Pacific Journal of Medical and Health Sciences
ISSN: 2456-7450 | Vol.4, No.3, 2022, pp-40-44

PJMHS
PACIFIC JOURNAL OF MEDICAL AND HEALTH SCIENCES

Review

The Ocular Surface Chemical Injuries: A Review

Sagar Dhillon[1], Rajendra Choudhary[2]

[1]PG Resident, [2]Assistant Professor

Department of Ophthalmology, Pacific Medical College and Hospital, Udaipur. Rajasthan, India

*Corresponding author Email: drsagardhillon@gmail.com

ABSTRACT

Chemical burns are serious ocular emergencies that require early evaluation and immediate management since they can result in blindness. The majority of casualties are children, and these injuries take place at home, at work, while playing and can also occur as intentional crimes. Injuries from alkali happen more common and severe than those from acids. Chemical eye injuries result in severe damage to the cornea, anterior segment, limbal stem cells, and ocular surface epithelium, which can cause impaired vision partially or permanently in one or both eyes. Therefore, emergency management is the most crucial element in selecting the visual outcome.

This paper discusses epidemiology, etiopathophysiology and mainstay of emergency response and available methods of management for enhancing patient's prognosis after presentation with ocular chemical injuries.

KEYWORDS: Ocular burn, Ocular chemical injury, Alkali injury, Acid injury.

INTRODUCTION

Ocular chemical injuries are a serious ocular emergency that calls for quick, thorough assessment and management. Delays in treatment may lead the chemical agent to penetrate the tissues more deeply, causing more extensive damage. Permanent blindness, limbal stem cell insufficiency, and secondary glaucoma can occur as long-term effects of chemical ocular burns[1].

The after-effects of a chemical trauma to the eye can be quite serious and difficult to treat. The knowledge of the pathophysiology of chemical damage as well as advanced oculoplasty has given promising results in management of such injuries.

The treatment for chemical injuries aims to revert the corneal transparency and ocular surface to normal. If there is severe corneal scarring, amniotic membrane transplantation, limbal stem cell grafting, keratoplasty and keratoprosthesis may be required to restore the eyesight.

EPIDEMIOLOGY

According to recent studies, ocular burns account for 7.7–18% of all ocular injuries. The majority of victims are children, and exposure happens at home, at work, and in connection with violent crimes. Injury from alkali is more common than injury from acid[2-5].

Ocular chemical injuries can happen in a variety of situations and settings, including the home, the construction sites, schools and industries, accidental or intentional crimes. These accidents frequently occur among children, laborers and construction workers, as well as in machine industries, agricultural settings, and industrial chemical laboratories. Additionally, they are regularly reported from cleaning and sanitizing teams, vehicle repair shops, and fabric mills. Ages 20 to 40 account for the majority of cases of chemical burns to the eyes, morbidity being higher in males than females. While children are more prone to suffer from exposure to domestic chemicals including topical personal agents[6] and laundry detergent capsules[7], males make up more than 81% of workplace-related eye injuries.

A retrospective research was conducted on the incidence and prevalence of ocular chemical burns, and 171 patients were followed up continuously over the course of a year. 61% of these burns were caused by industrial incidents, while 37% happened at home. The remainder had an unidentified origin[8].

Sulfuric acid is the most often used chemical agent in purposeful chemical assault attacks, and non-accidental injuries from these attacks has been documented in several nations[9,10]. Rare cases of chemical ocular burns have also been linked to chemical face peeling treatments as 35% trichloroacetic acid[11].

PATHOGENESIS

Alkaline agents penetrate deeper in the tissues than acids. Cellular disruption is brought on by the hydroxyl ion's saponification of fatty acids in cell membranes[12]. Alkaline solutions quickly penetrate into the underlying tissues once the epithelium is damaged, damaging the collagen matrix and proteoglycan ground material. If the substance penetrates the trabecular meshwork's collagen fibrils, it may result in scarring that prevents aqueous outflow and causes secondary glaucoma. Strong alkaline substances enter the anterior chamber and significantly inflame the uvea (ciliary body, iris) and the lens[12,13].

In comparison, acids have the ability to trigger necrosis and denature proteins, creating a barrier that can limit further tissue entry[13-15].

It should be emphasized that acidic compounds are still highly capable of severely damaging the ocular surface, even if they cannot penetrate as rapidly and readily as alkaline agents.

CLASSIFICATION OF INJURIES

Predicting the fate of a chemical eye burn is made much easier by knowing its stage. Most notably, it has been demonstrated that a significant prognostic component is the proportional proportion of intact limbal tissue[16,17].

The Roper-Hall classification was devised in the middle of the 1960s, first by Ballenand later refined by Roper-Hall[18]. It is based on quantity of perilimbal ischemia and the degree of corneal haze (Table 2). Following this, Pfister published a categorization system based on images showing corneal haze and perilimbal ischemia that rated the damage from mild to severe to extremely severe[2].

Table 1: Typical etiological agents that can cause ocular chemical damage

CHEMICALS	EXAMPLE
1. Sulfuric acid	Acid in batteries, industrial cleansers
2. Acetic acid	Vinegar
3. Hydrochloric acid	Laboratories chemicals
4. Sulfurous acid	Bleaching, Refrigerant, vegetable preservative
5. Hydro fluoric acid	Glass polishers, silicone production
6. Ammonia	Fertilizers, refrigerants, Drain cleaners
7. Potassium hydroxide	Caustic potash
8. Magnesium hydroxide	Sparklers, incendiary devices
9. Lime	Plastic, whitewash, cement, mortar

Table 2: Roper-Hall Grading for the severity of burns to the ocular surface

GRADING	CLINICAL RESULTS		PROGNOSIS
	Corneal Manifestation	Conjunctial/ Limbal Manifestation	
I	Corneal epithelial damage	No limbal ischemia	Good
II	Corneal haze, iris details visible	<1/3 limbal ischemia	Good
III	Total epithelial loss, stromal haze, and iris details obscured	1/3–1/2 limbal ischemia	Guarded
IV	Corneal opacity, iris and pupil obscured	>1/2 limbal ischemia	Poor

CLINICAL COURSE

There are four phases to the clinical course of ocular chemical injury: immediate, acute, early reparative (8–20 days), and late reparative[19].

Immediate phase:

The minute a chemical agent comes into contact with the surface of the eye, the immediate phase begins[1,19]. The total area of the corneal epithelial defect, the area of the conjunctival epithelial defect, degree of limbal blanching, area and density of corneal opacification, evidence of increased intraocular pressure at presentation, and loss of lens clarity are the key factors for determining the severity of chemical ocular injury and prognosis.

Acute phase:

The acute phase of healing lasts the first seven days following chemical eye damage. There occurs re-establishing the superficial layer of cornea i.e. epithelium. The anterior chamber and ocular surface both experience significant inflammatory reactions[1,19].

Early Reparative phase (8-20 days of injury):

In this phase, persistent inflammatory response, stromal repair, and scarring take their place[1,19]. During this phase, a chronic epithelial defect may cause corneal ulcers. Collagenase, metalloproteinase, and other proteases secreted by polymorphonuclear leukocytes and the healing epithelium have all been implicated in its cause[20].

Late Reparative phase (after 3 weeks of injury):

Complications in patients with guarded visual prognosis and completion of healing with favourable visual prognosis characterise this stage. Products of the injured ocular tissue's disintegration that function as new antigens and infiltrate leukocytes and macrophages frequently cause a persistent, severe inflammatory response. In extreme situations, the retina, peripheral vitreous, and eyelids may all be affected[21].

MANAGEMENT

Immediate:

Ocular chemical injuries, in almost all the cases, presents as emergency. The chemical exposure should be diagnosed if at all feasible once a history of exposure is taken, but this should not delay treatment.

Before the ocular assessment, urgent therapy involves extensive irrigation with isotonic saline or lactate ringer solution and occasionally irrigation quantities of up to 20 L or more are needed (pH testing should be done). Following the irrigation and pH neutralisation, the detailed ocular examination should be done, including assessment of fornices, visual acuity, IOP, and depth of penetration of the chemical.

In case of pediatric patient, general anaesthesia is advised when examination under topical anaesthesia is not possible.

Acute phase treatment:

Promoting reepithelialization, reducing inflammation, avoiding infection, stopping additional epithelial and stromal breakdown, and minimising the squeal are the primary goals of acute phase therapy.

Lubricating ointment and tear substitutes without preservatives can lessen the severity of chronic epitheliopathy and speed up the recovery of vision. In situations when the healing of the epithelium is delayed, bandage contacts can also be used[13].

Anti-inflammatory therapy:

After chemical injuries, topical corticosteroids are essential for managing initial inflammation. They reduce inflammatory cells infiltration and prevent neutrophilic membrane damage[22].

Treatment of raised Intraocular pressure:

As previously indicated, trabecular meshwork damage from alkali can result in high intraocular pressure. Therefore, oral aqueous suppressive agents are often recommended over topical treatments to minimize toxicity and therefore epithelial re-growth[23].

SEQUALE PREVENTION

Everyday inspection should be done for the development of symblepharon. The fornices can also be fitted with a symblepharon ring for prevention. The greatest size, which effectively separates the palpebral conjunctiva from the bulbar conjunctiva, is preferred[13].

The significant risk arises when intact epithelium is not achieved by the 3rd week. Surgical techniques are the mainstay of treatment at this stage of ocular injury, along with ongoing medical care. Such techniques include tenonplasty, tissue adhesives, amniotic membrane grafting, limbal stem cell transplantation, therapeutic penetrating keratoplasty and kerato-prosthesis[24].

CONCLUSION

Patients with chemical ocular injuries require a thorough assessment at the initial presentation, as well as acute care. The advancement with understanding the pathophysiological mechanisms, and improvement in the treatment modalities like use of topical ascorbate and citrate, as well as surgical treatments like amniotic membrane transplantation, stem cell transplantation, penetrating keratoplasty, and ultimately the placement of a keratoprosthesis if necessary, have all made possible in restoring the maximum visual outcome.

The mainstay goal of management is to restore the corneal transparency, restore the normal ocular surface anatomy, lid position, control of glaucoma and ultimately to preserve the maximum vision for the patients.

CONFLICTS OF INTEREST: None

FINANCIAL SUPPORT: None

REFERENCES

1. P. Singh, M. Tyagi, Y. Kumar et al. Ocular chemical injuries and their management. *Oman J Ophthalmol,* 2013;6(2): 83-86
2. Pfister RR. Chemical injuries of the eye. Ophthalmology. 1983; 90(10):1246-53.
3. Liggett PE, Pince KJ, Barlow W, Ragen M, Ryan SJ. Ocular trauma in an urban population: review of 1132 cases. Ophthalmology. 1990; 97(5):581-4.
4. Macewen CJ. Eye injuries: a prospective survey of 5671 cases. Br J Ophthalmol. 1989; 73(11):888-94. DOI:10.1136/bjo.73.11.888.
5. Zagelbaum BM, Tostanoski JR, Kerner DJ, Hersh PS. Urban eye trauma: a one-year prospective study. Ophthalmology. 1993; 100(6):851-6.
6. Haring RS, Sheffield ID, Channa R, Canner JK, Schneider EB. Epidemiologic trends of chemical ocular burns in the United States. JAMA ophthalmology. 2016; 134(10):1119-24.
7. Breazzano MP, Day Jr HR, Tanaka S, Tran U. Prospective analysis of paediatric ocular chemical burns: laundry detergent pods. J AAPOS. 2018; 22(6):426-8.
8. Kuckelkorn R, Makropoulos W, Kottek A, Reim M. Retrospective study of severe alkali burns of the eyes. Klin. Monatsblatter fur Augenheilkd. 1993; 203(6):397-402.
9. Morgan SJ. Chemical burns of the eye: causes and management. Br J Ophthalmol. 1987; 71(11):854-7.
10. Yeong EK, Chen MT, Mann R, Lin TW, Engrav LH. Facial mutilation after an assault with chemicals: 15 cases and literature review. J Burn Care Res. 1997; 18(3):234-7.
11. Fung JF, Sengelmann RD, Kenneally CZ. Chemical injury to the eye from trichloroacetic acid. Dermatologic surgery. 2002; 28(7):609-10.
12. M. D. Wagoner. Chemical injuries of the eye: current concepts in pathophysiology and therapy. Surv. Ophthalmol, , 1997;41(4):275-313
13. Lin A, Patel N, Yoo D, DeMartelaere S, et.al. Management of ocular conditions in the burn unit: thermal and chemical burns and Stevens-Johnson syndrome/toxic epidermal necrolysis. J Burn Care Res. 2011; 32(5):547-60.
14. Spector J, Fernandez WG. Chemical, thermal, and biological ocular exposures. Emerg. Med. Clin. N. Am. 2008; 26(1):125-36.
15. Salzman M, O'Malley RN. Updates on the evaluation and management of caustic exposures. Emerg. Med. Clin. N. Am. 2007; 25(2):459-76.
16. Fish R, Davidson RS. Management of ocular thermal and chemical injuries, including amniotic membrane therapy. Curr Opin Ophthalmol. 2010; 21(4):317-21.
17. Schrage NF, Langefeld S, Zschocke J, et.al. Eye burns: an emergency and continuing problem. Burns. 2000; 26(8):689-99.
18. Roper-Hall MJ. Thermal and chemical burns. Trans Ophthalmol Soc UK. 1965; 85: 631-53.
19. McCulley JP, Smolin G, Thoft RA. The cornea: scientific foundation and clinical practice.
20. Ralph RA. Tetracyclines and the treatment of corneal stromal ulceration: a review. Cornea. 2000; 19 (3):274-7.
21. Kuckelkorn R, Kottek A, Reim M. Intraocular complications following severe eye burns-frequency and management. Klin. Monatsblatter fur Augenheilkd. 1994; 205(2):86-92.

22. Saud EE, MoraesJr HV, Marculino LG, et.al. Clinical and histopathological outcomes of subconjunctival triamcinolone injection for the treatment of acute ocular alkali burn in rabbits. Cornea. 2012; 31(2):181-7.

23. Tsai JH, Derby E, Holland EJ, Khatana AK. Incidence and prevalence of glaucoma in severe ocular surface disease. Cornea. 2006; 25(5):530-2.

24. Dohlman CH, Schneider HA, Doane MG. Prosthokeratoplasty. Am J Ophthalmol 1974; 77: 694-70?