Vol 19 | No 1 • Summer 2024
SA Ophthalmology Journal

An overview of acute management in chemical eye injuries

E Jansen MBChB, *Registrar, Division of Ophthalmology, Neuro-Sciences Department, University of Witwatersrand, Johannesburg, South Africa.*
ORCID: https://orcid.org/0009-0006-5455-5863

A Moodley MBBCH, FC Ophth(SA), MEd(Ophth); *Consultant ophthalmologist, Department of Neurosciences, Division of Ophthalmology, University of the Witwatersrand, Johannesburg, South Africa.*
ORCID: https://orcid.org/0000-0003-2306-654X

Corresponding author: Dr E Jansen, Email: steljansen@gmail.com

Abstract

Chemical eye injuries are true ophthalmic emergencies and require immediate intervention. Causative agents can be classified as alkalis, acids, and irritants. Alkali burns are more common and can cause significant tissue damage due to deep tissue penetration. Grading of ocular injuries is critical for determining acute treatment and visual prognosis. A thorough assessment of all the ocular structures assists with comprehensive and individualised treatment. Immediate management starts with copious irrigation, aimed at diluting and removing the inciting agent. Thereafter, the main goals of treating chemical eye injuries are to minimise inflammation, promote re-epithelialization, support stromal collagen synthesis, and inhibit collagen breakdown. Acute surgical management includes amniotic membrane transplant and tuboplasty. Poor immediate management results in more challenging treatment of acute disease, while poorly controlled acute disease results in more treatment-resistant chronic ocular disease. Early and appropriate intervention is fundamental to a good visual outcome in chemical eye injuries.

Keywords: PubMed was searched using the keywords chemical eye injury, chemical eye injuries and ocular chemical burns.

Disclosure: No financial interest to declare

Introduction

Chemical eye injuries are true ophthalmic emergencies and require immediate intervention. Early recognition and treatment play a big role in the final visual outcome. The socio-economic and educational status of countries, along with the rate of protective eye equipment, correlates with the frequency and severity of these types of injuries.[1] Two population groups most commonly affected by chemical eye injuries include young males working in factories and construction industries, as well as children between the ages of 1-2 years old who are affected by domestic eye injuries.[2] The global incidence of acid attacks has increased in past years, which often results in more severe burns.[1,3,4] As these injuries occur in younger age groups, it is essential to manage burn patients well to reduce ocular morbidity and long-term healthcare costs.[5]

Etiology

Causative agents can be classified as alkalis, acids, and irritants.[6]

Alkaline mediums have a pH of greater than 7. It causes ocular damage by the saponification of fatty acids in cell

Table I. Roper Hall classification of chemical injuries[3]			
Grade	Cornea	Limbus	Prognosis
I	Corneal epithelial damage	No limbal ischaemia	Good
II	Corneal haze, iris details visible	<1/3 limbal ischaemia	Good
III	Stroma; haze, iris details obscured	1/3-1/2 limbal ischaemia	Guarded
IV	Opaque corneal, iris and pupil obscured	>1/2 limbal ischaemia	Poor

membranes and liquefactive necrosis.[4] Hydrolysis of proteins leads to extensive damage due to deep tissue penetration. An exception is lime, which results in the formation of lime salts after the dissolution of the cell membrane, which prevents further penetration. It may, however, act as a reservoir with prolonged exposure if lodged in the conjunctiva.[5]

Alkaline burns are more common due to their widespread use in industrial and domestic cleaning products.[7] This could explain why ammonia is a commonly used agent in assaults.

In contrast, acidic mediums have a pH of less than 7. Upon contact with the eye, coagulative necrosis takes place, which creates a barrier to deeper tissue penetration.

Alcohols result in de-epithelialization of the ocular surface. During the COVID-19 pandemic, chemical eye injuries due to alcohol-based hand sanitiser increased significantly.[8] The recovery process is usually rapid and the visual prognosis is excellent.[6]

Ultimately, damage to the eye occurs due to a rapid change in pH. The degree of damage correlates with the degree of pH change.[4] Eslani *et al.* report that other factors impact the severity of chemical tissue damage, which includes the temperature of the causative agent, the impact force, the concentration, as well as the duration of contact.[2]

Clinical assessment

It is important to note that many causative agents release toxic vapours upon contact and that the patient may have multi-organ involvement. The most fatal is laryngeal oedema with loss of a patent airway. If the vital signs are compromised, seek immediate help.

A thorough assessment of all the ocular structures assists with comprehensive and individualised treatment.

The eyelid should be inspected for skin and lash burns, distortion of lid margins, and abnormal lid movements. This will affect tear distribution and contribute to ocular surface pathology.

The ocular surface may be difficult to assess due to tight lids and chemosis. A Desmarre's retractor can assist with double eversion of the lid to aid the view. Limbal ischaemia will present as pale or blanched limbal areas with an epithelial defect. Accurate assessment of the proportion of surviving limbal tissue is important as it impacts corneal healing and ultimately the visual prognosis. Corneal involvement is recorded as the degree of haze and transparency. The sensation can be assessed once the topical anaesthetic has worn off or before instilling topical anaesthesia.

Anterior segment involvement may be in the form of iris changes – colour, hyperaemia, haemorrhage, and necrosis should be noted.[4] Both anterior and posterior synechiae can rapidly develop. Pupillary responses may be abnormal.

Lastly, measure the intraocular pressure. This can be either low, normal, or elevated. Due to eyelid involvement, corneal oedema, and an irregular corneal surface, Dua *et al.* claim that digital palpation may be the only accurate way of assessing intraocular pressure.[6]

Classification

Various classification systems are available, but the two most used systems are the Roper-Hall classification and the Dua classification. The Roper Hall classification is easy to apply and commonly used. A drawback of this classification is that it does not include conjunctival involvement, which is an important component in the prognostication of corneal melting and symblepharon formation.[3]

Dua *et al.* introduced a new system, which includes the percentage of conjunctival damage, as well as the degree of limbal damage – which can be assessed by fluorescein staining.[6] The proportion of surviving limbal tissue carries major prognostic value.[9] Therefore, authors agree that the Dua classification system is superior to the Roper Hall classification in predicting the visual outcome, especially in severe ocular burns.[6,10,11]

Table II. Dua classification of chemical injuries[3]				
Grade	Limbal involvement (clock hours)	Conjunctival involvement (%)	Analogue scale	Prognosis
I	0	0	0/0%	Very good
II	≤3	≤30	0.1-3/1-30%	Good
III	>3-6	>30-50	3.1-6/30.1-50%	Good
IV	>6-9	>50-75	6.1-9/51-75%	Good – Guarded
V	>9 to <12	>75 to <100	9.1-11.9/75.1-99.9%	Guarded – Poor
IV	12 (total limbus)	100 (total conjunctiva)	12/100%	Very poor

Stages of ocular surface recovery as described by McCulley[12]

Acute (Day 0-7)

During the first week of recovery, epithelial regrowth begins, provided there are adequate healthy limbal stem cells.[5] Treatment aims to support epithelial regrowth and decrease factors that delay this process, such as inflammation and preservatives in topical treatment.

Early reparative (Day 7-21)

If the epithelial defect is small, complete re-epithelialization is seen during the second phase of healing. More severe defects will show little re-epithelialization, while eyes with grade IV burns will still appear ischaemic. Surface inflammation will be ongoing while there is an epithelial defect. Keratocytes continue to synthesise collagen to repair the damaged stroma, while on the other hand, collagen production is inhibited by collagenase, which is released by inflammatory cells. The aim is therefore to maximise collagen production while minimising collagenase activity.

Late reparative (>D21)

Inflammation begins to subside and mild eye injuries show full resolution at this stage, while persistent epithelial defects, scarring, and infection become problematic in more severe injuries.[3] Clinical features of limbal stem cell deficiency also become evident three weeks after the injury.

Acute management

Immediate intervention

Regardless of the causative agent,

irrigation remains the first and most important intervention – aimed at diluting and removing the inciting agent.[13] The pH should be measured in both eyes before commencing irrigation, which should continue for at least 30 minutes and at least one litre of fluid should be used.[6] The pH measurements should be repeated every 30 minutes until a normal pH is obtained. This can either be done with litmus paper or a urine dipstick. Once the pH is within the normal range between 7 to 8, a second pH test should be performed after five minutes to ensure that the pH remains unchanged. If not, irrigation should be resumed.

The ideal fluid is isotonic, such as Balanced Saline Solution or Ringer's lactate. There is some concern that a hypotonic solution, such as water, can worsen corneal oedema, but there is not enough evidence to substantiate this.[3]

Recently, amphoteric irrigation fluids have been proposed as the preferred solution. This solution is hypertonic, and the amphoteric properties enable it to neutralise both acid and base ions. It rapidly neutralises the ocular surface pH, which limits tissue necrosis. In comparison to water, Solim *et al.* report that up to 17 times less volume is required to achieve a neutral pH.[14] Furthermore, it exerts a minimal exothermic reaction.[6] Currently there is a lack of large studies and no definitive recommendations can be made regarding the use of amphoteric irrigation fluids in chemical eye injuries.[9]

However, authors emphasise that the lack of the ideal solution should not delay immediate irrigation, as there is a correlation between time to irrigation and visual outcome.[2,9,10,13]

Irrigation will be easier to administer if the patient receives topical anaesthetic drops, as the patient will be more comfortable and able to keep their eyes open.

Crystals and embedded debris should be removed with forceps or cotton buds, as these objects continue to damage ocular structures while in situ.

Aims of management

The main goals of treating chemical eye injuries are to minimise inflammation, promote re-epithelialization, support stromal collagen synthesis, and inhibit collagen breakdown.

Decrease inflammation

Corticosteroids

Corticosteroids act by reducing inflammatory cell infiltrate, while at the same time stabilising the cell membrane of polymorphonucleocytes. Steroids should be used intensively for the first 10 days but should be rapidly tapered after this to avoid corneal melting.[10,13] The increased risk of corneal melting has been attributed to coagulative necrosis caused by ischaemia.[13] Dexamethasone 0.1% or Prednisolone acetate 1% are common choices. Dosing depends on the severity of the injury, ranging from hourly to six hourly instillations of treatment.

Progestational steroids are less potent in terms of their anti-inflammatory effects, but they also have a smaller negative impact on stromal repair and collagen synthesis.[10] Medroxyprogesterone can be administered parenterally or a topical 1% solution can be instilled six hourly.[4]

Non-steroidal anti-inflammatory drugs (NSAIDs)

There is no role for NSAIDs in the treatment of chemical eye injuries. Hossain claims that it can worsen the melting process.[15]

Promote re-epithelialization

Lubricants

Artificial tears are the mainstay of treatment in chemical eye injuries to promote corneal re-epithelialization.[10] Preservative-free drops are preferred, with hourly instillation.

Bandage contact lenses

Silicone hydrogel contact lenses can be used to protect a compromised ocular surface and promote epithelialization. Large-diameter gas-permeable scleral contact lenses are an alternative option in patients with severe pain and photophobia. It provides a hydrating reservoir of fluid between the contact lens and the ocular surface, furthermore, it protects the epithelium against friction caused by blinking.[3] The Prosthetic Replacement of Ocular Surface Ecosystem (PROSE) has also been successfully used in multiple studies.[9]

Other modalities

Fibronectin, epidermal growth factor, retinoic acid, N-acetylcysteine, and sodium hyaluronate are still in the experimental phase of development and more human studies are needed to determine their efficacy.[10]

Biological fluids

This includes autologous serum, umbilical cord serum, amniotic membrane suspension, and autologous platelet-rich plasma.[10] These agents have been used to promote re-epithelialization and accelerate wound healing. It has been used in a variety of ocular surface disorders, such as dry eyes, persistent epithelial defects, and neurotrophic ulcers, to name a few.

Biological fluids contain various components, such as growth factors, that are not found in standard medical treatments. The composition is similar to natural tears and these fluids also have the advantage of being preservative-free.[10] Barriers to use include a complex manufacturing process, cold storage, and risk of contamination and infection.[4]

Tarsorrhaphy

A tarsorrhaphy decreases the risk of exposure, as well as blink-related microtrauma. It can be considered if the tarsal conjunctiva is vascularised with intact epithelium.[13]

Promote corneal stromal healing

Ascorbate (vitamin C) supplements

Chemical eye injuries result in a decreased ascorbate concentration in the aqueous humour. Ascorbic acid levels are about 15 times higher in aqueous, compared to the ascorbic acid levels in plasma – this suggests a possible role in ocular protection.[6] Sharma *et al.* report that ascorbate levels are about a third of normal values following chemical eye injuries.[10] Topical or systemic ascorbate is needed to reverse this deficiency.

A low level of anterior chamber ascorbate can result in corneal ulceration with subsequent perforation, as ascorbate is a cofactor in the rate-limiting step of collagen synthesis.[3,5]

Authors are not in agreement as to which method of administration is best. Some authors argue that topical administration is more effective, as anterior segment penetration is ineffective with systemic administration.[10] Others suggest that compliance is poor with topical treatment, due to pain on instillation of drops[3], while Dua *et al.* state that intravenous ascorbate is more effective than oral administration.[6] The recommended oral dose ranges from 500mg to 2000mg daily, while the topical 10% ascorbic acid can be given six hourly.[4,12,16]

However, all authors agree that ascorbate is an essential component in

management. Numerous studies have shown that the risk of corneal melt secondary to topical corticosteroids is minimal if used in combination with ascorbate.

Minimise ulceration

Tetracyclines

Tetracyclines inhibit collagenase, which decreases the risk of corneal ulceration. Increased tetracycline concentration in ocular tissues has been linked to decreased incidence of corneal ulceration.[10] Both topical and systemic tetracycline have beneficial effects and the tetracycline of choice is Doxycycline, dosed at 100mg twice daily.

Citrate

Sodium citrate has been shown to significantly decrease the development of corneal ulcers, especially following alkali burns. According to Sharma *et al*., the combination of citrate and ascorbate is superior to either supplement alone.[10] The recommended dose is 10% topical citrate drops every two hours.

Adjuvant therapy

Topical antibiotics are indicated in the first stages of healing, especially if there is an epithelial defect. It is used to prevent secondary infection and to reduce the load of commensal flora. Trimethoprim/ Polymyxin B or Fluoroquinolone drops can be used six hourly; Erythromycin ointment is an alternative option. In patients with large epithelial defects, Ciprofloxacin should be used with caution as it can precipitate in the cornea.[16]

Topical cycloplegics can be used for the treatment of ciliary spasms and the prevention of posterior synechiae formation. Atropine drops twice daily are recommended. Phenylephrine should be avoided as the vasoconstriction properties can aggravate ischaemia.[16]

Oral analgesia is indicated.

IOP control

IOP-lowering treatment is often necessary. Oral acetazolamide is the preferred treatment.[4,6] Agents that utilise outflow tracts may not be effective, as the trabecular meshwork and uveoscleral pathway are distorted by the chemical injury. Episcleral vasculopathy further impairs aqueous outflow.[3] Steroid response should also be considered as a cause. As the etiology of glaucoma is multifactorial, standard topical treatments often fail. Tube surgery is preferred, but remains challenging due to conjunctival cicatrisation, and frequent revisions are often necessary to maintain the target IOP.[3]

Debridement

Debridement should be done as early as possible. Necrotic tissue serves as a source of inflammation, which furthermore inhibits re-epithelialization.[5]

Acute surgical management

Surgical options include amniotic membrane transplant (AMT) and tenoplasty. AMT provides symptomatic relief, promotes re-epithelialization, and reduces inflammation scarring, inflammation, and neovascularization.[9,10] AMT can be used as a graft and as a patch.[9] Tenoplasty is a procedure where the Tenon's capsule is bluntly separated from the globe and the Tenon flap is then advanced to the limbus and sutured to the ischaemic sclera. It is the procedure of choice in eyes with extensive ischaemia as it has the potential of decreasing long-term complication risk, by re-establishing limbal blood supply.[4,13]

Long term complications

This includes dry eye disease, conjunctival adhesions, symblepharon, non-healing epithelial defects (neurotrophic keratopathy), limbal stem cell deficiency with secondary corneal sc≤≤arring, decompensation, thinning, melting, and perforation.

Conclusion

Successful management is often compromised by a lack of follow-up and poor treatment compliance. This has been attributed to a poor understanding of treatment benefits, lack of social support, psychosocial difficulties, polypharmacy, and pain with the instillation of treatment.[17] Good communication between the patient, the family, and the doctor is therefore essential to ensure a good outcome.

Early and appropriate intervention is fundamental to a good visual outcome in chemical eye injuries.

References

1. Akgun Z, Selver OB. Epidemiology and etiology of chemical ocular injury: A brief review. *World J Clin Cases*. 2023;11(6):1245-1251.
2. Eslani M, Baradaran-Rafii A, Movahedan A, Djalilian AR. The Ocular Surface Chemical Burns. *J Ophthalmol*. 2014;2014:196827.
3. Bizrah M, Yusuf A, Ahmad S. An update on chemical eye burns. *Eye*. 2019;33(9):1362-1377.
4. Lorenzana-Blanco N, Santander-García D, Güell J, Alejandre-Alba N. Acute management of ocular chemical burns: A review. *J EuCornea*. 2023;11(3).
5. Colby, K. Chemical Injuries of the Cornea. *Focal Points Am Acad Ophthalmol*. 2010;XXVIII(28):1-14.
6. Dua HS, Ting DSJ, Al Saadi A, Said DG. Chemical eye injury: pathophysiology, assessment and management. *Eye*. 2020;34(11):2001-2019.
7. Seraj H, Khawandanh S, Fatani A, Saeed A, Alotaibi G, Basheikh A. Population-level investigation of the knowledge of ocular chemical injuries and proper immediate action. *BMC Res Notes*. 2020;13:103.
8. Akbas E, Korkmaz I, Palamar M, Barut Selver O. Shifting trends in demographic features of chemical eye injuries during COVID-19 pandemic. *Int Ophthalmol*. 2022;42(7):2127-2132.
9. Baradaran-Rafii A, Eslani M, Haq Z, Shirzadeh E, Huvard MJ, Djalilian AR. Current and Upcoming Therapies for Ocular Surface Chemical Injuries. *Ocul Surf*. 2017;15(1):48-64.
10. Sharma N, Kaur M, Agarwal T, Sangwan VS, Vajpayee RB. Treatment of acute ocular chemical burns. *Surv Ophthalmol*. 2018;63(2):214-235.
11. Gupta N, Kalaivani M, Tandon R. Comparison of prognostic value of Roper Hall and Dua classification systems in acute ocular burns. *Br J Ophthalmol*. 2011;95(2):194-198.
12. Singh P, Tyagi M, Kumar Y, Gupta KK, Sharma PD. Ocular chemical injuries and their management. *Oman J Ophthalmol*. 2013;6(2):83-86.
13. Iyer G, Srinivasan B, Agarwal S. Algorithmic approach to management of acute ocular chemical injuries-I's and E's of Management. *Ocul Surf*. 2019;17(2):179-185.
14. Nahaboo Solim MA, Lupion-Duran TM, Rana-Rahman R, Patel T, Ah-Kine D, Ting DSJ. Clinical outcomes and safety of Diphoterine® irrigation for chemical eye injury: A single-centre experience in the United Kingdom. *Ther Adv Ophthalmol*. 2021;13:25158414211030428.
15. Hossain P. The corneal melting point. *Eye*. 2012;26:1029-1030.
16. Bagheri N, Wajda B, Calvo C, Durrani A, eds. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*. Seventh edition. Lippincott Williams and Wilkins; 2016.
17. Bizrah M, Yusuf A, Ahmad S. Adherence to Treatment and Follow-Up in Patients with Severe Chemical Eye Burns. *Ophthalmol Ther*. 2019;8(2):251-259.