate digital pressure. Intravenous Fluorescein Angiography (IVFA) demonstrated small peripheral ischemic areas, but there was no filling defect. Urgent magnetic resonance scan of the orbits performed 2 hours after injection showed enlargement of the right medial rectus muscle and the lacrimal gland, without other orbital abnormalities (Fig. 1B).

Repeat examination after the magnetic resonance imaging, approximately 4 hours later, showed the patient had

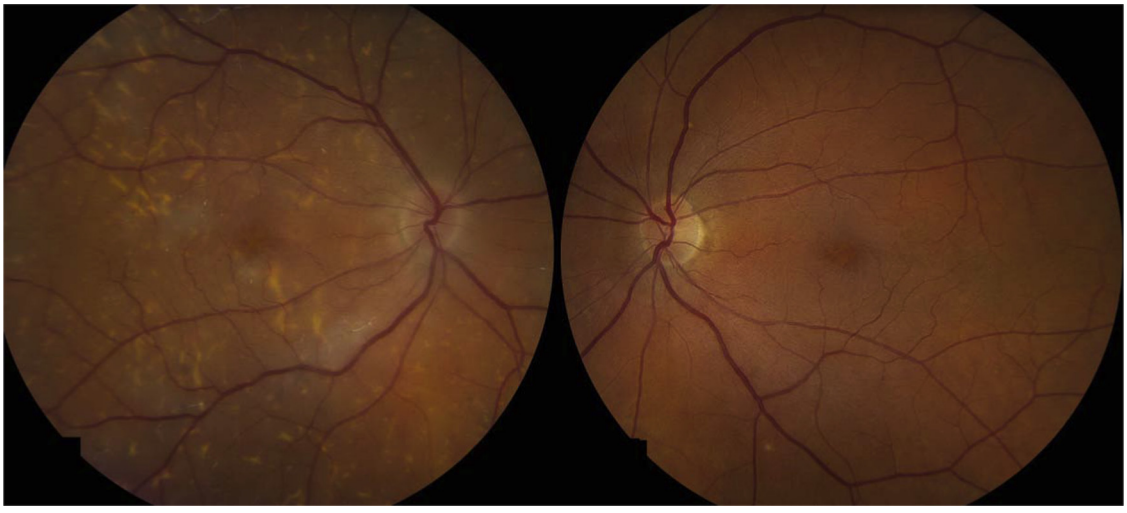

Fig. 2—Fundus photograph after subtenon Kenalog injection with complete vision loss. Visible white crystal-like lesion of likely Kenalog suspension in the choroidal and retinal vasculature of right eye.

experienced a steady vision improvement after injection back to the 20/80 level. At 1-week follow-up, vision improved to 20/50 with dramatic resolution of her CME on optical coherence tomography. Fundus examination demonstrated complete resolution of the white crystal-like lesions within the retinal arteries and choroidal vasculature. At 1-month follow-up, patient's best corrected visual acuity improved to 20/40 without any residual signs of CME.

Posterior subtenon injection of corticosteroid was first described by Nozik in 1972 for the treatment of uveitis.[9] Since then, subtenon Kenalog injection has been used extensively among ophthalmologists as an effective method for controlling uveitis refractory to topical medications. Although subtenon injection of Kenalog has been generally considered safe, numerous complications have been reported, such as soft tissue swelling, cataract formation, increased intraocular pressure, pseudoptosis, accidental injection into the subretinal space and globe penetration, retrobulbar hemorrhage, and retinal and choroidal vascular occlusion.[10–12] Among these, vascular occlusion is considered the most serious and has been associated with permanent and irreversible visual loss.

In our case, we propose that the vision loss was due to vascular compromise from Kenalog being accidentally injected into the lacrimal artery and travelling retrogradely into the ophthalmic artery, resulting in transient OAO. The enlargement seen in the lateral rectus muscle and lacrimal gland was likely due to local accumulation of Kenalog and vascular congestion of the lacrimal artery. Luckily, there was no permanent vision loss as the OAO from Kenalog suspension was transient. The Kenalog preparation is made of a suspension free of large particles/emboli that could result in permanent or prolonged occlusion. After the injection pressure dissipates, the Kenalog suspension could disperse and flow into distal smaller vessels under the arterial pressure, thereby relieving and recanalizing the transient vascular obstruction but travelling to more distal branches of the ophthalmic artery, resulting in clinically visible deposits within the choroid via the short posterior ciliary arteries and the retina through the central retinal artery. As the blood flow is re-established within the ophthalmic artery and central retina artery, the vision recovered spontaneously and the CME resolved quickly with the high concentration of intravascular Kenalog.

To our knowledge, this is the first case in the literature of transient OAO with full and spontaneous visual recovery. Also, it is important to note that the manufacturer has specifically recommended against the use of intraturbinal, periocular, and intraocular Kenalog injections because of the lack of safety studies and risk for blindness.[13] Indeed, vision loss caused by vascular compromise after intranasal, forehead, and eyelid Kenalog injection has been well documented in the literature.[14–18] Therefore, it is critical for physicians to document and have thorough discussions about all the potential risks associated with facial and periocular injections as part of the informed consent process.

In addition to periocular Kenalog injections, facial filler injections for cosmetic wrinkle removal and facial augmentation have also been reported to have severe visual-threatening complications secondary to vascular occlusion. It is important for ophthalmologists to be aware of the potential visual complications of facial filler injections because of their rising popularity for cosmetic purposes. Currently, facial filler injection is the second most commonly performed minor procedure by plastic surgeons and dermatologists after botulinum toxin injections.[19,20]

Vision loss presents as an immediate complication of facial filler injection with other associated signs and symptoms depending on the location of vascular occlusion.[7,8] OAO, CRAO, and branch retinal artery occlusion (BRAO) after filler injections have all been reported. OAO and CRAO have been associated with the worst visual prognosis. Given the important vascular supply from branches of ophthalmic artery, OAO is commonly associated with pain, pupillary abnormalities, ophthalmoplegia, ptosis, decreased choroidal thickness, and absence of cherry red spot.[8]

The mechanism of vascular compromise from facial filler injections is thought to be due to retrograde injection of filler materials through distal arterioles under high injection pressure.[7,8] Once the injection pressure has been removed, the filler materials travel distally and result in vascular occlusion depending on the filler particle size and the diameter of affected arteries. Many filler materials are available, differing by particle sizes and duration of effect. Fillers made of autologous fat, hyaluronic acid, collagen, and polymethyl methacrylate have been reported with visual complications. Of these, autologous fat and hyaluronic acid have been reported to account for the vast majority of filler-related vision loss reported in the literature, with autologous fat being responsible for approximately 50% of all cases. Autologous fat has also been established as the most likely filler material to cause OAO. The high risk associated with autologous fat and hyaluronic acid is likely due to their large particle size with hyaluronic acid particles measuring 400 μm in diameter and autologous fat composed of even larger and variable-sized fat globules.[21] Given the 2-mm diameter of the ophthalmic artery and 160-μm diameter of the central retinal artery, it is understandable why autologous fat results in more OAO, whereas hyaluronic acid filler is more likely to result in CRAO.

The site of facial filler injections is also a factor in the risk for visual complications with filler injections. Among all published reports, injections over glabella, nasolabial folds, and forehead regions are associated with the greatest risk. The risk for vascular compromise at each site is likely determined by the underlying vascular anastomoses and proximity to the ophthalmic artery. For instance, retrograde travel of emboli from the glabellar region likely comes from

the supratrochlear or supraorbital artery, whereas dorsal nasal, angular, and/or lateral nasal arteries likely account for emboli travel of injections over the nasolabial folds.

Contrasted with the complete visual recovery from transient OAO seen with Kenalog injection in the case presented, facial filler injection–related visual complications have poor prognosis, with approximately 90% of all patients who have suffered OAO or CRAO from facial filler injection ending up with vision of NLP.[8] Visual recovery is also extremely limited in BRAOs and reported in only 1 case of CRAO. Many management options with the goal of dislodging the emboli and re-establishing the vascular flow have been mentioned in the literature, such as intraocular pressure–lowering medications, anterior chamber paracentesis, ocular massage, medical thrombolysis, hyperbaric oxygen, hemodilution, among others.[7,20] To date, no management has proved effective, and no consensus exists on the proper management of injection–related vision loss.

Given the frequent use of periocular injections and the increasing popularity of facial filler injections, it is crucial to raise physician awareness about the anatomical concerns that may lead to this potentially blinding complication. There is also a need to discuss the potential risk for vision loss with patients who are considering periorbital or facial injections, or both, especially for injection over higher-risk regions such as glabella and nasolabial fold and with higher-risk materials such as autologous fat and hyaluronic acid.

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An atypical case of bilateral posterior scleritis in a patient with chronic lymphocytic leukemia

Chronic lymphocytic leukemia (CLL) is the most common leukemia in the Western world, with 75% of affected individuals diagnosed between 55 and 84 years of age.[1] The prevalence rate of ocular involvement in CLL has been estimated to be approximately 12%, and presentations remain variable and inconsistent.[2] To our knowledge, bilateral posterior scleritis in a patient with CLL has not been reported in the literature.

A 70-year-old male was admitted to Grand River Hospital in Kitchener, Ontario, for severe fatigue and malaise. He had a 9-year history of CLL, for which he was treated with 3 cycles of oral chlorambucil in the past. Approximately 11 days after conclusion of the second

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