EMERGENCY TREATMENT & MANAGEMENT OF OCULAR BURNS IN TEHRAN CLINICS

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Abstract

Chemical or thermal eye burns account for a significant fraction of ocular trauma. The speed at which initial irrigation of the eye begins, has the most influence on the prognosis of eye burns. Water is mostly recommended as an irrigation fluid. However, water is hypotonic to the corneal stroma. The osmolarity gradient causes an increased water influx into the cornea and the invasion of the corrosive substance into deeper corneal structures. So we recommend higher osmolarities for the initial rinsing to mobilize and the dissolved corrosives out of the burnt tissue. General systems such as amphoteric solutions, which have an unclear binding with bases and acids, afford a convenient answer for emergency neutralisation. Both conventional anti-inflammatory therapy and early surgical intermediation are important to decrease the inflammatory response of the burnt tissue. In most severe eye burns, tenonplasty re-establishes the conjunctival apparent and limbal vascularity and avoids anterior section necrosis.

Introduction

Prompt appreciation and proper treatment of ocular emergencies are necessary in the main care setting when the result may depend on timely management. All ocular emergencies, including a penetrating globe injury, retinal detachment, central retinal artery blocking, acute angle-closure glaucoma, and chemical burns, should be stated immediately to the emergency department or an ophthalmologist. Careful eye checkup and simple tests can help primary care physicians make choices about appropriate treatment and appointment. All patients with eye problems should be tested for visual acuity and ocular movements. Opposition visual field examination, pupillary examination, and direct ophthalmoscopy of
both eyes also should be done. Ocular injury from high-velocity trauma or from chemicals may be easily misdiagnosed.

After a chemical burn, thorough eye washing for at least 30 minutes or until the pH of the eye is within physiologic range is serious to prevent extra damage. Use of an eye shield is required in patients with a ruptured globe to protect the injured eye and preserve the patient's vision. Eye injury, retinal detachment, and central retinal artery occlusion (CRAO) are between the most common ocular emergencies seen in the emergency department (1). It is expected that about 2 million eye injuries occur every year in the United States, a significant number of which lead to vision loss (2,3). Recent studies put the frequency of chemical and thermal injuries to the eye at 7.7%–18% of all ocular trauma (85; 56; 42; 44; 87). Most of these injuries are unimportant and do not cause any lasting lesions, others result in permanent unilateral or bilateral visual damage and a life of dependency (32).

The majority of sufferers are young and exposure happens at home, work and in association with criminal attacks (27; 51; 84). Alkali injuries occur more frequently than acid injuries (55; 50), eye burns caused by detergents and thermal agents being less frequent again (30). The most common agents causing alkali burns are ammonia (NH₃), potassium hydroxide (KOH), lye (NaOH) and lime (CaOH₂). Sulfurous (H₂SO₃), Sulfuric (H₂SO₄), hydrofluoric (HF) and hydrochloric (HCL) acids are the most common reasons of acid burns. Table 1 lists the data from 191 patients with 260 severely burnt eyes who were treated in some of Tehran eye clinics between 1995 and 2015. There was a high incidence of bilateral injuries and most of the injuries occurred at home or during leisure activities.

Table 1: Severe chemical and thermal eye burns in some of Tehran eye clinics (1995–2015): 191 patients (260 eyes).
<table>
<thead>
<tr>
<th></th>
<th>Number of patients</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilateral</td>
<td>69</td>
<td>36</td>
</tr>
<tr>
<td>Eyes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occupational injuries</td>
<td>177</td>
<td>68.1</td>
</tr>
<tr>
<td>Private injuries</td>
<td>63</td>
<td>24.1</td>
</tr>
<tr>
<td>Others</td>
<td>20</td>
<td>7.8</td>
</tr>
<tr>
<td>Eyes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alkalis</td>
<td>151</td>
<td>58.1</td>
</tr>
<tr>
<td>Acids</td>
<td>37</td>
<td>14.1</td>
</tr>
<tr>
<td>Thermal</td>
<td>42</td>
<td>16.2</td>
</tr>
<tr>
<td>Others</td>
<td>30</td>
<td>11.6</td>
</tr>
</tbody>
</table>


**Action of alkalis and acids**

The severity of ocular injury is associated to the kind of chemical, the volume and concentration (pH) of the resolution and the length of exposure (23). Alkalis enter more quickly than acids. The hydroxylon (OH) saponifies the fatty acid components of the cell membranes with consecutive cell disturbance and cell loss, while the cation is responsible for the diffusion process of the specific alkali (45). The diffusion rate increases from calcium hydroxide (slowest), potassium hydroxide (faster), sodium hydroxide (even faster) to ammonium hydroxide (fastest) (18). Depending on the grade of penetration, there is a injury of corneal and conjunctival epithelium, stromal keratocytes and endothelium. Hydration of
the glycosaminoglycans results in loss of clearness of the stroma (19). Destruction to the vascular endothelium of conjunctival and episcleral vessels clues to thrombosis of the episcleral vessels. The stronger the alkali, the faster its penetration. Permanent injury occurs at a pH above 11.5 (13). The pH in the aqueous humour rises within a few seconds of interaction with ammonium hydroxide (20). Intraocular structures such as the iris, lens and ciliary body are quickly impaired. Apart from hydrofluoric acid and, to a lesser extent, sulfuric acid, acids infiltrate the corneal stroma much less readily than alkalis (18). The hydrogen ion reasons damage due to pH alteration, while the anion produces protein precipitation and denaturation in the corneal epithelium and superficial stroma (14). Precipitation of the epithelial proteins proposes some guard to the corneal stroma and intraocular constructions. However, very strong acids penetrate just rapidly as alkalis. No statistical difference among strong alkali and acids burns was revealed in the clinical course and prognosis of such eyes (29).

Clinical classification of chemical and thermal burns

Eye burns are categorized in 4 grades (60; 62). The clinical course and ultimate forecast associates with the extent of limbal ischemia (23; 1; 72). The prognosis also depends on the extent of damage to conjunctival and episcleral tissue, severity of top burn and hurt to intraocular structures (Table 2).

Table 2: Clinical classification and prognosis of eye burns.

<table>
<thead>
<tr>
<th>Grade</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
<td>Erosio</td>
<td>Erosio</td>
<td>Erosio</td>
<td>Erosio</td>
</tr>
<tr>
<td>Limbal ischemia &gt; 3/4</td>
<td>Hyperemia</td>
<td>Limbal ischemia &gt; 1/3</td>
<td>Limbal ischemia &gt; ½</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chemosis</td>
<td>Chemosis</td>
</tr>
<tr>
<td>Grade</td>
<td>I</td>
<td>II</td>
<td>III</td>
<td>IV</td>
</tr>
<tr>
<td>-------</td>
<td>------------</td>
<td>-------------</td>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Opacification</td>
<td>Opacification</td>
<td>Recirculation</td>
<td>Vascularisation</td>
<td>Ulceration</td>
</tr>
<tr>
<td>Clinical outcome</td>
<td>Regeneration</td>
<td>Ulceration</td>
<td>Iris atrophy</td>
<td>Cataract</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Regeneration</td>
<td>Proliferation</td>
<td>Cataract</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cicatization</td>
<td>Glaucoma</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Complete restitution</td>
<td>Complete restitution</td>
<td>Scars</td>
<td></td>
</tr>
<tr>
<td>Prevention of the globe</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple operations for</td>
<td>Slight scars</td>
<td>Multiple operations</td>
<td>for limited visual</td>
<td></td>
</tr>
<tr>
<td>cosmetic rehabilitation</td>
<td></td>
<td></td>
<td>rehabilitation</td>
<td></td>
</tr>
</tbody>
</table>

Mild burns of grades I and II are related with hyperemia, small conjunctival ecchymosis and chemosis as fine as erosion of the corneal epithelium (Figs 1 and 2). In mild acid burns, the coagulated corneal epithelium often has a ‘ground-glass’ appearance. After removal of the epithelium, the clear corneal stroma is observable.

![Figure 1.](image1)


![Figure 2.](image2)
Grade II chemical injury: lime (CaOH). Central epithelial defect, partial limbal ischemia in the nasal inferior quadrant.

Grade III, and specially grade IV, burns are attended by wide and deep damage to the tissue (Figs 3 and 4). Typically, large areas of the conjunctival and subconjunctival tissue are involved. The visible blood vessels are thrombosed and appear dark. The corneal keratocytes are lost and hydration of the denatured proteins results in corneal opacification. Chemical injury to the iris and crystalline lens may produce mydriasis, a greyish advent of the iris and the fast development of a cataract. The lysis of cells of the anterior chamber rescinds the blood aqueous barrier and leads to iridocyclitis and fibrinous exsudation.

Figure 3.

Grade III chemical injury: sodium hydroxide (NaOH). Complete corneal and proximal conjunctival epithelial defect with loss of corneal stromal clarity. Limbal ischemia in the inferior quadrants.

Figure 4.

Grade IV chemical injury: sodium hydroxide (NaOH). Loss of corneal transparency, ectropion uveae and cataract formation, circular loss of conjunctival and episcleral tissue down to the fornices. The sclera is ischemic. Toxic materials such as superoxide radicals, prostaglandines, and presumably histamine, angiotensin, leukotriens and others are released from the burnt cells of the necrotic tissue (11; 37; 70). An inflammatory response is started, when they prolix into surviving tissues. In mild burns this reaction resolves rapidly, while in severe burns a severe and long-term inflammatory procedure is began, defining the clinical course of the burnt eyes (59-62; 75; 48).
Emergency treatment

Instant irrigation is of dominant importance after chemical or thermal burns (43; 7; 71). In most cases the wounded are disabled by severe refrectory blepharospasm with ensuing confusion. In this situation the victims are improbable to be accomplished of reaching the nearest body or eye shower and need champions who remove them from hazardous areas and apply fast and efficient help to their eyes and body (50). Effective first aid involves knowing how to overwhelmed blepharospasm by a passive opening of the lids and how to perform effective irrigation of the eye. All characteristics of the conjunctiva and cornea should be irrigated, and the patient should be asked to look in all directions (82). Topical anesthetic drops may be applied to decrease the pain and to facilitate irrigation. According to the American National Standards Institute (ANSI) standard (Z358.1–2015) severe eye burns have to be rinsed for 15 min. At least 500–1000 mL of irrigation fluid are thus essential. Amphoteric or buffered resolutions can normalize the pH of the anterior chamber within that time (76). Elements are sometimes trapped in the fornizes or under the upper lid. Therefore, ectropinisation and thorough cleaning of the cul-de-sac are obligatory after every burn. Materials containing calcium oxid (lime, cement dust) react avidly with water to produce a calcium hydroxide solution with a pH of 12.4 (48). A cotton-tipped applicator soaked in EDTA 1% (EDTA, di-sodium-ethylendiamintetra-acetat) can be used to facilitate cleaning of the cul-de-sac from calcium hydroxide (55). Instant irrigation is also imperative in thermal burns, because this cools the ocular surface (77). Continuous irrigation also removes inflammatory materials from the ocular surface (62; 66). First aid with rigorous irrigation immediately after the injury has a conclusive influence on the clinical course and prediction of such eyes (75; 4). Assessment between visual outcomes of better than 1/50 with that of less than 1/50 exposed a highly significant difference, with significantly better results after immediate irrigation. Visual acuity of > 1/50 enables the patient to move unaided. The number of operations and the length of stay on the ward are significantly reduced for eyes that received prompt irrigation (Table 3; 30).

Table 3: Value of immediate rinsing therapy versus clinical results in severe eye burns (101 patients, 131 eyes).

<table>
<thead>
<tr>
<th>Rinsing</th>
<th>Number of operations</th>
<th>Hospitalization (months)</th>
<th>Visual acuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate</td>
<td>6.5 ± 4.6</td>
<td>4.2 ± 2.8</td>
<td>45 (76%)</td>
</tr>
<tr>
<td>Delayed or no</td>
<td>10.4 ± 10</td>
<td>6.0 ± 4.5</td>
<td>22 (55%)</td>
</tr>
</tbody>
</table>

Unpaired t-test, P < 0.05.
Choice of irrigation fluid: Burns are attended by a loss of the corneal epithelium within a few seconds. The acutely burnt cornea takes up the burning material by osmolar forces resulting in a high osmolarity. One of the aims of rinsing therapy is to remove this chemical burden.

Water is commonly suggested as an irrigation fluid. It is available almost ubiquitously, and copious amounts of water have a dilutive effect. However, water is hypotonic to the corneal stroma and intraocular milieu. In measurements of osmolarity, Schrage et al. (unpublished) found the corneal stroma to have an osmolarity of 420 mOsm/L. The corneal tissue is diluted by rinsing with water and this is accompanied by an increased uptake of additional water and diffusion of the corrosive into the deeper layers of the cornea. We thus recommend the use of irrigation fluids with higher osmolarities for initial rinsing in order to avoid water invasion into the cornea and to empower the mobilisation of water and the melted corrosives out of the burnt tissue.

Normal saline (NS), which is often suggested as irrigation fluid, also has a lower osmolarity than tear fluid. It fails to normalize the pH of the anterior chamber even after lengthy irrigation (Table 4).

Table 4: PH on the corneal surface and in the anterior chamber after rinsing with different irrigation fluids
(experimental eye burn for 30 s/1n NaOH).

<table>
<thead>
<tr>
<th></th>
<th>pH corneal surface</th>
<th>anterior chamber</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Concentration of phosphat buffer:</strong> 440 mg sodium dihydrogenphosphat and 4040 mg sodium monohydrogenphosphat in 100 mL H₂O.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Directly after burn</td>
<td>13 ± 0</td>
<td>10 ± 0</td>
</tr>
<tr>
<td>5 minutes after rinsing</td>
<td>9 ± 0</td>
<td>10 ± 0</td>
</tr>
<tr>
<td>with 500 mL normal saline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 minutes after rinsing</td>
<td>7.5 ± 0</td>
<td>9.25 ± 0.44</td>
</tr>
<tr>
<td>with 500 mL phosphat buffer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 minutes after rinsing</td>
<td>7.5 ± 0</td>
<td>9.34 ± 0.59</td>
</tr>
<tr>
<td>with 500 mL diphtherine</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Phosphate buffer is frequently cited as an ultimate buffer solution (83; 40; 58; 73). For this reason, there is prevalent use of this buffer in many factories. However, in an experimental study complete calcification of the apparent stroma occurred in 100% of all animals after burning with 1 n NaOH for 30 s and instant rinsing with 500 mL phosphate buffer.
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We suggest that exogenously practical phosphate responds with endogenous calcium released from ruptured cells to produce calcium-phosphate complexes.

At present, there is constant investigational research to find irrigation fluids with an osmolarity similar to the corneal stroma. Currently available fluids which are appropriate for irrigation are sterile, lactated Ringer's (LR) and balanced saline solution (BSS; 21). Lactated Ringer is a buffered solution and may be more operative than normal saline. The osmolarity of BSS is similar to that of aqueous humour; its pH is neutral and it contains sodium acetate and citrate (46). According to Pfister, isotonic citrate buffer initiates chelation-complexes and binds unclear metal-ions resultant from the corrosive (57; 56). Balanced saline solution has an improved buffering capacity; it avoids the cornea from distension and preserves the corneal endothelium (47).

The pH, osmolarity and buffer capacity of the aqueous humour, corneal stroma and some irrigations fluids are listed in Table 5.

**Table 5: PH, osmolarity, constituents and buffer capacity of the aqueous humour, corneal stroma and different irrigation fluids.**

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>Osmolarity</th>
<th>Constituents</th>
<th>Buffer Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aqueous humour</td>
<td>7.4</td>
<td>304</td>
<td>Na, K, Cl, Ca, PO₄, proteins</td>
<td>0.0008</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Na, K, Cl, Ca, S, SO₄</td>
<td></td>
</tr>
<tr>
<td>Stroma</td>
<td>7.4</td>
<td>420</td>
<td>PO₄, proteins, lipids, glycosaminoglycans</td>
<td>0.0004</td>
</tr>
<tr>
<td>Normal saline</td>
<td>7.0</td>
<td>290</td>
<td>Na, Cl</td>
<td>0.0002</td>
</tr>
<tr>
<td>Phosphate buffer</td>
<td>7.4</td>
<td>260</td>
<td>Na, K, PO₄</td>
<td>0.00625</td>
</tr>
<tr>
<td>Lactated Ringer's</td>
<td>5.0–7.5</td>
<td>280–309</td>
<td>Na, K, Ca, Cl, lactat</td>
<td>0.00069</td>
</tr>
<tr>
<td>BSS</td>
<td>7.2</td>
<td>310</td>
<td>Na, K, Ca, Cl, citrat, acetat</td>
<td>0.001</td>
</tr>
<tr>
<td>Diphoterine</td>
<td>7.4</td>
<td>820</td>
<td>Diphoterine, Na, Cl, glycine 0.02</td>
<td></td>
</tr>
</tbody>
</table>

A new amphoteric resolution which is appropriate for irrigation is Diphoterine (Previn®, Fa. Prevor). This newly synthetized fluid is talented to bind both alkalis and acids. 0.4% Diphoterine has a pH of 7.4 and an osmolarity of 820 mosml/L. The pH in the conjunctival sac and in the corneal stroma is condensed as quickly as after irrigation with phosphate buffer. The ingredients of Diphoterine are listed in Table 6.
Table 6: Constituents of diphoterine.

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amphoteric</td>
<td>3.8/100 mL</td>
</tr>
<tr>
<td>NaCl</td>
<td>1.8/100 mL</td>
</tr>
<tr>
<td>Glycin</td>
<td>0.75/100 mL</td>
</tr>
<tr>
<td>Preservative</td>
<td>0.05/100 mL</td>
</tr>
<tr>
<td>Aqua destillata</td>
<td>ad 100 mL</td>
</tr>
</tbody>
</table>

**Transport problems**

As strong acids and alkalis enter within seconds or minutes and endure for hours (19), irrigation should not be intermittent during transference to a specialized eye-care unit. The recommendations for minimum irrigation times range from 15 min (ANSI standard; 43) to 2–4 h (55; 74). We applaud the use of an intravenous infusion set to supply at least 500–1000 mL of irrigation fluid. As defined above, one of the rescuers should hold the eyelids open though a second salvation flushes the eye with a mild, directable and controllable stream of fluid. Local anesthetic drops should be administered recurrently if necessary to release the patient from pain and to simplify irrigation.

Some authors favor the use of particularly intended irrigation structures (52; 16; 78; 81; 49; 39). While these structures provide constant irrigation of the eye, they fail to flush the ocular superficially unvaryingly and appropriately, particularly the cul-de-sac. A promote risk with lime or cement burns is that elements retained under the eye lids are not identified and detached once the loop or lens has been applied. Furthermore, salvationsunaware with the handling of these systems will lose priceless time when connecting the slings or lenses, which may cause additional harm to the eye.

The usefulness of rinsing therapy can be measured by using general indicator paper to define the pH of the external eye. Irrigation must be continued as long as the pH remains outside the normal range. If extended irrigation does not achieve normalization of the pH, one must deliberate the opportunity that there are still elements in the superior or inferior cul-de-sac.

**Subsequent care**

The subsequent care of eye burns is reliant on the severity of the injury. Additional therapeutical events are applied rendering to the extent of the injury. If the injury is mild (grades I and II) and irrigation started instantly, most eyes will rectify without permanent damage within a few days (48; 50; 2; 32). Current steroid/antibiotic drops and ointment plus padding may serve for the treatment of these mild burns. Follow-up treatment within 24 h is obligatory.
Severe ocular eye burns (grades III and IV) are hard to treat and the course of healing frequently takes several months. In these cases, precise organization with respect to the extent of (limbal) ischemia and penetration of tissue devastation is crucial. An investigation with the operating microscope is thus obligatory. Parabulbar or general anesthesia are occasionally needed if the patient suffers pain and local anesthetic drops are not enough.

Less severe eye burns (grade III) are considered by superficial ischemia of the conjunctival tissue. In these cases where regular anterior chamber constructions are well-preserved and there is no hurt to the iris, ectropium uveae or fibrinous exsudation, following management takes the form of a more traditional therapy. Admission to and treatment in a local eye clinic are thus adequate (66). Most severe eye burns (grade IV) lead to important limbal ischemia and necrosis of the bulbar and tarsal conjunctiva as fine as of the episcleral tissue down to the fornices. In cases with shallow necrosis, the deep episcleral vessels are still perfused while necrosis of the deeper episcleral tissue is related with thrombosis of the episcleral vessels. In these severe cases, opacification of the cornea is common and the anterior chamber constructions are covered. A greyish feature of the iris, ectropium uveae and the breakdown of the blood aqueous barricade with fibrinous exsudation into the anterior chamber authorize the devastation of the deep anterior piece. The lids and facets of the tarsal conjunctiva are regularly intricate. Numerous problems arise in the acute phase of the burn. The most delicate problem is averting the eye from early melting. Necrosis of the conjunctiva and subconjunctival tissue is attended by a significant exsudation of leucozytes (PMN's). These leucozytes release large amounts of lysosomal enzymes. The matrix metalloproteinases (MMP), collagenase (MMP-1 and MMP-8; 25; 22; 53; 26; 38; 12), gelatinase (MMP-2, MMP-9) (8; 12; 24), and stromelysin (MMP-3; 8; 6) in specific are accountable for the splitting of the collagen molecules and the growth of corneoscleral and corneal ulceration, characteristically 4–6 weeks after the accident.

The basic attitude in the treatment of these eyes is to decrease the inflammatory answer caused by the necrotic tissue. The traditional support of therapy is the primary and rigorous application of corticosteroids (10; 41; 68; 28; 60; 62). Additionally, local antibiotics are necessary to prevent microbiobal infections until the ocular surface has reepithelialized (15; 31; 2). Tetracycline derivates play an important role because they have been shown to inhibit metallo-proteinases (3; 17; 79; 5; 54) independently of their anti-microbial properties. Besides conventional therapy, active surgical interference with the débridement of necrotic conjunctival and subconjunctival tissue is essential in order to eliminate a nidus of continued inflammation from reserved caustic resources, although also any accumulation of PMN and to avoid the
continued release of their destructive enzymes. Special reconstructive methods such as tenonplasty allow the denuded avascular sclera to be enclosed with vital connective tissue prepared from the equator of the globe (69; 64-69; 30). The main benefit of this tissue is that it enables the rebuilding of the conjunctival matrix and of limbal vascularity. These interferences avoid anterior section necrosis and/or sterile ulceration and the eye is conserved. It is advisable to admit these cases to an eye clinic particular in the treatment of these eyes and familiar with specialized processes in plastic reconstruction.

References:


