

Ocular Trauma — Chemical Injuries

Ramesh Venkatesh*, Hemkala L Trivedi**

Introduction

Background : Chemical injuries to the eye represent one of the true ophthalmic emergencies. While almost any chemical can cause ocular irritation, serious damage generally results from either strongly basic (alkaline) compounds or acidic compounds. Alkali injuries are more common and can be more deleterious. Bilateral chemical exposure is especially devastating, often resulting in complete visual disability. Immediate, prolonged irrigation, followed by aggressive early management and close long-term monitoring, is essential to promote ocular surface healing and to provide the best opportunity for visual rehabilitation.

Pathophysiology : The severity of this injury is related to type, volume, concentration, duration of exposure, and degree of penetration of the chemical. The mechanism of injury differs slightly between acids and alkali.

Acid injury : Acids dissociate into hydrogen ions and anions in the cornea. The hydrogen molecule damages the ocular surface by altering the pH, while the anion causes protein denaturation, precipitation, and coagulation. Protein coagulation generally prevents deeper penetration of acids and is responsible for the ground glass appearance of the corneal stroma following acid injury. Hydrofluoric acid is an exception; it behaves like an alkaline substance because

the fluoride ion has better penetrance through the stroma than most acids, leading to more extensive anterior segment disruption.

Alkali injury : Alkaline substances dissociate into a hydroxyl ion and a cation in the ocular surface. The hydroxyl ion saponifies cell membrane fatty acids, while the cation interacts with stromal collagen and glycosaminoglycans. This interaction facilitates deeper penetration into and through the cornea and into the anterior segment. Subsequent hydration of glycosaminoglycans results in stromal haze. Collagen hydration causes fibril distortion and shortening, leading to trabecular meshwork alterations that can result in increased intraocular pressure (IOP). Additionally, the inflammatory mediators released during this process stimulate the release of prostaglandins, which can further increase IOP.

Frequency : Chemical injuries represent roughly 10% of patients seeking treatment for eye complaints at US hospitals.

Mortality/Morbidity

- More than 60% of injuries occur in workplace accidents, 30% occur at home, and 10% are the result of an assault.
- As many as 20% of chemical injuries result in significant visual and cosmetic disability; only 15% of patients with severe chemical injuries achieve functional visual rehabilitation.

Race

No overall racial predilection exists;

*Resident; **Assoc. Prof., Department of Ophthalmology, BYL Nair Hospital, Mumbai Central, Mumbai 400 008.

however, young black males are more likely to have high-concentration, high-impact alkaline chemical injuries secondary to assault.

Sex

Males are 3 times more likely to experience chemical injuries than females.

Age

Chemical injuries can strike any population; however, most injuries occur in patients aged 16-45 years.

Clinical

History : Most often, the patient gives a history of a liquid or a gas being splashed or sprayed into the eyes or of particles falling into the eyes. Regardless of the specific mechanism of injury, the patient's complaints are frequently related to the severity of the exposure. Common specific complaints elicited are as follows:

- Pain (often extreme)
- Foreign body sensation
- Blurred vision
- Excessive tearing
- Photophobia
- Red eye(s)

Physical

A thorough physical examination should be deferred until the affected eye is irrigated copiously, and the pH of the ocular surface is neutralized. After irrigation, a thorough eye examination is performed with special attention given to clarity and integrity of the cornea, degree of limbal ischaemia, and IOP. The eye examination can be facilitated by using topical anaesthetic drops to aid in patient comfort and cooperation. Common physical manifestations of chemical injuries to the eye include the following:

- Corneal epithelial defect: Corneal

epithelial damage can range from mild diffuse punctate epithelial keratitis (PEK) to a complete epithelial defect. A complete epithelial defect may not take up fluorescein dye as rapidly as in a routine corneal abrasion; therefore, it may be missed. If an epithelial defect is suspected but not found on the initial evaluation, the eye should be reexamined after several minutes.

- Stromal haze: Haze can range from a clear cornea to a complete opacification with no view into the anterior chamber.
- Corneal perforation : A very rare finding at presentation, it is more likely to occur after the initial presentation (from days to weeks) in severely injured eyes that have poor healing capacity.
- Anterior chamber inflammatory reaction: This condition can present as varying degrees of inflammation (flare and cell) in the anterior chamber. This finding is more common with alkaline chemicals and is related to the greater depth of penetration.
- Increased IOP: This finding is related to both the degree of anterior segment inflammation and the degree of corneal collagen deformation and shortening, which essentially shrinks the anterior chamber, thus raising IOP and reducing uveoscleral outflow.
- Adnexal damage/scarring: Similar to chemical injuries on other skin areas, this finding can lead to severe exposure problems if eyelid scarring prevents proper closure, thereby exposing an already damaged ocular surface.
- Conjunctival inflammation: Varying degrees of conjunctival hyperaemia and chemosis are possible, and even a mild chemical injury can elicit an exuberant

conjunctival response.

- Perilimbal ischaemia: The degree of limbal ischaemia (blanching) is perhaps the most significant prognostic indicator for future corneal healing because the limbal stem cells are responsible for repopulating the corneal epithelium. The greater the extent of blanching, the worse the prognosis.
- Particles in the conjunctival fornices: This finding is more common with particulate injuries, such as plaster. If not removed, the residual particles can serve as a reservoir for continued chemical release and injury. These particles must be removed before ocular surface healing can begin.
- Decreased visual acuity: Initial visual acuity can be decreased because of epithelial defects, haze, increased lacrimation, or discomfort. In moderate-to-severe chemical burns seen soon after the injury, the corneal haze may be minimal on presentation with good vision, but it can increase significantly with time, severely reducing vision.

Causes

- Alkali
 - Ammonia (most serious)
 - Lye
 - Potassium hydroxide
 - Magnesium hydroxide
 - Lime (most common)
- Common sources of alkali are as follows:
 - Fertilizers
 - Cleaning products (e.g., ammonia)
 - Drain cleaners (e.g., lye)
 - Oven cleaners
 - Potash (e.g., potassium hydroxide)
 - Fireworks (e.g., magnesium

hydroxide)

- Cement (e.g., lime)
- Acids
 - Sulphuric acid
 - Sulphurous acid (most common)
 - Hydrofluoric acid (most serious)
 - Acetic acid
 - Chromic acid
 - Hydrochloric acid
- Common sources of acids are as follows:
 - Battery acid (sulphuric)
 - Glass polish (hydrofluoric)
 - Vinegar (acetic)

Work - Up

Lab Studies

- Periodically test the pH of the ocular surface, and continue irrigation until the pH reaches neutrality. No other laboratory tests are generally necessary unless other systemic injuries are concurrent.

Treatment

Medical Care

- Remove inciting chemical (irrigation)
 - Immediate copious irrigation remains the single most important therapy for treating chemical injuries. Effective irrigation dilutes and flushes away the inciting chemical.
 - The injured eye is irrigated with a sterile balanced buffered solution, such as Ringer's lactate solution or normal saline solution. Immediately beginning irrigation, without waiting for the ideal fluid, is important; therefore, plain tap water is usually the solution of first choice for initial irrigation. Irrigation should be

- continued until the pH of the ocular surface is neutralized, usually using 1-2 litres of fluid.
- The irrigation solution must contact the ocular surface; therefore, the eye must remain open by means of a lid speculum, or, more ideally, the irrigation should be conducted with the use of special irrigating tubing (e.g., Morgan lens). If available, the eye should be anaesthetized prior to irrigation.
 - Control inflammation
 - Inflammatory mediators released from the ocular surface at the time of injury cause tissue necrosis and attract further inflammatory reactants, which can inhibit reepithelialization. Breaking this inflammatory cycle can enhance the rate of epithelial regrowth.
 - Topical steroids, which have no direct effect on the rate of epithelial regrowth, are useful during the early recovery phase. Cycloplegics also reduce inflammation by stabilizing the blood-aqueous barrier.
 - Prevent infection: The corneal epithelium serves as a barrier to infection. When this layer is absent, the eye is susceptible to infection. For prevention, the use of prophylactic topical antibiotics is warranted during the initial treatment stages.
 - Reduce IOP: The use of aqueous suppressants is advocated to reduce IOP secondary to chemical injuries, both as an initial therapy and during the later recovery phase, if IOP is high (> 30 mm Hg).
 - Promote epithelial healing
 - Once the inciting chemical has been completely removed, epithelial healing can begin. Chemically injured eyes have a tendency to poorly produce adequate tears; therefore, artificial tear supplements play an important role in healing.
 - Ascorbate plays a fundamental role in collagen remodeling, leading to an improvement in corneal healing.
 - Placement of a therapeutic contact lens until the epithelium has regenerated can be helpful in some patients.
 - Control pain: Severe chemical burns can be extremely painful. Ciliary spasm can be managed with the use of cycloplegic agents; however, oral pain medication may be necessary initially to control pain.

Surgical Care

- Sweep fornices to remove retained particles. This technique is especially important when particulate matter (e.g., plaster) is responsible for the injury. A moist sterile cotton swab can be used to sweep the superior and inferior fornices after instilling topical anaesthesia.
- Debride necrotic conjunctival/corneal tissue. Removal of severely necrotic material from the ocular surface is necessary because this tissue can stimulate a more intense inflammatory reaction, which inhibits ocular surface regeneration.
- Lyse conjunctival adhesions. Adhesions are a later finding, and they can be managed with repeated lysis using a glass rod or a sterile cotton swab.
- Cyanoacrylate tissue adhesive may be applied for the treatment of corneal melting.
- In the acute phase, temporary amniotic membrane patching may be considered.

- Long-term surgical options include penetrating keratoplasty, limbal stem cell transplant, amniotic membrane transplant, cultivated corneal epithelial stem cell sheet transplant, or any combination of the above.

Medication

Medical therapy following irrigation in chemical injuries is geared toward promoting epithelial healing, preventing infection, preventing damage from increased IOP, and controlling pain.

Epithelial healing is promoted through aggressive lubrication, ascorbate replenishment, and judicious use of topical corticosteroids. Artificial tears and ointments are especially important with severely scarred and exposed eyes. Ascorbate, both oral and topical, aids in the synthesis of collagen fibrils. Topical steroids decrease ocular surface inflammation, facilitating new epithelial cell growth and ocular surface regeneration. The presence of epithelial defects and corneal exposure necessitates the use of prophylactic topical antibiotics to prevent infection in the already compromised eye.

Antibiotic ointments can serve the dual purpose of providing lubrication and preventing infection. Broad-spectrum antibiotic coverage is required to most effectively minimize the infection.

Moderate and severe injuries often stimulate an increase in IOP due to anterior chamber inflammation and collagen fibril shortening. This condition is treated most effectively with aqueous suppressants, especially oral carbonic anhydrase inhibitors and topical beta-adrenergic blockers.

Inflamed eyes often experience ciliary spasm, which can be painful. This spasm is blocked by relatively long-acting mydriatic cycloplegics. In severe chemical injuries, oral

pain medication may be required to comfort the patient.

Drug Category

Topical antibiotics

Prevent ocular surface infection and effectively lubricate the eye.

Carbonic anhydrase inhibitors

Carbonic anhydrase inhibitors reduce aqueous humor production, which then reduces IOP.

Cycloplegic mydriatics

Cycloplegic mydriatics reduce pain by blocking ciliary spasm, and they reduce intraocular inflammation by stabilizing the blood-aqueous barrier. Drugs from this category are chosen based on their duration of action. Intermediate-acting compounds, such as homatropine or scopolamine, are preferred to short-acting compounds, such as tropicamide, or extremely long-acting compounds, such as atropine sulphate.

Ascorbate

Critical cofactor necessary for collagen fibril synthesis. Released from the damaged cornea and the anterior chamber, and it must be replenished to promote corneal wound healing.

Beta-adrenergic blockers

Topical beta-blockers reduce aqueous humor production, which then reduces IOP.

Topical corticosteroids

Steroids decrease ocular surface inflammatory response, facilitating earlier epithelial healing and regeneration. These medications must be tapered after 7-10 days because of the risk of corneal melting with prolonged use.

Further Inpatient Care

- In patients with severe chemical injuries,

short hospitalization may be warranted to closely monitor IOP, integrity of the cornea, medication usage, and pain control.

Further Outpatient Care

- Close follow-up care is mandatory in the first weeks following a severe chemical injury to assess epithelial regeneration and corneal melting, to change medications, to control inflammation and IOP, and to prevent secondary infection. Patients should be under the care of an ophthalmologist during this critical period.

In/Out Patient Medications

- Prednisolone acetate 1% (1 gtt qid)
- Erythromycin ophthalmic ointment (4-8 times/d)
- Homatropine 5% or scopolamine 0.25% (1 gtt tid)
- Ascorbate (500 mg PO qid)
- Levobunolol hydrochloride 0.5% (1 gtt bid) or acetazolamide (500 mg PO bid) -

Pressure lowering agents, such as levobunolol and acetazolamide, are only indicated if IOP is increased (> 30 mm Hg).

Complications

- Primary complications
 - Conjunctival inflammation
 - Corneal abrasions
 - Corneal haze and oedema
 - Acute rise in IOP
 - Corneal melting and perforations
- Secondary complications
 - Secondary glaucoma
 - Secondary cataract
 - Conjunctival scarring
 - Corneal thinning and perforation
 - Complete ocular surface disruption with corneal scarring and vascularization
 - Corneal ulceration (sterile or infectious)
 - Complete globe atrophy (phthisis)

Roper Hall's Classification

Grades	Cornea	Conjunctiva	Prognosis
Grade I	Epi. Cell loss only	No ischaemia	Full recovery
Grade II	Visible iris	Ischaemia < 1/3 limbus	Some scarring
Grade III	Iris details obscured	Ischaemia 1/3 – ½ limbus	Impaired vision
Grade IV	No view of iris/ pupil	Ischaemia > ½ limbus	Perforation expected

DUA's Classification

Grade	Prognosis	Limbal Involvement	Conj. Involvement
I	Very Good	0 clock hrs.	0%
II	Good	= 3 clock hrs.	30%
III	Good	> 3-6 clock hrs.	> 30-50%
IV	Good-guarded	> 6-9 clock hrs.	> 50-75%
V	Guarded – poor	> 9 < 12 clock hrs.	>75-100%
VI	Very poor	Total limbus	Total conj.

bulbi)

Prognosis

- With chemical injuries, the severity correlates with the prognosis, especially with regard to the corneal integrity (corneal epithelial defects and stromal clarity) and the degree of limbal ischaemia (blanching). Limbal stem cells provide the epithelial cells necessary for adequate corneal wound healing; therefore, the evaluation of limbal damage is especially important in determining the prognosis.
- Grades 0-2 can be expected to heal well with proper care and follow-up examinations. The course for grades 3-5 is more tenuous and may require surgical intervention, either limbal stem cell transplantation or penetrating keratoplasty, to regenerate the corneal surface. Higher-grade injuries are more susceptible to secondary complications.

Medical/Legal Pitfalls

- Failure to evaluate patients with extensive facial chemical injuries for other nonocular potentially life-threatening injuries
- Failure to thoroughly remove the inciting chemical from the ocular surface through extensive irrigation and removal of particulate matter from the fornices, which can worsen the injury
- Failure to closely observe the patient during the immediate recovery phase, which can lead to more severe secondary complications

- Failure to document whether or not the patient was properly using safety glasses at the time of injury, especially if the injury occurred at work

References

1. Chiou AG, Florakis GJ, Kazim M. Management of conjunctival cicatrizing diseases and severe ocular surface dysfunction. *Surv Ophthalmol* 1998; 43(1) :19-46.
2. Dua HS, King AJ, Joseph A. A new classification of ocular surface burns. *Br J Ophthalmol* 2001; 85 (11) : 1379-83.
3. Inatomi T, Nakamura T, Koizumi N. Midterm results on ocular surface reconstruction using cultivated autologous oral mucosal epithelial transplantation. *Am J Ophthalmol* 2006; 141 (2) : 267-75.
4. Kobayashi A, Shirao Y, Yoshita T. Temporary amniotic membrane patching for acute chemical burns. *Eye* 2003; 17 (2) : 149-58.
5. Ozdemir O, Tekeli O, Ornek K. Limbal autograft and allograft transplantations in patients with corneal burns. *Eye* 2004; 18 (3) : 241-8.
6. Pfister DA, Pfister RR. Acid injuries of the eye. In: *Fundamentals of Cornea and External Disease*. *Cornea* 1997; 1 : 1437-42.
7. Pfister RR, Pfister DA. Alkali injuries of the eye. In: *Fundamentals of Cornea and External Disease*. *Cornea* 1997; 1 : 1443-51.
8. Ucakhan OO, Koklu G, Firat E. Nonpreserved human amniotic membrane transplantation in acute and chronic chemical eye injuries. *Cornea* 2002; 21 (2) : 169-72.
9. Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. *Surv Ophthalmol* 1997;41 (4) : 275-313.
10. Wagoner MD, Kenyon KR. Chemical injuries of the eye. Clinical Practice. In: Albert, Jakobiec, eds. *Principles and Practice of Ophthalmology* 1994; 1 : 234-45.