

Glaucoma Associated With Chemical Burns

Often overlooked after an injury, this serious complication is the most important limiting factor of these patients' visual rehabilitation.

BY TERRI-DIANN PICKERING, MD

Chemically burned eyes often develop multiple ocular diseases, including opaque corneas, ocular surface derangements, cataract, and uveitis.¹ The most important preventable complication is glaucoma, however, which occurs in up to 75% of eyes with severe chemical burns (Figure 1).² Patients are typically male industrial workers, but chemical injuries also occur in the domestic and/or assault setting.^{3,4} Glaucoma is a serious complication due to its high prevalence and severity, the difficulty of diagnosis, and its general refractoriness to treatment.⁴

PATHOPHYSIOLOGY

The severity of damage from a chemical burn varies with the type and concentration of the causative agent and with exposure time.⁵ Acids denature, precipitate, and coagulate corneal proteins. By acting as a barrier, protein coagulation helps to prevent the chemicals' penetration into deeper structures. Thus, acid burns tend to be less severe than other chemical burns. The single exception is hydrofluoric acid (found in antirust solutions), which may rapidly penetrate the cornea and anterior chamber.³

Alkalis are lipophilic, and they penetrate ocular tissues more deeply and more rapidly than acids. Alkalis quickly enter ocular surface cells, producing a saponification reaction. During the inflammatory response, damaged cells secrete proteolytic enzymes, causing further surrounding damage.^{3,5} The pH correlates with the depth of penetration, which continues long after the initial exposure.⁵



(Images courtesy of Nathan Radcliffe, MD.)

Figure 1. The eye of a patient with a severe chemical burn and glaucoma that ultimately required the placement of a keratoprosthesis and a glaucoma drainage device.

Strong alkalis can reach the anterior chamber and damage the lens, iris, trabecular meshwork (TM), and ciliary body in as little as 5 to 15 minutes. These chemicals can even produce retinal scarring.

Overall, the pattern of IOP alterations is complex.⁶ A rapid, acute rise may result from collagen shrinkage and contraction and from increased uveal blood flow.^{3,7} This increase may be followed by a return to normal IOP or hypotony (due to ciliary body damage) and then a sustained elevation of IOP.⁸ Long-term glaucoma may be caused by multiple mechanisms, including pupillary block, an accumulation of inflammatory debris in the TM, and direct damage to the TM.^{3,9} Chronic and



Figure 2. A chemical injury to the ocular surface resulted in inferior limbal stem cell deficiency, conjunctivalization of the inferior cornea, and secondary open-angle glaucoma, presumably from damage to the TM or inflammation from the chemical injury.

subacute inflammation also contribute. Not only can the primary insult cause glaucoma, but necessary treatments can cause or worsen the disease.⁴ The standard of care for a chemical injury demands the long-term use of topical steroids. Multiple surgeries can also necessitate months of topical and/or oral steroids and possibly a peribulbar and/or intravitreal steroid. Steroid treatment can cause glaucoma. In addition, multiple surgeries may damage the TM or cause progressive angle closure. Finally, trauma and scarring from limbal stem cell surgery may impair episcleral venous outflow (Figure 2).⁴

MANAGEMENT

After chemical burns, physicians' attention focuses on the cornea, and glaucoma is a secondary consideration. IOP spikes may be missed, and glaucoma is frequently overlooked entirely. This disease, however, is the single most important factor limiting patients' visual outcomes.^{10,11}

Unfortunately, measuring the IOP of chemically burned eyes is notoriously difficult, because diseased corneas render indentation tonometry and finger palpation inaccurate.² Cade et al reviewed data on 28 eyes with severe chemical burns that underwent the implantation of a Boston Keratoprosthesis (Massachusetts Eye and Ear Infirmary).² Twenty-one eyes (75%) had severe glaucoma preoperatively, which progressed in nine of the eyes postoperatively. Additionally, two eyes developed glaucoma postoperatively. The IOP could only be measured by palpation due to the rigidity of the prosthesis' back plate, further limiting glaucoma management.

The treatment of elevated IOP must be a priority in the early phase of managing a chemical burn through the initial

tion of generously administered glaucoma drops. Oral aqueous suppression is an option to avoid additional toxicity to the corneal surface, but glaucoma must be treated aggressively.³ If the disease progresses on maximal medical therapy, tube shunts and/or cyclophotocoagulation are necessary.² Trabeculectomy is fraught with potential complications. There may not be enough healthy conjunctiva available for the surgery. Additionally, the use of an antimetabolite may result in a further loss of limbal stem cells and increase the risk of a bleb leak from chronic dry eye disease.⁴ Chronic, subacute inflammation may also severely shorten the lifespan of the bleb.

CONCLUSION

Physicians continue to underestimate the danger of IOP spikes and the potential for long-term glaucomatous damage after an ocular chemical burn.² Classification systems devised to indicate the prognosis of ocular chemical burns ignore IOP and glaucoma altogether.^{10,11} Data have suggested a 22% incidence of secondary glaucoma in patients with severe chemical burns, but disturbingly, 75% of the patients in a study by Cade et al had advanced glaucoma prior to vision rehabilitation surgery.^{2,10} Despite the heroic efforts of doctors and patients alike, visual rehabilitation surgery is futile if glaucoma goes unrecognized or undertreated. Occupational and public health efforts must continue to encourage people to protect their eyes when working with hazardous chemicals and to immediately irrigate their eyes after an injury. ■

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