

Chemical Ocular Burns: A Case Review

Adeola Kosoko, BA, M3

Qui Vu, BS, M3

Omofolasade Kosoko-Lasaki, MD, MSPH, MBA

Abstract

Chemical injury to the eye accounts for a significant portion of ocular trauma. Proper management in the acute setting as well as follow-up by an ophthalmologist is crucial in limiting adverse effects of ocular tissue damage secondary to the chemicals. This case study describes the presentation and treatment of an adult male who presented to the Emergency Department (ED) at Creighton University Medical Center (CUMC) following an accidental occupational facial and ocular chemical injury. Described is the immediate management of ocular injury secondary to chemical exposure and the follow-up care provided by the ophthalmologist. In addition, detailed is the medical and surgical management of ocular chemical burns including the preventative strategies utilized. The management of the case described may serve as an approach to the care of patients presenting with facial and ocular burn injury in the ED.

Introduction

Chemical injuries to the eye are common and represent one of the “true” ophthalmic emergencies. Practically any chemical can cause ocular irritation. Most of these injuries are inconsequential and do not cause lasting lesions (e.g., shampoos, defense sprays, household cleaning solutions, etc.) while others may result in permanent debility.¹ Severe ocular damage is most commonly associated with strong alkaline or acidic compounds. Chemical burns may be induced by means of vapor, solid, or liquid. Nonetheless, the majority occur in industrial environments, in laboratories, in combative environments, or as a result of an accident.^{2,3}

The severity of chemical injury is related to the type of chemical, the volume of direct exposure, the pH of the solution, and the duration of exposure.^{3,4} Alkali burns are the most common cause of ocular burn and also tend to cause the most ocular damage.³ Bilateral caustic exposure has been shown to be particularly injurious. Regardless of the type of chemical, the

treatment modalities are similar with an initial immediate and extensive ocular irrigation for best patient outcomes.⁵

In this paper, we submit the case of a patient that presented to Creighton University Medical Center (CUMC) Emergency Department (ED) after an accidental chemical exposure, resulting in injury to the face and eyes.

Case Report

B.P. is a 29 year-old male patient who presented to the CUMC Eye Clinic a few hours after he was seen, evaluated, and referred by the ED physicians at the same hospital. The patient had presented to the ED via ambulance approximately two hours after an accidental chemical explosion while performing maintenance duties in a laboratory at his workplace. According to the patient, the cleaning solution that exploded was a mixture of sulfuric acid, chromic acid, and sodium hydroxide. He reported wearing only his prescription glasses at the time of the accident. Because of ocular discomfort, foreign body sensation, burning sensation, blurred vision, excess tearing in the right eye, and a partial thickness laceration above the right eyebrow, B.P. was transferred to the ED at CUMC.

Image 1: First day post chemical burn injury to right eye



The ED notes indicated that B.P.'s eyes were irrigated with one liter of water until the ocular pH was 7. The ED physicians applied proparacaine hydrochloride (Alcaine® 0.5%, Alcon Laboratories, Inc.) to the eyes along with fluorescein sodium (Fluor-1-Strip, fluorescein sodium 1mg, Bausch & Lomb). Examination of the eye with cobalt blue light confirmed multiple superficial corneal abrasions. There was no evidence of corneal lacerations. B.P. was referred to the CUMC Eye Clinic after his eyebrow superficial laceration was secured with 5-0 prolene sutures, and he was administered intramuscular tetanus toxoid (Adacel™, Sanofi Pasteur) because his current immunization status was unknown.

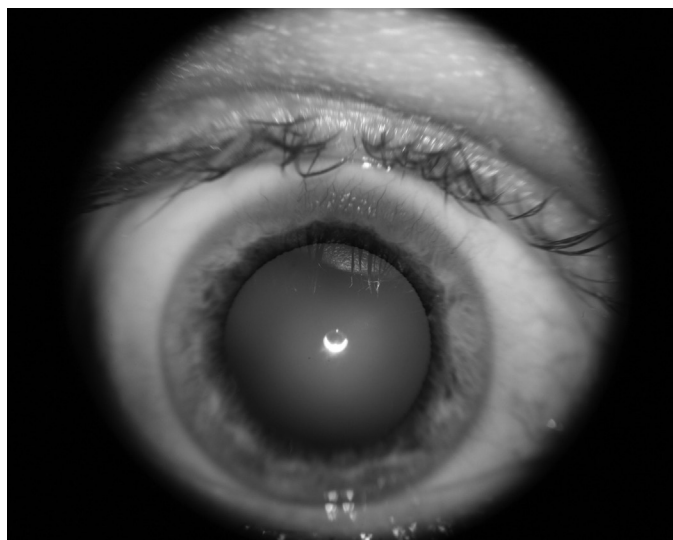
B.P. presented as a "walk-in" to the CUMC Eye Clinic with a history as stated above. His past medical history was unremarkable. He denied any allergies to food or medications. He was not taking any prescription, over-the-counter, or herbal medications. His past ocular history was significant for myopia, corrected with eyeglasses that were last renewed two years ago. In fact, he had on his prescription eyeglasses during the time of the chemical explosion in the laboratory. B.P. denied past history of ocular trauma, ocular surgery, or contact lens wear. Family history was non-contributory. He lives at home with his girl friend and is an employee of a local engineering specialty firm, the place where the chemical explosion had occurred. He admits to occasional alcohol consumption but denied any history of cigarette smoking or illicit drug use.

On examination, B.P. was a well-developed 29 year-old male in acute ocular distress. Inspection of the patient's face (see Image 1) revealed complete chemosis of the right eye that was more severe in the superior palpebral aspect. He had four interrupted 5-0 prolene sutures above the right upper eyebrow that was placed in the ED. His visual acuity was 20/80 in the right eye, pinhole to 20/30 and 20/100 in the left eye, pinhole to 20/30 using the Snellen's visual acuity chart at distance. Pupillary exam shows his uncorrected Snellen both pupils were equal, round, and reactive to light and to accommodation. There was no afferent pupillary defect. Confrontation visual field was full in the left eye. The right eye had some limitation because of his severe eyelid chemosis. Slit-lamp examination was significant for conjunctival congestion in the right eye with an inverted V-shape abrasion of the right cornea. A complete ocular exam was deferred until his returning visit on the following day. Examination of the left eye was normal without signs of trauma or abnormalities.

The diagnoses of chemical burn to the face with right eyebrow superficial laceration and right corneal abrasion were made. The patient's right eye was irrigated again with eye wash in the CUMC Eye Clinic, and the conjunctival cul-de-sacs were cleaned with Q-tips to remove any residual chemical or debris. The right eye was dilated with cyclopentolate (Cyclogyl® 1%, Alcon Laboratories, Inc.), and erythromycin ophthalmic ointment 5 mg/g (Romycin® 0.5%, OCuSOFT) was applied. After the eye was protected with an eye patch, the patient was discharged home, instructed to leave the patch on until the next day, and he was to follow up in the CUMC Eye Clinic in 24 hours.

The following day, patient was seen again in the CUMC Eye Clinic. His visual acuity in the right eye without correction had improved to 20/50 with a pinhole of 20/30. There was also a slight decrease in chemosis of the right eye conjunctiva, and the corneal abrasion was almost healed. A complete exam was done at this time which was normal. The left eye exam was also normal, which included applanation tonometry and a dilated ocular exam.

Image 2: Right eye with faint corneal opacity at the 7 o'clock position



The patient was instructed to continue with the erythromycin ointment application three times a day to the right eye and was given Maxitrol® (Alcon Laboratories Inc.), a neomycin-polymyxin B sulfate-dexamethsone combination ophthalmic ointment, to apply to the upper eyebrow laceration skin three times daily. The patient was instructed to follow up in one week or earlier if he experiences increased ocular pain or changes in vision. Subsequent visits to the CUMC Eye Clinic were uneventful with the skin laceration healing well. Skin sutures were removed after five days of placement. The corneal abrasion had also resolved; however, it was noted that the patient had developed a stromal opacity around the area of the initial abrasion. He was subsequently started on 1% prednisolone acetate (Pred Forte 1% Ophth Susp, Allergan) to the right eye with a slow taper over one month, while his intraocular pressure was monitored.

Discussion

Chemical injuries of the eye are true emergencies requiring a good history, prompt clinical evaluation, and treatment. The severity of the injury relates directly to the duration, the type of the chemical, and the deviation of the corrosive substance from the physiological pH.⁶ In the case presented, the patient sustained an explosive chemical injury caused by a cleaning solution comprising both acidic and alkali chemicals, with the primary component being acidic in nature.

The principles of management in either an acid or alkali injury are similar, but the long-term prognoses are quite different.

Rapid irrigation and dilution of the chemical with a neutralizing solution, preferably water, or even tap water, is the immediate first step of treatment in order to reduce tissue damage and preserve vision.^{7,24} The patient, B.P., apparently did not irrigate his eyes before presenting to the ED. Once the physiological pH is restored by performing frequent checks between irrigation episodes, the medical history and examination should be conducted. Baseline visual acuity and ophthalmic exam findings are important in documenting and counseling patients and family members regarding the prognosis and final visual outcomes. Findings supporting a favorable outcome include a visual acuity that is unchanged or minimally deviated from the patient's baseline and the absence of limbal ischemia or corneal opacity.^{3,7} Findings present in the case being discussed. However, more discouraging findings for final visual outcome will include the presence of ischemic necrosis of the proximal conjunctiva and sclera, significant corneal opacification, and ischemia in the area of the limbus.

Application of topical antibiotics is generally necessary until the ocular surface has re-epithelialized.⁶ The slit lamp examination with the use of fluorescein staining is essential to determine the extent of the injury, past the corneal endothelium and into anterior segment structures, such as the iris, lens, and ciliary body.³ Cycloplegic agents, such as cyclopentolate 1% or atropine 1% (Atropine-Care 1%, Akorn), were administered twice daily to prevent ciliary spasm.¹⁷ The late stage corneal stromal opacity was treated with mild steroid. Studies have shown that steroids enhance deep stromal healing after the initial epithelial abrasion has been addressed.^{3,17} Since severe ocular eye burns are difficult to treat and the course of healing often takes several months, all cases of ocular chemical injuries should be closely monitored, especially within 24 hours of the injury, in order to monitor progress and identify any sign of complications.⁶

Epidemiology of Ocular Chemical Injury

Ocular burns may represent up to 18% of ocular traumas presenting to emergency departments. Of those ocular burns, the majority, 84%, are secondary to chemical injury.⁸

Ocular chemical injuries most commonly occur in industrial work environments, though there are a significant number of cases that occur in the home.⁵ Occupational chemical exposure causes about 7% of work-related ocular injury that presents to emergency departments in the United States. Workplace accidents are responsible for 63% of chemical injuries to the eye while 33% can be attributed to accidents within the home. Ten percent of cases are a result of assault, which is a significant cause in lower socioeconomic groups of urban areas.^{5,9}

Men have a threefold greater predilection for chemical ocular burns compared to women. Although, chemical injury has been identified in various ages, victims tend to be younger, 16-45 years of age. There is no specific racial group more predisposed to ocular chemical injuries.

Caustic agents are the primary offenders for severe chemical ocular burns, with alkali burns being reported more commonly than acid burns.^{3,5} Some of the more common chemical agents are listed in Figure 1.^{3,9} Although calcium hydroxide is the most common cause of alkali ocular burns, ammonia tends to cause the most serious burns.¹⁰ Regarding acids, hydrofluoric acid causes the most devastating injury, while sulfuric acid is the most common acidic caustic agent.¹⁰

It is not uncommon for ocular burns to be bilateral. Reported complications of chemical injury include vision loss, glaucoma, cataracts, corneal ulceration/perforation, corneal scarring, retinal detachment, and conjunctival and eyelid defects.¹

Figure 1: Common Caustic Agents and Their Sources

<u>Substance</u>	<u>Class</u>	<u>Common pH</u>	<u>Source</u>
Sodium hydroxide	Alkali	14.0	lye soaps, airbags, hair relaxer
Calcium hydroxide		12.4	mortar, plaster, cement
Ammonium hydroxide		11.6	fertilizers, refrigerants, sparklers
Sodium hypochlorite		11.0	bleaches, drain cleaners
Magnesium hydroxide		10.0	oven & drain cleaners
Acetic acid	Acid	2.9	high vinegar concentrations
Hydrofluoric acid		2.1	rust removers, glass, mineral, gasoline, silicone industries
Sulfurous acid		1.5	bleach, refrigerants
Sulfuric acid		1.2	industrial cleaners, battery acid
Hydrochloric acid		1.1	household and pool cleaners

Figure adapted from *Emergency Medicine Procedures*⁹

Pathophysiology of Ocular Chemical Injury

The major pathophysiology during chemical ocular burns causes damage to the eyelids, conjunctiva cornea, and the anterior segment of the eye. At these locations, the damage has the potential to cause permanent visual impairment based on the volume, pH, duration of exposure, and degree of penetration of the chemical offender.³ It is important to note the type of chemical, because the mechanism of injury varies between acidic and alkaline exposure.

Acid Burns

Acidic chemicals are categorized by having a low pH and readily dissociate into hydrogen ions and anions within the anterior surface. They are less common than alkali burns and are usually less damaging. The hydrogen ions produced by chemical dissociation cause a change in pH within the ocular plane itself. The anions produced from dissociation cause the denaturation, precipitation, and coagulation of proteins (coagulation necrosis), which causes clouding of the once transparent ocular surfaces. It is of note that the protein coagulation makes acid injury less damaging than alkali injury, often limiting the burn to the anterior of the eye. There is indeed damage due to the coagulation process, but it is protective in that it prevents further penetration of the insulting ocular offender.^{9,11}

Hydrofluoric acid is a notable exception of the typical chemical course of acidic substance and ocular damage. The fluoride ion is able to penetrate deeply despite the protein coagulation that may have occurred. It, therefore, causes more invasive damage than most acidic compounds, comparable to damage caused by an alkali substance. Still, higher concentrations of any acid or failure of rapid intervention with any acid has the potential for more invasive damage than is typically expected for an acidic compound.^{3,12}

Alkali Burns

Alkali chemicals are categorized by having a high pH and readily dissociate into hydroxyl ions and cations within the anterior surface. They are the more popular form of ocular caustic agent and tend to be more damaging. The resulting hydroxyl ions cause saponification, which combines with fatty acids and proteins, notably causing liquefactive necrosis, as opposed to the coagulative necrosis of acids. The cation dissociated from the offending agent is also active in interaction with collagen and glycosaminoglycans of the stroma, causing a fogging of the stroma. The extensive breakdown of tissue within the cornea is significantly detrimental, because it facilitates deeper penetration of the chemical and infiltration of the anterior segment.^{9,11}

Penetration of the chemical into the anterior segment, along with collagen hydration, malignant fibril changes, and trabecular changes may cause a rapid (seconds to minutes) and significant change in intraocular pressure (IOP) second to a rapid rise in aqueous humor.^{3,12} This may result in iritis, glaucoma, and decreased visual acuity, with increased morbidity.

Management of Ocular Chemical Burns

History

A thorough history will aid in identifying the chemical agent(s) involved and detail the circumstances surrounding the injury. In the patient's own words, the history should include the type and form of the chemical, if known by the patient, the quantity, concentration, duration of exposure, mechanism of injury, and the events occurring from the time of injury until the time of presentation.¹³ These questions help the physician assess the extent and severity of the injury, which frequently correlate with the patient's complaints. Examples of common complaints are acute onset of pain or burning sensation, foreign body sensation, excessive lacrimation, blurred vision, red eye(s), swollen eyelids, and photophobia.⁷ Query the patient regarding whether the affected eye had received copious irrigation, since a thorough history and physical examination may be deferred until that is done. It is important to determine the pH of the tears in the conjunctival cul-de-sac, since irrigation may have to be repeated until a neutral pH is obtained. The physician should inquire into the patient's past ocular history including any previous chemical eye injuries or otherwise, ocular surgeries, contact lens use, or history of amblyopia. The list of current medications, allergies, family history, social history, and date of last tetanus vaccination will complete the highlights of the patient's medical history.

Ophthalmic Examination

The most important initial intervention for all chemical ocular injuries is copious irrigation and restoration of the physiologic pH of the eye. Therefore, a comprehensive ocular examination should be deferred until the affected eye(s) has received copious irrigation.⁷ After irrigation, topical anesthetic drops may be used to enhance patient comfort and cooperation.²⁰ In some instances, the patient may have severe blepharospasms warranting the instillation of a topical anesthetic drop before copious irrigation. A complete ocular exam is warranted. This includes visual acuity, slit lamp exam with corneal staining with fluorescein dye and exam with Cobalt blue light, applanation tonometry, and a dilated eye exam. Both eyes should be examined, even if it is obvious that the other eye is not affected.

The details of the complete eye exam can be assessed by using the following sequence of testing,^{3,14,15} which is also summarized in Figure 2. The examination of the eye should commence with an assessment of visual acuity. A Snellen visual acuity chart or a pocket eye chart can be utilized; however, counting fingers, hand movement, or light perception should be documented if the patient is unable to read the largest line on the visual acuity chart. Visual acuity should be repeated on subsequent visits, since patients may initially present with minimal corneal haze and good vision, then may experience reduced vision due to increasing corneal opacification that develops with time. Evaluate the eyelids for laceration or other abnormalities and note any involvement of the lacrimal system. Palpate the orbital rim for

the presence of fractures or crepitus, especially if the patient was involved in a traumatic chemical explosion. Note signs of conjunctival inflammation, such as conjunctival hyperemia and chemosis.¹⁶ Examine the conjunctival fornices for retention of chemical particles, especially in particulate chemical explosion, since the particles can serve as reservoir for continued chemical release and inflammation. Perform the pupillary examination, noting the shape, size, symmetry, and the direct and consensual responses to light of both pupils and an afferent pupillary defect. Evaluate extraocular muscle function in all directions of gaze.

Slit lamp examination of the orbit with the use of fluorescein staining and cobalt blue light enable the physician to determine the presence and extent of any corneal epithelial defect or abrasion.³ Depth of corneal penetration can be estimated by evaluating the loss of stromal clarity, and the depth of conjunctival penetration can be assessed by observing for signs of vascular ischemia and necrosis of limbal and bulbar conjunctiva. The degree of limbal ischemia,¹⁸ which has a blanching appearance, should be documented in terms of clock hours, since stem cells from the limbus move centripetally to repopulate the posttraumatic corneal epithelium; thus, it is an important prognosticator of future corneal healing. Additionally, the anterior segment fluorescein angiography may be helpful in estimating conjunctival and intraocular penetration by documenting the severity of anterior segment vascular ischemia. Intraocular pressure should be measured, since there may be thickening and shortening of ocular collagen fibrils distorting the trabecular meshwork and resulting in the release of prostaglandins, which in turn may increase the IOP in the future.^{3,19} The dilated fundoscopic examination is performed to document the status of the retina, macula, optic disc, and ocular vasculature. The pH of the ocular surface should be periodically tested and irrigation should be continued until the pH reaches physiological neutrality.

Classification Schema to Grade the Severity of Ocular Chemical Injuries

There are various classification schema for grading the severity of ocular chemical injuries, but the Hughes classification system, later modified by Ballen and Roper-Hall, is commonly used in the acute stage due to its greater simplicity.^{3,13} This classification system, as displayed in Figure 3, recognizes the correlation between the loss of corneal clarity and degree of limbal ischemia with the ultimate prognosis. This is an excellent indirect means of assessing the extent of limbal stem cell injury and is therapeutically useful, since it can form the basis of recommendations regarding appropriate consideration for early limbal stem cell replacement. Grade I injuries show neither limbal ischemia nor corneal opacity, thus, have an excellent prognosis. In Grade II injuries, there is less than one-third limbal ischemia and, despite the presence of cornea haziness, the iris details are still visible. The prognosis is good. Grade III injuries involve significant corneal haze as to obscure iris details, and ischemia is one-third to one-half of the limbus; thus, the prognosis is guarded. In Grade IV injuries, the cornea is

Figure 2: Synopsis of the Management of Ocular Chemical Burns

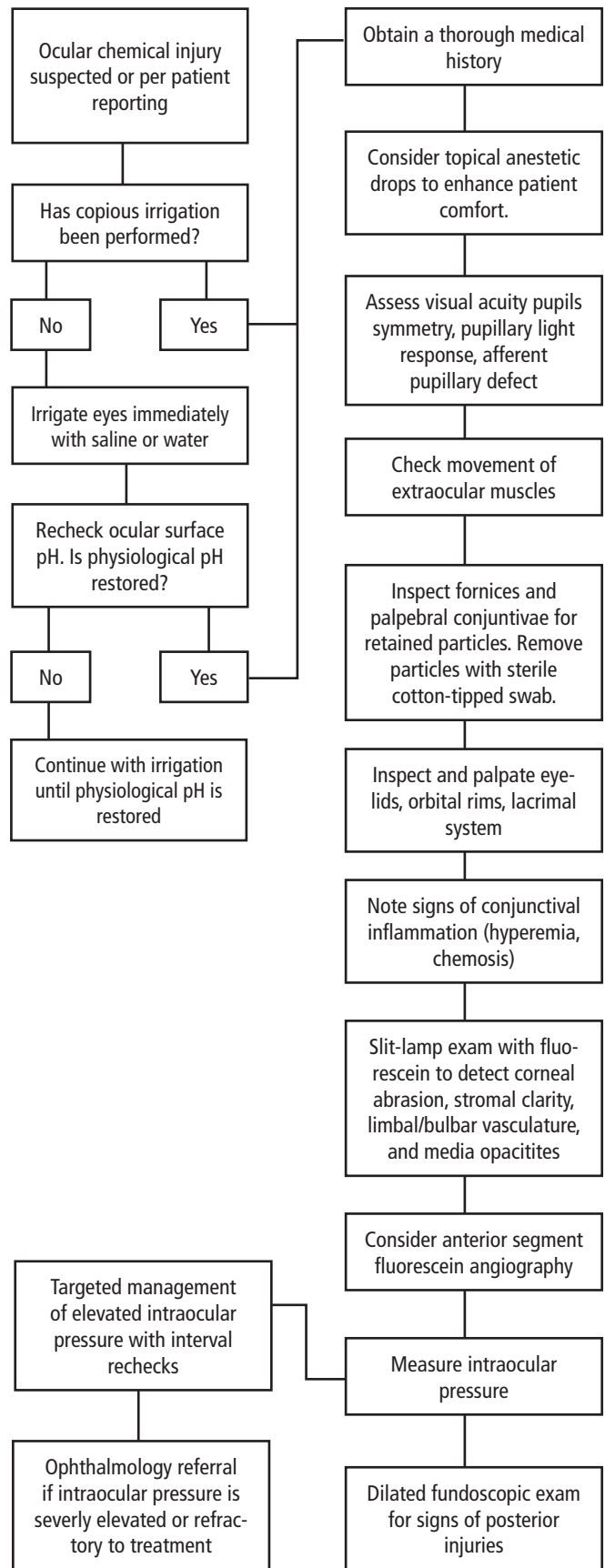


Figure adapted from references 3, 7, 14-16, and 18-20

Figure 3: Modified Hughes Grading of Ocular Chemical Injury Severity

Grade of Injury	Clinical Findings	Prognosis
Grade I	No corneal opacity No limbal ischemia	Excellent
Grade II	Cornea haziness, but visible iris details Ischemia < 1/3 of limbus	Good
Grade III	Significant corneal haziness to obscure iris details Ischemia 1/3 to 1/2 of limbus	Guarded
Grade IV	Cornea is opaque, no view of iris or pupil Ischemia > 1/2 of limbus Ischemic necrosis of proximal conjunctiva and sclera	Dismal

Figure adapted from references 3 and 13

opaque with no view of the iris or pupil, the ischemia is more than one-half of the limbus, there is ischemic necrosis of the proximal conjunctival and sclera, and the prognosis is dismal.

Medical and Surgical Management

Regardless of the type of the chemical agent involved, the common goals of management in ocular chemical injuries should include the removal of the injurious chemical agent; control of pain, intraocular pressure, and inflammation; prevention of infection; tetanus vaccination; and promotion of ocular epithelial healing.^{6,20} To achieve these goals and restore damaged visual structure, both medical and surgical interventions (Figure 4) can be employed acutely and in the long term, depending on the extent of ocular injury.

As stated previously, immediate copious irrigation for at least 15 minutes, but optimally until the ocular surface is neutralized, is the most imperative treatment after chemical burns.^{6,20,21} In a study by Rihawi et al. which looks at severe alkali eye burns in enucleated pig eyes, a delay in rinsing of just 20 seconds yielded an increase in intraocular pH from a minimum pH of 6.76±0.55 to a maximum pH of 10.32±0.33.²¹ A number of studies have looked for the best irrigation agent. One study has proposed that solutions containing buffering agents are ideal,²² and another study remarked that the solutions should not be phosphate-based, as this can lead to acute corneal calcification.²³ An iso-osmotic agent, such as normal saline or lactated Ringer's solution, is effective as well. However, immediate irrigation with tap water is preferred to waiting for the ideal fluid to be available.^{7,24} In most cases, the patient experiences severe reflexive blepharospasm causing disorientation and hindering him or her from reaching the nearest body or eye shower.⁶ Inability to overcome blepharospasm also leads to insufficient irrigation of all aspects of the eyes; therefore, the physician may need to apply topical anesthetic drops to facilitate the process. Chemical particles are sometimes retained in the cul-de-sac or

under the upper eyelid; therefore, ectropinization and cleaning of these areas with a moist sterile cotton swab are mandatory.²⁰ In addition to these conservative measures, active surgical debridement of necrotic conjunctival and corneal tissue is essential, since retained caustic agents may be the culprits causing continuing inflammation and necrosis.⁶

Severe chemical burns can be extremely painful. Topical anesthesia, such as proparacaine hydrochloride 0.5%, tetracaine hydrochloride 0.5% (Altacaine®, Altaire), or fluorescein sodium-benoxinate hydrochloride ophthalmic (Fluress®, Akorn) solutions, may help.^{7,17} Additionally, the physician may need to provide the patient with systemic analgesics, such as parenteral nonsteroidal anti-inflammatories or narcotic analgesics, for rapid relief and for ease of administration during irrigation.⁷ A cycloplegic agent is added to ease the pain of ciliary spasm and iritis and to prevent synechiae that may accompany the injury.^{7,25} Homatropine hydrobromide 5% (Isopto Homatropine 5%, Alcon Laboratories, Inc.) is used because it has a complete recovery time of about 36-48 hours, a time within which the patient should have a follow-up examination by an ophthalmologist; however, longer acting cycloplegics, such as scopolamine hydrobromide 0.25% (Isopto® Hyoscine, Alcon Laboratories, Inc.) or atropine sulfate, can also be used.¹⁷

In alkali burns, an immediate rise in intraocular pressure is due to contraction of the sclera and trabecular meshwork damage.^{7,26} A secondary pressure rise occurs two to four hours later due to the release of prostaglandins; thus, control of high intraocular pressure, such as with a carbonic anhydrase inhibitor or a beta-adrenergic blocker, is advocated as an initial therapy and during the later recovery phase. Immediate referral to an ophthalmologist or a glaucoma specialist is important in cases where intraocular pressure is severely high or is recalcitrant to medical management because a trabeculectomy or glaucoma Seton valve placement may be necessary.

In regard to the control of inflammation, topical corticosteroids, such as prednisolone acetate 1% or fluorometholone acetate 0.1% (Flarex 0.1%, Alcon Laboratories, Inc.), may be used for reducing inflammatory cell infiltration and stabilizing polymorphonuclear leukocyte, cytoplasmic, and lysosomal membranes^{3,17} after the corneal epithelium has healed. Some physicians may be reluctant to use corticosteroids due to their possible interference with stromal wound repair by impairing keratocyte migration into the area of injury and preventing collagen synthesis.³ However, this phase of repair usually peaks 10-14 days after the occurrence of the injury. Thus, the key to successful use of corticosteroids is to maximize their anti-inflammatory effect during the first 7-10 days; then tapering the dose and eventually discontinuing their use to prevent corneal thinning. Progestational steroids have less anti-inflammatory potency than corticosteroids but only exert minimal effect on stromal repair and collagen synthesis. For this reason, topical, subconjunctival, or systemic medroxyprogesterone acetate (Provera®, Pfizer) can be substituted for corticosteroids at 10-14 days when further suppression of inflammation is needed. Topical NSAIDs, such as diclofenac sodium (Voltaren Ophthalmic®

Figure 4: Medical and Surgical Management Modalities

Goals of Management	Medical Management	Surgical Management
Removal of the chemical agent	<ul style="list-style-type: none"> Irrigation solution (saline, lactated Ringer's, tap water) Open eyelids (\pm speculum) Sterile cotton swab 	<ul style="list-style-type: none"> Surgical debridement
Pain control	<ul style="list-style-type: none"> Topical anesthetic drops (proparacaine HCl 0.5%, tetracaine HCl 0.5%, benoxinate HCl) Systemic analgesics (NSAIDs, narcotics) Cycloplegic agents (homatropine HBr 5%, scopolamine HBr 0.25%, atropine sulfate) 	
Intraocular pressure control	<ul style="list-style-type: none"> Carbonic anhydrase inhibitor Betaadrenergic blocker 	<ul style="list-style-type: none"> Trabeculectomy Glaucoma Seton valve placement
Inflammation control	<ul style="list-style-type: none"> Topical corticosteroids (prednisolone acetate 1%, fluorometholone acetate 0.1%) Progestational steroids (topical, subconjunctival, systemic medroxyprogesterone) NSAIDs (diclofenac sodium, ketorolac tromethamine) Citrate 	
Infection prevention	<ul style="list-style-type: none"> Topical ophthalmic antibiotic drops/ointment (erythromycin, bacitracin, gentamicin, Neosporin) Tetanus immunization 	
Promote ocular epithelial healing	<ul style="list-style-type: none"> Artificial tear supplements Ascorbate Bandage soft contact lenses Temporary amniotic membrane patch 	<ul style="list-style-type: none"> Conjunctival/Tenon's advancement Limbal stem cell transplant Conjunctival/mucous membrane transplant Amniotic membrane transplant In vitro corneal stem cells transplant Symblepharon lysis (\pmlimbal transplantation) Keratoprosthesis Osteodontokeratoprosthesis

Figure adapted from references 3, 6, 7, 15, 17, 20-26, 28-32

0.1%, Novartis Pharmaceuticals) and ketorolac tromethamine (Acular® 0.5%, Allergan), have shown promising results in experimental trials to reduce ocular inflammation after chemical injuries. Finally, to further promote corneal wound healing and impair chemotaxis and functions of PMN leukocytes, a calcium-chelator, known as sodium citrate, can be taken orally as adjunctive therapy.

In the general initial management, patients who sustained burns to the eyelids, cornea, conjunctiva, sclera, and the skin peripheral to the affected eye should be given prophylactic, broad spectrum, topical ophthalmic antibiotic drops or ointment.^{15, 20} Patients with chemical particles embedded in the abrasions of the eyelids should have them removed.²⁸ Partial thickness lacerations of the eyelids not involving the lid margin may be surgically repaired in the same manner as other skin lacerations. However, full thickness lid lacerations involving the lid margin must be repaired meticulously to prevent marginal lid notching and trichiasis. Then the lid wounds may be irrigated with saline and covered with an antibiotic ointment and sterile dressing. Ointments are more soothing and persist on the cornea longer than ophthalmic drops. Erythromycin and bacitracin (Ak-Tra-

cin, Akorn) are preferred over gentamicin (Gentak®, Akorn), which may be toxic to corneal epithelium and Neosporin® Ophthalmic Solution (neomycin-polymyxin B-gramicidin, Monarch Pharmaceuticals Inc.), which has a relatively high allergic reaction rate.^{17,28} In regards to prophylactic immunization, Mukherjee et al. performed a literature search to answer whether tetanus prophylaxis is indicated after non-penetrating corneal abrasion.²⁹ Altogether, 30 relevant articles were found, but only one experimental animal study by Benson et al.³⁰ provided the best evidence to answer this clinical question. Benson et al. concluded that there is no reason clinically to provide tetanus prophylaxis in the emergency department following superficial corneal abrasion without evidence of perforation, infection, or devitalized tissue. However, Mukherjee et al. encourages tetanus prophylaxis whenever the opportunity arises.

Once the initial management has been performed, various measures can be taken to further aid and promote the epithelial healing process. In healthy individuals who sustained chemical injury to the eyes, the ability to produce copious lacrimation is intact; hence, tear substitution is usually not necessary.³ However, artificial tear supplements may be of value following

re-epithelialization to ameliorate the risk of recurrent corneal erosion and accelerate visual rehabilitation. Ascorbate, an essential water-soluble vitamin that plays a fundamental role in collagen polymerization, has been shown to reduce corneal thinning and ulceration following experimental alkali injuries. Placement of bandage soft contact lenses has been shown to promote epithelial migration, basement membrane regeneration, and epithelial-stromal adhesion by limiting the ocular surface from contacting the eyelids. Unfortunately, these lenses are poorly tolerated by the acutely injured eye. The use of amniotic membrane as a temporary patch or biological bandage for acute chemical burns provided more encouraging results overall.³¹ In Grades III and IV ocular chemical burns, various ocular surface transplantation techniques can be implemented to further promote re-epithelialization of the ocular surface.³ Examples of these surgical techniques include the conjunctival/Tenon's advancement (tenoplasty) procedure for Grade IV injuries, limbal stem cell transplantation for Grade III or IV, *in vitro* amplified corneal epithelial stem cells transplantation,²³ and conjunctival or mucous membrane transplantation. Clinically, transplantation of amniotic membrane as a permanent surgical graft has been shown to promote epithelialization and reduce inflammation, scarring, and neovascularization.³¹ In the late stage of ocular burns, severe scarring of both the palpebral and bulbar conjunctivae may occur, resulting in adhesions between the eyelids and the globe.³² This is known as symblepharon, which can be managed with repeated lysis of the conjunctivae with a glass rod. In extensive symblepharon that completely covers the entire corneal surface and destroys all limbal stem cells, the patient may subsequently undergo limbal transplantation. Secondary glaucoma and cataract may result; thus, keratoplasty and cataract surgery may be required to clear the visual axis,²³ and, as mentioned previously, the glaucoma could be managed both medically and surgically as indicated. Unfortunately, not all severely injured eyes are candidates for ocular surface reconstruction; therefore, keratoprosthesis and osteo-olontokeratoprosthesis remain to be reasonable options for these patients.

Prevention of Ocular Chemical Injury

Although health care professionals need to be prepared to treat ocular chemical burns, educating the public should be the first line of prevention. Since ocular chemical injuries most commonly occur in the agriculture and industrial workplace, precautionary measures, such as wearing eye protection, can prevent many of these work-related eye injuries from occurring.²⁸ Employers must provide their employees with adequate eye protection when working with hazardous materials or in hazardous situations as mandated by the Occupational Safety and Health Administration (OSHA). Safety goggles should have proper vents and side shields. OSHA Standard-29 CFR 1910.151(c) requires that suitable facilities for quick drenching or flushing of the eyes and body be provided within the work area for immediate use if an employee's eyes or body may be exposed to injurious corrosive materials.³³ Employees should receive safety training regarding the actions they must

take when these injuries occur, placing great emphasis on the importance of immediate copious eye irrigation for at least 15 minutes in duration.

Conclusion

Despite advances in medical and surgical treatment modalities, the consequences of severe ocular chemical burns can have profound psychological, economic, and social consequences for the patient. For this reason, a proactive approach to prevention becomes the most effective approach to prevent ocular chemical injuries from occurring. The principles of primary prevention include knowledge of risks via patient education and utilizing proper safety equipment and practices, all of which are the best measures to avoid the arduous therapeutic course for recovery of vision. For patients presenting with chemical ocular injuries, whether they occur in the workplace or at home, early recognition and prompt treatment by the treating physician remain the standards for maximal preservation of ocular tissue and provide hope for preservation of vision.

Acknowledgment

The authors would like to thank Reba Donahue for her editorial assistance.

Adeola Kosoko, BA, M3, is a student at the University of Kansas School of Medicine, Kansas City, KS.

Qui Vu, BS, M3, is a student at Creighton University School of Medicine, Omaha.

Omofolasade Kosoko-Lasaki, MD, MSPH, MBA, is Professor of Surgery (Ophthalmology), Creighton University School of Medicine, Omaha.

Potential Financial Conflicts of Interest: By AJCM® policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The authors have stated that no such relationships exist.

References

1. Kuckelkorn R, Kottek A, Schrage N & Reim M. Poor prognosis of severe chemical and thermal burns. The need for adequate emergency care and primary prevention. *Int Arch Occup Environ Health* 1995; 67:281-284.
2. Edwards RS. Ophthalmic emergencies in a district general hospital casualty department. *Br J Ophthalmol* 1987 Dec;71(12):938-42.
3. Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. *Surv Ophthalmol*. 1997; 41(4):275-313.
4. Hughes WF. Alkali burns of the cornea. I. Review of the literature and summary of present knowledge. *Arch Ophthalmol*. 1946;35:423-426.
5. Burns FR, Paterson CA. Prompt irrigation of chemical eye injuries may avert severe damage. *Occup Health Safety*. 1989; 58: 33-36.
6. Kuckelkorn R, Schrage N, Keller G, Redbrake C. Emergency treatment of chemical and thermal eye burns. *Acta Ophthalmol Scand*. 2002 Feb; 80(1): 4-10.
7. Spector J, Fernandez WG. Chemical, Thermal, and Biological Ocular Exposures. *Emergency Medicine Clinics of North America*. 2008 Feb;26(1):125-36.

8. Melsaether, CN, Rosenm, CL. "Burns, Ocular." *Emedicine: The Continually Updated Clinical Reference*. 1 Nov. 2007. 07 May 2009 <<http://emedicine.medscape.com/article/798696>>.
9. Socransky SJ. *Ocular burn management and eye irrigation*. In: Reichman, Eric, and Robert R. Simon. *Emergency Medicine Procedures*. New York: McGraw-Hill Professional, 2003.
10. Trudo, EW, Rimm, W. *Chemical injuries of the eye*. In: *Ophthalmic care of the combat casualty*. Falls Church, Va: Office of the Surgeon General, United States Army; Washington, D.C., Borden Institute, Walter Reed Army Medical Center, United States Army Medical Dept. Center and School, Uniformed Services University of the Health Sciences, 2003.
11. Kimi, T, Khosla-Gupta, BA. *Chemical and thermal injuries to the ocular surface*. In: Holland, EJ, Mannis. *Ocular Surface Disease Medical and Surgical Management*. New York: Springer, 2002.
12. Sharma, A, Smilkstein, MJ, Fraunfelder, FW. *Ophthalmic principles*. In: *Goldfrank's toxicologic emergencies*. New York: McGraw-Hill, 2006.
13. Macdonald EC, Cauchi P, Azuara Blanco A, Foot BG. Surveillance of severe chemical corneal injuries in the UK. *Br J Ophthalmol*. 2009 May 4.
14. Rowe JA, Kosoko-Lasaki O. Review of Ocular Trauma. *Archives of Ibadan Medicine*. 2006 Vol 7 (No. 1). 46-9.
15. Havens S, Kosoko-Lasaki O, Palmer M. Penetrating eye injury: a case study. *American Journal of Clinical Medicine*®. 2009; 6(1): 42-49.
16. Schirner G, Schrage NF, Salla S, Reim M, Burchard WG. Conjunctival tissue examination in severe eye burns: a study with scanning electron microscopy and energy-dispersive X-ray analysis. *Graefes Arch Clin Exp Ophthalmol*. 1995; 233: 251-256.
17. Flach AJ, Fraunfelder FW. Chapter 3: Ophthalmic Therapeutics. Riordan-Eva P, Whitcher JP: *Vaughan & Asbury's General Ophthalmology*, 17th Edition, The McGraw-Hill Companies, Inc. 2008.
18. Sun TT, Lavker RM. Corneal epithelial stem cells: past, present, and future. *J Invest Dermatol Symp Proc*. 2004 Sep; 9(3): 202-7.
19. Paterson CA, Pfister RR. Intraocular pressure changes after alkali burns. *Arch Ophthalmol*. 1974 Mar; 91(3): 211-8.
20. Tsai LM, Kamenetzky SA. Chapter 39: The Eye and Ocular Adnexa. Doherty GM, Way LW: *CURRENT Surgical Diagnosis and Treatment*, 12th Edition, 2006.
21. Rihawi S, Frenzt M, Becker J, Reim M, Schrage NF. The consequences of delayed intervention when treating chemical eye burns. *Graefes Arch Clin Exp Ophthalmol*. 2007 Oct; 245 (10): 1507-13.
22. Rihawi S, Frenzt M, Schrage NF. Emergency treatment of eye burns: which rinsing solution should we choose? *Graefes Arch Clin Exp Ophthalmol*. 2006 Jul; 244(7): 845-54.
23. Tuft SJ, Shortt AJ. Surgical rehabilitation following severe ocular burns. *Cambridge Ophthalmological Symposium*, *Eye* 2009: 1-6.
24. Ikeda N, Hayasaka S, Hayasaka Y, Watanabe K. Alkali burns of the eye: effect of immediate copious irrigation with tap water on their severity. *Ophthalmologica*. 2006; 220 (4): 225-8.
25. Braverman RS. Chapter 15: Eye. Hay WW, Jr, Levin MJ, Sondheimer JM, Deterding RR. *CURRENT Diagnosis and Treatment: Pediatrics*, 19th Edition. The McGraw-Hill Companies, Inc. 2009.
26. Augsburger J, Asbury T. Chapter 19: Ocular & Orbital Trauma. Riordan-Eva P, Whitcher JP: *Vaughan & Asbury's General Ophthalmology*, 17th Edition. The McGraw-Hill Companies, Inc. 2008.
27. Peate WF. Work-related eye injuries and illnesses. *Am Fam Physician*. 2007 Apr 1; 75 (7): 1017-22.
28. Augsburger J, Taylor A. Chapter 19: Ocular and Orbital Trauma. Riordan-Eva P, Whitcher JP: *Vaughan & Asbury's General Ophthalmology*, 17th Edition, The McGraw-Hill Companies, Inc. 2008.
29. Mukherjee P, Sivakumar A. Tetanus prophylaxis in superficial corneal abrasions. *Emerg Med J*. 2003 Jan; 20(1): 62-64.
30. Benson WH, Snyder IS, Granus V, Odom JV, Macsai MS. Tetanus prophylaxis following ocular injuries. *J Emerg Med*. 1993; 11: 677-83.
31. Kheirkhah A, Johnson DA, Paranjpe DR, Raju VK, Casas V, Tseng SCG. Temporary sutureless amniotic membrane patch for acute alkaline burns. *Arch Ophthalmol*. 2008; 126 (8): 1059-1066.
32. Shi W, Wang T, Gao H, Xie L. Management of severe ocular burns with symblepharon. *Graefes Arch Clin Exp Ophthalmol*. 2009 Jan; 247 (1): 101-6.
33. U.S. Department of Labor Occupational Safety and Health Administration. Title 29-Labor. Part 1910-Occupational safety and health standards. Sec 1910.151. Subpart K. Medical and First Aid. Revised 1998.