Chemical, Thermal, and Biological Ocular Exposures

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Chemical or radiant energy injuries to the eyes are considered ocular burns. The majority of these injuries are occupation-related \cite{1}. Chemical burns are by far more common and represent a true emergency. Thermal and UV injuries are associated with severe pain, but often result in less long-term sequelae than chemical injuries do. The term “biologic exposure” refers to an exposure to human blood or other body fluid. This article describes patterns of these injuries and exposures, with particular emphasis on emergent management and including acute diagnostic and treatment considerations.

**Chemical burns**

Chemical burns to the eye are common, particularly in industrial settings, and constitute an ocular emergency. In fact, chemical burns were the second leading cause of work-related eye injury treated in United States emergency departments in 1999 \cite{2}. A burn may occur with exposure of the eye to any chemical, solid, liquid, or aerosol. Household cleaning supplies and cosmetics are common offenders. The potent alkaline or acidic substances contained within these products cause the burn injury. Accidents involving industrial materials in the workplace are a frequent cause of eye burns.

Chemical exposures to the eye can result in significant damage to the ocular surface epithelium, the cornea, and the anterior segment. Permanent unilateral or bilateral visual impairment may result. Alkaline substances can
be particularly injurious. An alkali causes liquefactive necrosis of the surface epithelium, leading to rapid penetration of the substance to the deeper layers of the eye. There can be irreversible damage to the corneal stroma and endothelium, as well as to anterior segment structures. Most acidic substances cause coagulation necrosis when exposed to the cornea. The reaction precipitates surface proteins, which serve as a barrier to deeper damage, and acidic injury tends to be superficial [3].

**Pathophysiology**

The external portion of the eye is covered by the cornea centrally and the conjunctiva peripherally. The cornea is a multilayered structure atop a basement membrane, all covered by a thin film of tears. Both the cornea and the conjunctiva produce a nonkeratinized epithelium, and both have the ability to rapidly regenerate and renew surface epithelium during injury repair [3]. Within minutes of a small injury to the corneal epithelium, the regenerative process begins. However, after a larger injury, healing may take 4 to 5 hours to begin. Injuries that destroy the basement membrane may require up to 6 weeks to complete healing. After significant injuries, full restoration of the cornea may never occur [4].

**Alkali injury**

The severity of chemical burn injury is directly proportional to the surface area of contact on the cornea and the depth of penetration of the substance. Alkalis penetrate into the eye more readily than do acids. The hydroxyl ion of a base causes saponification of the fatty acids within the cornea, resulting in epithelial cell disruption and death. The associated cation then penetrates toward deeper structures. Depending on the degree of penetration, a number of structures may suffer injury, including the corneal and conjunctival epithelium, the basement membrane, stromal keratocytes, the lens, stromal nerve endings, the episclera, the iris, and the ciliary body (Figs. 1 and 2) [3].

A number of alkaline substances cause ocular injury. Some of the more common agents are listed in Table 1. The most serious alkali injuries are associated with exposures to ammonia (anterior segment injury in <1 minute) and to lye (deep injury within 3–5 minutes). Magnesium hydroxide, which is present in fireworks, may produce a severe injury because of the coexistent thermal burn [3]. Eye injuries secondary to methamphetamine production have been described as well [5].

On presentation, a patient with an alkaline eye burn demonstrates conjunctival hyperemia, chemosis, and corneal clouding. The stroma may have mild edema, and the anterior chamber may develop cells and flare. Severe alkali burns are characterized by corneal opacification and limbal ischemia [6].
Acid injury

Acids cause less severe, more focal tissue injury. The corneal epithelium offers moderate protection against weaker acids. The hydrogen ion alters surface pH, while the associated anion reacts with epithelial and superficial stromal cells to precipitate and denature surface proteins [3]. The coagulated proteins function as a superficial barrier and prevent intraocular injury. Stronger acids may penetrate and produce an injury pattern comparable to that of an alkali burn, as deep tissue damage occurs when the exposed eye reaches a pH of 2.5 or less [6].

Some common acidic agents that cause ocular injury are listed in Table 2. Sulfuric acid is the most common cause of acid injury. It seldom produces
serious injury unless there is additional damage from thermal injury or high-velocity penetration of a foreign body into the eye, as may occur in the explosion of an automobile battery [3]. Acids containing heavy metals may penetrate and produce injury patterns similar to those seen in alkali burns [6].

Hydrofluoric acid (HF) exposure can cause the most potentially serious ocular acid injury and deserves special consideration. HF is a low molecular-weight acid found in many industrial and commercial products. It can exist in gaseous or aqueous form. After exposure, HF penetrates deeply in tissues and causes liquefaction necrosis, much like an alkali. HF contamination may cause significant injury to multiple organ systems. In the eye, HF can rapidly penetrate to anterior chamber structures and cause devastating ocular injury [7,8].

In burns caused by strong acids, the cornea and conjunctiva rapidly become white and opaque. The epithelium may slough, leaving a relatively clear stroma. This may initially mask the severity of the injury. The cornea eventually becomes opacified. Very severe acid burns also cause complete corneal anesthesia, limbal pallor, and uveitis [6].

Table 1
Common products containing alkali

<table>
<thead>
<tr>
<th>Product</th>
<th>Chemical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lime</td>
<td>Calcium carbonate or magnesium carbonate</td>
</tr>
<tr>
<td>Plaster and mortar</td>
<td>Calcium hydroxide</td>
</tr>
<tr>
<td>Oven and drain cleaner (lye)</td>
<td>Sodium hydroxide or potassium hydroxide</td>
</tr>
<tr>
<td>Fireworks and sparklers</td>
<td>Magnesium hydroxide</td>
</tr>
<tr>
<td>Ammonia (in cleaning agents and fertilizers)</td>
<td>Ammonium hydroxide</td>
</tr>
<tr>
<td>Dishwasher detergent</td>
<td>Sodium tripolyphosphate</td>
</tr>
</tbody>
</table>


Table 2
Common products containing acid

<table>
<thead>
<tr>
<th>Product</th>
<th>Chemical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toilet cleaner</td>
<td>Sulfuric acid (80%)</td>
</tr>
<tr>
<td>Battery fluid</td>
<td>Sulfuric acid (30%)</td>
</tr>
<tr>
<td>Pool cleaners</td>
<td>Sodium hypochlorite or calcium hypochlorite (70%)</td>
</tr>
<tr>
<td>Bleaches</td>
<td>Sodium hypochlorite (3%)</td>
</tr>
<tr>
<td>Vinegar or essence of vinegar</td>
<td>Acetic acid</td>
</tr>
<tr>
<td>Glass polishers, silicone production agents, rust removal agents</td>
<td>Hydrofluoric acid</td>
</tr>
<tr>
<td>Food- and leather-processing compounds</td>
<td>Hydrochloric acid</td>
</tr>
</tbody>
</table>

Cyanoacrylate exposure

Cyanoacrylate is an adhesive that results in strong polymer bonds with a variety of materials and is used in both industrial and domestic settings [9]. It is a substance considered to have relatively little toxicity. It has also been used by ophthalmologists during surgical procedures to seal globe injuries without incident [10]. However, the emergency department clinician may encounter a patient who inadvertently instilled cyanoacrylate-containing adhesives into the eye. Alternatively, the clinician may accidentally spill skin adhesives into a patient’s eye when repairing lacerations on the forehead or face. Such occurrence can result in adherence of the upper and lower eyelids. This may lead to local dermatitis of the eyelids, as well as to eyelash loss [10]. Occasionally, cyanoacrylate may collect over the cornea and irritate the eyes, resulting in mild to moderate corneal abrasions [11]. In such exposures, treatment as per the management for corneal abrasions is indicated. Attempting to pry the eyelids open should be done with caution, as this may result in further injury [12]. In the vast majority of cases, the adhesive separates from the eyelids within 1 week. As needed, irrigation with normal saline may remove particulate debris in and about the eye. Otherwise, no other specific interventions are indicated, and expectant management is appropriate [9–12].

Treatment

For the emergency physician, the management of chemical burns is contingent upon the time elapsed since injury. Eye injury due to chemical exposure occurs in phases. The immediate or acute phase occurs at the time of the injury and results in corneal and conjunctival epithelium necrosis and chemical invasion into the anterior chamber, the ciliary body, and the iris. The later phases of eye burns (intermediate and chronic) occur over the subsequent days to weeks and require management by ophthalmologic and plastic surgery specialists.

The goal of treatment after a chemical burn is to restore physiologic pH of the eye as rapidly as possible. If the external pH is restored to normal, the aqueous pH within the eye returns to normal within 30 minutes [3]. The most important initial intervention for all chemical exposures is irrigation. Delays in the initiation of irrigation by as little as 20 seconds have been demonstrated in animal models to result in a higher maximal pH and higher risk for more severe injury after alkaline exposure [13]. If possible, irrigation should begin immediately after injury, even before a patient presents to an emergency department. A number of studies have looked for the best agent for irrigation, and certain solutions containing buffering agents have been proposed as ideal [14]. However, even tap water is an appropriate irrigating solution [15,16], and any nontoxic solution, such as normal saline or lactated Ringer’s solution, is effective. If the nature of the chemical injury is
unknown, one may apply a piece of pH paper to the inferior fornix to determine if the offending substance was basic or acidic, but this should not delay the start of irrigation.

To permit appropriate irrigation of the chemically exposed and inflamed eye, topical anesthesia with 0.5% proparacaine may be necessary. Lid retractors or a lid speculum may be helpful. Prolonged irrigation can be accomplished with the use of a polymethylmethacrylate scleral lens with an attached perfusion tube. If such a lens is not available, an angiocath may be inserted percutaneously across the upper lid, though this should be reserved for rare instances and after discussion with consultants [6].

Chemical burns can be quite painful. Topical anesthesia may help. Additionally, the clinician should be prepared to provide the patient with systemic analgesics. For rapid pain relief and for ease of administration during irrigation, parenteral nonsteroidal anti-inflammatories or narcotic analgesics should be strongly considered.

Classically, ophthalmologists have suggested irrigation for 30 minutes or until 2 L of irrigant has been applied [6]. However, most now advocate continued irrigation until the eye is restored to normal pH (checked at 30-minute intervals). In animal models, greater than 2 hours of irrigation were necessary to achieve normal pH in the aqueous humor after a severe alkali burn [17]. Many ophthalmologists recommend reassessing pH approximately 30 minutes after the eye is restored to normal pH, especially after alkali burns, as particulate matter lodged in the conjunctiva can dissolve slowly and cause persistently increased pH, requiring further irrigation [6].

Emergent referral to an ophthalmologist is indicated in all but the most minor of injuries. Considering that injury severity can be difficult to ascertain upon emergent presentation, urgent follow-up is recommended for all chemical burns that cannot be evaluated by the ophthalmologist in the emergency department.

In a patient that reports an eye splash of rust remover, leather-tanning fluid, high-octane gasoline or glass or enamel etching materials, the clinician should be suspicious for an HF exposure. This is significant because, even though irrigation with water or saline should be instituted immediately, some reports suggest prolonged or repetitive irrigation can increase corneal ulceration and worsen outcome following HF exposure [8,18]. As topical calcium gels are indicated for dermal exposures to HF, some have suggested that application of 1% calcium gluconate drops to affected eyes may be protective after an HF burn [18,19]. However calcium salts can be quite irritating to the eye, and most would advise use of calcium gluconate drops only after discussion with an ophthalmologist [7,8].

Once irrigation is complete, a thorough evaluation for any particulate matter should be performed. This must include eversion of both upper and lower lids, and retraction of redundant conjunctival tissue. Topical anesthetic may be helpful. Any particles can be removed with a moistened cotton-tip applicator. Larger particles can be removed with smooth forceps.
As with all patients who present to an emergency department with eye complaints, an assessment of visual acuity must be performed. For patients who do not arrive in the emergency department with their corrective lenses, pinhole refraction can aid in the evaluation. A comprehensive slit-lamp examination is indicated for all patients. All but the mildest of burns develop a significant uveitis. In such cases, a clinician should appreciate cells and flare in the anterior chamber. For pain relief and prevention of synechiae (adhesion of the iris to the cornea or lens), cycloplegic eye drops (eg, cyclopentolate 0.5%) are indicated. Phenylephrine is contraindicated because it can exacerbate ischemic injury to deeper structures. Intraocular pressure should be assessed after a burn and, if elevated, systemic treatment with carbonic anhydrase inhibitors is indicated [6].

As with all patients presenting with superficial injury to the eye, a fluorescein exam is indicated. Fluorescein uptake occurs in all areas where the chemical exposure produced injury. An ophthalmologist will follow the observed injury pattern over time.

The goal of treatment is to promote epithelial cell growth as rapidly as possible. Topical antibiotic ointment, such as erythromycin or tetracycline, should be applied. Some investigators suggest topical corticosteroid to reduce the inflammatory response to chemical injury (ie, prednisolone or dexamethasone) [6]. However, this should be used in close collaboration with an ophthalmologist [20]. Although a semipressure patch can be applied to relieve pain [4,6], this has been shown to provide no benefit in the emergent treatment of traumatic corneal abrasions [21–23]. Rapid follow-up with an ophthalmologic specialist to manage care is vital.

**Thermal injuries**

Thermal injuries to the eye generally occur as a result of exposure to scalding liquid, direct flame, or such items as cigarettes and curling irons. Although a direct thermal injury to the eye is a rare event, burns to the surrounding adnexal structures (ie, eyebrow, eyelid, eyelashes) are not uncommon [6,20]. Due to the rapidity of the lid reflex, the eyelid provides protection for the eye itself, and long-term visual acuity is preserved. However, the eyelid takes on the majority of insult in a thermal injury, which leads to an inordinately high rate of postinjury lid contractures [24]. Much like chemical burns, the severity of thermal eye burns is related to the duration of exposure and the nature of the causative agent. Compared to water-based liquids, hot oils and greases are more adherent and subsequently result in deeper thermal injury [25,26]. The grading system for thermal burns to the eyelid is similar to that for burns in other areas of skin [27]. First-degree or superficial partial-thickness burns to the eyelid affect only the epidermal layer of skin. This type of burn pattern is characterized by pain, erythema, and edema. Second-degree or deep partial-thickness burns affect both the epidermal and dermal layers of skin, and also exhibit pain and erythema. However,
second-degree burns blister. Third-degree or full-thickness burns of the eye-lid affect the entire dermal layer and may extend into the subcutaneous tissue. Patients present with a black or white eschar over the adnexal structures of the eye, which may lead to contractures. Full-thickness burns damage nerve endings and are less painful or even painless.

Radiation injuries

UV radiation (wavelengths 295–400 nm) is primarily absorbed by the corneal surface [28,29]. Prolonged unprotected exposure to sunlight, particularly at high elevation or with highly reflective surfaces, result in damage to the corneal surface (eg, snow blindness) [6,30]. UV damage to the corneal epithelium results in an inflammatory keratitis known as superficial punctate keratitis. The pinpoint corneal defects characteristic of superficial punctate keratitis can readily be found on fluorescein staining. Additionally, there is often a varying degree of conjunctival injection and eyelid edema. The onset of pain and decreased visual acuity is delayed and occurs approximately 6 to 12 hours after prolonged UV light exposure. Overall, the epithelial layer heals within 72 hours of the injury, and the long-term prognosis for UV burns is excellent.

Artificial sources of UV radiation include welding arcs, sun-tanning beds, electric sparks, and halogen desk lamps. In contrast, prolonged exposure to infrared radiation (wavelengths of >700 nm) damages the anterior lens and may lead to cataract formation (eg, glassblowers cataracts) [6,30]. This occupational injury is rarely seen now that appropriate eye protection is used.

Prolonged exposure to visible solar light (wavelengths 400–700 nm) may rarely result in retinal injury and subsequent visual loss [6,30]. This delayed photochemical retinal injury most commonly occurs among beachgoers and persons engaged in maritime industries who are exposed for long periods in the sun without eye protection. Patients often present 1 to 2 days after exposure with complaints of pain and visual change. On examination, there are often minimal findings apart from a reduction in visual acuity. Although no immediate management is necessary, outpatient ophthalmology follow-up is necessary, as the visual loss may take several months to improve.

Treatment

The treatment for thermal injuries is comparable to treatment for chemical burns. For all thermal injuries of the eye, the first priority is to remove the individual from the source and to cool the tissues as rapidly as possible. The eye should be irrigated with copious normal saline, lactated Ringer’s solution, or tap water. Ideally, this should be instituted at the scene (eg, coworkers at the worksite) or before emergency department evaluation (eg, by pre–hospital-care providers), and should be continued in the emergency department. The removal of adherent debris or retained contact lenses
is facilitated via copious irrigation. The initial use of topical anesthetic (eg, proparacaine 0.5%) is suggested in the emergency department, but long-term application of these agents delays epithelial healing. Cold compresses should be applied to the affected areas to decrease thermal injury and to relieve discomfort. Both thermal and UV injuries are quite painful, so oral and parenteral nonsteroidal and narcotic analgesics should be prescribed. Topical analgesics have not been shown to be superior to oral agents [31,32]. Once pain control is addressed, visual acuity testing should be performed to identify any baseline visual deficits. Pinhole refraction may optimize visual acuity assessment among selected patients who arrive in the emergency department without their corrective lenses. While evaluating the eye, the eyelids should be everted so that a complete inspection of the stromal structures can be performed. Frequent application of artificial tears or ointment should be instituted in the event that tear production has been affected. This also helps to prevent long-term adhesion formation between the eyelid and the globe itself. The patient’s tetanus status should be updated. Systemic antibiotics are unnecessary unless the source of infection has been identified.

In addition to the above actions, the specific treatment for first- and second-degree thermal eye burns includes an antibiotic ointment (erythromycin 0.5%) or eye drops. Cycloplegic agents (eg, cyclopentolate 0.5%) are commonly administered. However, a recent systematic review found no compelling evidence to support this practice [33]. For third-degree burns, a nonocclusive dressing should be applied to prevent infection, and urgent ophthalmologic consultation is warranted.

The traditional therapy for UV keratitis is cycloplegic drops (eg, cyclopentolate 0.5%) to reduce reflex ciliary muscle spasm and to potentially reduce pain. However, as noted above, no studies have proven the efficacy of this treatment [33]. Topical antibiotic drops or ointment (eg, erythromycin 0.5%) should be prescribed. Although some ophthalmologists continue to recommend eye patching, this is viewed as controversial and should be done with caution [21–23].

### Biologic exposures

In practical terms, a “biologic” exposure refers to contact with human blood or body fluids (eg, saliva, semen, urine). The key issue that the emergency physician must confront is that such an exposure may result in an infection with blood-borne pathogen. The most serious of these pathogens include hepatitis B virus (HBV), hepatitis C virus, and HIV [34,35]. The infectious risk increases if the biologic exposure (ie, blood, body fluid) comes into direct contact with the exposed person’s bloodstream (eg, needle-stick incident). However, exposure to the mucous membranes of the eye can potentially result in infection. The risk of infection via exposure through mucous membranes or injured skin is considerably less than that via a percutaneous needle-stick
event. Although the true risk is lower with mucous membrane exposures, the risk is not zero [34]. The risk of infection is related to the amount (ie, volume) and concentration (ie, viral load) of the biologic exposure.

**Treatment**

The most important action that the emergency physician can make in the treatment of biologic exposures to the eye is to irrigate the eye with water or normal saline. For those who wear contact lenses, recommendations call for the lenses to be kept on during irrigation because the lens itself serves as a protective barrier for the eye. The use of soap or other agents may irritate and compromise the mucosal barrier of the eye. Lenses may be removed after irrigation and either discarded or cleaned as per the usual manner. Soap and water may be used, however, for biologic exposures to nonintact skin of the orbital adnexal structures [34].

Of the three viral agents discussed, the risk of hepatitis B infection is greatest. Fortunately, in the health care environment, most hospital personnel are vaccinated against HBV. Among unvaccinated persons, the emergency department clinician should consider initiating treatment with HBV vaccination in ocular exposures with blood from source individuals where the HBV status is unknown. The first dose should be given at the time of exposure; a follow-up vaccination is given at 1 month and 6 months postexposure. If the blood is known to be from an HBV-positive source, then hepatitis-B immune globulin, which contains antibodies to HBV, is recommended [34,35].

The Centers for Disease Control and Prevention recommends postexposure prophylaxis to reduce HIV infection if the exposure is discovered within 72 hours and one or more of the following bodily fluids was involved: blood, semen, vaginal secretions, rectal secretions, breast milk, or any body fluid that is visibly contaminated with blood [35]. However, given that mucous membranes of the eye are an extremely unlikely route of HIV transmission, it is recommended that each situation be considered on an individual basis [34,35].

Unfortunately, for biologic exposure to hepatitis C virus, there is no known effective preventive measure apart from copious irrigation with water or saline.

In cases of occupational biologic exposures to the eye, health care workers may be referred to the hospital’s occupational health clinic for same-day or next-day baseline serum testing, as well as follow care. All others should be referred to a primary care provider for further management.

**Disposition**

Most first-degree thermal burns and radiation injuries may be discharged from the emergency department with outpatient ophthalmology follow-up.
in 24 to 48 hours. Second-degree burns of the eye merit at least a phone call to the on-call ophthalmologist, or referral for urgent ophthalmology consultation. Third-degree eye burns require ophthalmologic consultation and either hospital admission or early burn-center transfer. All those with biologic exposures should be referred for baseline and follow-up testing and management.

References