Emergency treatment of chemical and thermal eye burns

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ABSTRACT.

Chemical and thermal eye burns account for a small but significant fraction of ocular trauma. The speed at which initial irrigation of the eye begins, has the greatest influence on the prognosis and outcome of eye burns. Water is commonly 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. We therefore recommend higher osmolalities for the initial rinsing to mobilize water and the dissolved corrosives out of the burnt tissue. Universal systems such as amphoteric solutions, which have an unspecific binding with bases and acids, provide a convenient solution for emergency neutralisation. Both conservative anti-inflammatory therapy and early surgical intervention are important to reduce the inflammatory response of the burnt tissue. In most severe eye burns, tenoplasty re-establishes the conjunctival surface and limbal vascularity and prevents anterior segment necrosis.

Key words: cornea - emergency treatment - eye burns - irrigation fluid - reconstructive surgery.

Recent studies put the incidence of chemical and thermal injuries to the eye at 7.7%−18% of all ocular trauma (Watz & Reim 1973; Pflister et al. 1984; Liggert 1989; MacEwen 1989; Zapf & Baum 1993). Most of these injuries are trivial and do not cause any lasting lesions, others result in permanent unilateral or bilateral visual impairment and a life of dependency (Kuckelkorn et al. 1993). The majority of victims are young and exposure occurs at home, work and in association with criminal assaults (Keeney 1974; Morris et al. 1987; Thiel et al. 1989). Alkali injuries occur more frequently than acid injuries (Pflister 1983; Morgan 1987); eye burns caused by detergents and thermal agents being less frequent again (Kuckelkorn et al. 1995). The most common agents causing alkali burns are ammonium (NH₄), lye (NaOH), potassium hydroxide (KOH) and lime (Ca(OH)₂). Sulfuric (H₂SO₄), sulfuric (H₂SO₄), hydrochloric (HCl) and hydrochloric (HCl) acids are the most common causes of acid burns. Table I lists the data from 191 patients with 269 severely burnt eyes who were treated in the eye center of the RWTH Aachen between 1980 and 1995. There was a high incidence of bilateral injuries and most of the injuries occurred at home or during leisure activities.

Action of alkalis and acids

The severity of ocular injury is related to the type of chemical, the volume and concentration (pH) of the solution and the duration of exposure (Hughes 1946). Alkalis penetrate more rapidly than acids. The hydroxyl (OH) ions sapontize the fatty acid components of the cell membranes with consecutive cell disruption and cell death, while the cation is responsible for the penetration process of the specific alkali (McCulley 1987). The penetration rate increases from calcium hydroxide (slowest), potassium hydroxide (fastest) to ammonium hydroxide (fastest; Grant 1974). Depending on the degree of penetration, there is a loss of corneal and conjunctival epithelium, stromal keratocytes and endothelium. Hydration of the glycosaminoglycans results in loss of clarity of the stroma (Grant & Kern 1955). Damage to the vascular endothelium of conjunctival and episcleral vessels leads to thrombosis of the episcleral vessels.

The stronger the alkali, the faster its penetration. Irreversible damage occurs at a pH above 11.5 (Friedewald et al. 1944). The pH in the aqueous humour rises within a few seconds of contact with ammonium hydroxide (Graupner & Haustmann 1970). Intracocular structures such as the iris, lens and ciliary body are rapidly damaged.

Table I. Severe chemical and thermal eye burns in the Department of Ophthalmology of the RWTH Aachen (1985–1995): 191 patients (269 eyes).

<table>
<thead>
<tr>
<th>Type of Injury</th>
<th>Number of Patients</th>
<th>Percent of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral</td>
<td>122</td>
<td>64</td>
</tr>
<tr>
<td>Bilateral</td>
<td>69</td>
<td>36</td>
</tr>
<tr>
<td>Occupational injuries</td>
<td>177</td>
<td>88.1</td>
</tr>
<tr>
<td>Private injuries</td>
<td>63</td>
<td>24.8</td>
</tr>
<tr>
<td>Others</td>
<td>20</td>
<td>7.8</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>39</td>
<td>11.6</td>
</tr>
</tbody>
</table>
Apart from hydrofluoric acid and, to a lesser extent, sulfuric acid, acids penetrate the corneal stroma much less readily than alkalis (Grant 1974). The hydrogen ion causes damage due to pH alteration, while the anion produces protein precipitation and denaturation in the corneal epithelium and superficial stroma (Friedenwald et al. 1946). Precipitation of the epithelial proteins offers some protection to the corneal stroma and intraocular structures. However, very strong acids penetrate just as quickly as alkalis. No statistical difference between strong alkali and acids burns was discovered in the clinical course and prognosis of such eyes (Kuckelkorn 1996).

**Clinical classification of chemical and thermal burns**

Eye burns are classified in 4 grades (Reim 1987, 1990). The clinical course and ultimate prognosis correlates with the extent of limbal ischemia (Hughes 1946; Bullen 1963; Roper-Hall 1965). The prognosis also depends on the extent of damage to conjunctival and episcleral tissue, severity of lid burn and damage to intraocular structures (Table 2).

Mild burns of grades I and II are associated with hyperemia, small conjunctival edema and chemosis as well 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 visible.

Grade III, and especially grade IV, burns are accompanied by extensive 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 grayish appearance of the iris and the fast development of a cataract. The lysis of cells of the anterior chamber destroys the blood aqueous barrier and leads to tridacylitis and fibrinous exudation.

Toxic substances such as prostaglandins, superoxide radicals, and presumably histamine, angiotensin, leukotrienes and others are released from the burnt cells of the necrotic tissue (Eakin & Bhattacharjee 1977; Kulka & Srinivasan 1993; Rochels et al. 1982). An inflammatory response is initiated, when they diffuse into surviving tissues. In mild burns this reaction resolves quickly, while in severe burns a severe and long-term inflammatory process is initiated, determining the clinical course of the burnt eyes (Reim 1982, 1987, 1992; Williams et al. 1983; Strack et al. 1991; Reim & Lechner 1993; Reim et al. 1993).

**Emergency treatment**

Immediate irrigation is of paramount importance after chemical or thermal burns (Lubeck & Greene 1988; Cohen & Hyndiuk 1978; Rodeheaver et al. 1982). In most cases the victims are disabled by severe reflexory blepharospasm with ensuing disorientation. In this situation the victims are unlikely to be capable of reaching the nearest body or eye shower and need res-

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![Fig. 1. Grade I chemical injury, hydrochloric acid (HCl). Burn of the cornea only. Coagulated corneal epithelium with 'ground glass' appearance. Partial removal of the epithelium, clear corneal stroma.](image)

**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>Erosion</td>
<td>Limbal ischemia &gt; 1/4</td>
<td>Limbal ischemia &gt; 1/2</td>
<td>Erosion</td>
</tr>
<tr>
<td>Limbal ischemia &gt; 1/4</td>
<td>Hyperemia</td>
<td>Chemosis</td>
<td>Chemosis</td>
<td>Chemosis</td>
</tr>
<tr>
<td>Opacification</td>
<td>Opacification</td>
<td>Recirculation</td>
<td>Vasculatisation</td>
<td>Extensive necrosis</td>
</tr>
<tr>
<td>Clinical outcome</td>
<td>Regeneration</td>
<td>Regeneration</td>
<td>Ulceration</td>
<td>Ulceration</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Complete restitution</td>
<td>Complete restitution</td>
<td>Proliferation</td>
<td>Iris atrophy</td>
</tr>
<tr>
<td>Preservation of the globe</td>
<td>Slight scars</td>
<td>Multiple operations</td>
<td>Cleartration</td>
<td>Cataract</td>
</tr>
<tr>
<td>Multiple operations for cosmetic rehabilitation</td>
<td></td>
<td></td>
<td></td>
<td>Glaucoma</td>
</tr>
</tbody>
</table>
The text describes a clinical scenario involving chemical injuries to the eye, specifically focusing on the management of injuries caused by substances such as lime and sodium hydroxide. The text highlights the importance of prompt irrigation with an appropriate fluid to prevent further tissue damage and emphasizes the role of topical anesthesia and the need for early surgical intervention in severe cases.

Key points from the text:
- Chemical injuries to the eye, particularly with substances like lime and sodium hydroxide, require immediate irrigation to prevent further damage.
- Topical anesthesia is often necessary to reduce pain during irrigation and facilitate the procedure.
- Surgical intervention may be required for severe injuries, especially if there is significant tissue damage.
- The use of irrigation fluids with specific osmolality can help manage the chemical burden and prevent further tissue necrosis.

A significant portion of the text is dedicated to the choice of irrigation fluid, discussing the importance of selecting fluids with appropriate osmolality to mitigate the effects of chemical injuries on the corneal stroma and intraocular milieu.

For a comprehensive understanding, the reader is encouraged to refer to the referenced literature for detailed guidelines on managing chemical injuries and selecting appropriate irrigation fluids. The text concludes with a reminder that the use of irrigation fluids with higher osmolality may be necessary in severe cases to effectively manage the chemical load on the ocular structures.
ties for initial rinsing in order to prevent water influx into the cornea and to enable the mobilization of water and the dissolved corrosives out of the burnt tissue.

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

Phosphate buffer is often cited as an ideal buffer solution (Thiel 1965; Laux et al. 1975; Poser 1983; Roth 1993). For this reason, there is widespread use of this buffer in many factories. However, in an experimental study complete calcification of the superficial stroma occurred in 100% of all animals after burning with 1 n NaOH for 30s and immediate rinsing with 500 mL phosphate buffer. (Schrage et al., unpublished). We suggest that exogenously applied phosphate reacts with endogenous calcium released from ruptured cells to produce calcium-phosphate complexes.

At present, there is ongoing experimental research to find irrigation fluids with an osmolality similar to the corneal stroma. Currently available fluids which are suitable for irrigation are sterile, lactated Ringer's (LR) and balanced saline solution (BSS; Herr et al. 1991). Lactated Ringer is a buffered solution and may be more effective than normal saline. The osmolality of BSS is similar to that of aqueous humour; its pH is neutral and it contains sodium acetate and citrate (McDermott et al. 1988). According to Pfister, isotonic citrate buffer initiates chelat-complexes and binds unspecific metal-ions derived from the corrosive (Pfister et al. 1981; Pfister et al. 1984). Balanced saline solution has an enhanced buffering capacity; it prevents the cornea from swelling and preserves the corneal endothelium (McNamar et al. 1987).

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

A new amphoteric solution which is suitable for irrigation is Diphotolrine (Previn®, Fa. Prevar). This newly synthesized fluid is able to bind both alcali and acids. 0.4% Diphotolrine has a pH of 7.4 and an osmolality of 820 mosm/L. The pH in the conjunctival sac and in the corneal stroma is reduced as rapidly as after irrigation with phosphate buffer. The constituents of Diphotolrine are listed in Table 6.

**Transport problems**

As strong acids and alkalis penetrate within seconds or minutes and remain for hours (Grant & Kern 1955), irrigation should not be interrupted during transport to a professional eye-care unit. The recommendations for minimum irrigation times range from 15 min (ANSI standard; Lubrecht & Greene 1988) to 2-4

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### 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>6.3 ± 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

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### Table 4. pH on the corneal surface and in the anterior chamber after rinsing with different irrigation fluids (experimental eye burn for 30s in NaOH).

<table>
<thead>
<tr>
<th></th>
<th>pH corneal surface</th>
<th>pH anterior chamber</th>
</tr>
</thead>
<tbody>
<tr>
<td>Directly after burn</td>
<td>13 ± 0</td>
<td>10 ± 0</td>
</tr>
<tr>
<td>5 minutes after rinsing with 500 mL normal saline</td>
<td>9 ± 0</td>
<td>10 ± 0</td>
</tr>
<tr>
<td>5 minutes after rinsing with 500 mL phosphate buffer</td>
<td>7.5 ± 0</td>
<td>9.25 ± 0.44</td>
</tr>
<tr>
<td>5 minutes after rinsing with 500 mL diphotolrine</td>
<td>7.5 ± 0</td>
<td>9.34 ± 0.59</td>
</tr>
</tbody>
</table>

Concentration of phosphate buffer: 440 mg sodium dihydrogenophosphate and 4640 mg sodium monohydrogenophosphate in 100 mL H₂O.
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>Stroma</td>
<td>7.4</td>
<td>429</td>
<td>Na, K, Cl, Ca, S, SO₄, 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>269</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>Diphosphate</td>
<td>7.4</td>
<td>820</td>
<td>Diphosphate, Na, Cl, glycin</td>
<td>0.02</td>
</tr>
</tbody>
</table>

h (Pistet 1983; Saari et al. 1984). We recommend the use of an intravenous infusion set to supply at least 500–1000 mL of irrigation fluid. As described above, one of the rescuers should hold the eyelids open while a second rescuer flushes the eye with a mild, directable and controllable stream of fluid. Local anesthetic drops should be administered repeatedly if necessary to relieve the patient from pain and to facilitate irrigation.

Some authors favor the use of specially designed irrigation systems (Naumann 1964; Girard & Super 1966; Schulze & Tost 1967; Tan 1970; Morgan 1971; Lau 1979). Whereas these systems provide continuous irrigation of the eye, they fail to flush the ocular surface homogeneously and appropriately, especially the cul-de-sac. A further risk with lime or cement burns is that particles retained under the eye lids are not detected and removed once the loop or lens is removed. Moreover, rescuers unfamiliar with the handling of these systems will lose precious time when inserting the slings or lenses, which may cause additional damage to the eye.

The effectiveness of rinsing therapy can be assessed by using universal indicator paper to determine the pH of the external eye. Irrigation must be continued as long as the pH remains outside the normal range. If prolonged irrigation does not achieve normalization of the pH, one must consider the possibility that there are still particles in the superior or inferior cul-de-sac.

Subsequent care

The subsequent care of eye burns is dependent on the severity of the injury. Further therapeutic procedures are applied according to the extent of the damage. If the injury is mild (grades I and II) and began immediately, most eyes will heal without permanent damage within a few days (Moon & Robertson 1983; Morgan 1987; Beare 1990; Kuckelkorn et al. 1993). Topical steroids/antibiotic drops and ointment plus padding may suffice for the treatment of these mild burns. Follow-up treatment within 24 h is mandatory.

Severe ocular eye burns (grades III and IV) are difficult to treat and the course of healing often takes several months. In these cases, accurate classification with regard to the extent of (limbal) ischemia and depth of tissue destruction is essential. An examination with the operating microscope is thus mandatory. Parabulbar or general anesthesia are sometimes needed if the patient suffers pain and local anaesthetic drops are not sufficient.

Less severe eye burns (grade III) are characterized by superficial ischemia of the conjunctival tissue. In these cases where regular anterior chamber structures are preserved and there is no damage to the iris, ectropion uvea or fibrous exudation, subsequent management takes the form of a more conservative therapy. Admission to and treatment in a local eye clinic are thus sufficient (Reim & Kuckelkorn 1995).

Most severe eye burns (grade IV) lead to significant limbal ischemia and necrosis of the bulbar and tarsal conjunctiva as well as of the epithelial tissue down to the fornix. In cases with superficial necrosis, the deep episcleral vessels are still perfused while necrosis of the deeper episcleral tissue is associated with thrombosis of the episcleral vessels. In these severe cases, opacification of the cornea is common and the anterior chamber structures are obscured. A greyish aspect of the iris, ectropion uvea and the breakdown of the blood aqueous barrier with fibrinous exudation into the anterior chamber confirm the destruction of the deep anterior segment. The lids and aspects of the tarsal conjunctiva are often involved. Many problems arise in the acute phase of the burn. The most delicate problem is preventing the eye from early melting.

Necrosis of the conjunctiva and subconjunctival tissue is accompanied by a considerable exudation of leukocytes (PMN's). These leukocytes release large amounts of lysosomal enzymes. The matrix metalloproteinases (MMP), collagenase (MMP-1) and MMP-8; Itoi et al. 1990; Honk et al. 1971; Newsome & Gross 1977; Johnson-Muller & Gross 1978; Kuter et al. 1989; Fini & Girard 1990), gelatinase (MMP-2, MMP-9) (Collier et al. 1988; Fini & Girard 1990; Huhtala et al. 1990), and stromelysin (MMP-3; Collier et al. 1988; Chin et al. 1995) in particular are responsible for the splitting of the collagen molecules and the development of corneoscleral and corneal ulceration, characterized typically 4-6 weeks after the accident.

The basic principle in the treatment of these eyes is to reduce the inflammatory response caused by the necrotic tissue. The traditional mainstay of therapy is the early and intensive application of corticosteroids (Donshik et al. 1978; Leibowitz 1980; Reim & Schmidt-Martens 1982; Kenyon 1985; Reim 1987). Additionally, local antibiotics are necessary to prevent microbial infections until the ocular surface has repithelialized (Girard et al. 1970; Kuckelkorn et al. 1987; Beare 1990). Tetracycline derivatives play an important role because they have been shown to inhibit metallo-proteinases (Brion et al. 1985; Goltup et al. 1987; Seedorf et al. 1987; Burns et al. 1989; Perry et al. 1993) independently of their anti-microbial properties.

Besides conservative therapy, active surgical intervention with the debridement of necrotic conjunctival and subconjunctival tissue is necessary in order to remove a nidus of continued inflammation from retained caustic materials.
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