

# EARLY AND INTENSIVE MANAGEMENT OF CHEMICAL EYE INJURIES: CASE SERIES

## ABSTRACT:

Chemical injuries of the eyes are true ophthalmic emergencies causing significant visual morbidities. They require urgent and immediate assessment and intervention. They are more common in young adults either due to accidental causes or due to criminal assaults. They severely damage the ocular structures; surface epithelium, cornea, conjunctiva, limbal stem cells and can cause permanent unilateral or bilateral vision loss. In this article, we are trying to emphasize on the importance of initiating treatment in the early phase of ocular chemical injury and also emphasizing on extent of chemical ocular injuries, their severity and their varied outcomes through two different types of cases; one with good visual recovery with early initiation of treatment and the other developing complications in spite of being initiating treatment in early phase.

Keywords- chemical ocular burn, severe damage, initiating treatment, varied outcomes

## INTRODUCTION:

“Ocular chemical injuries are a true ocular emergency and require immediate and intensive evaluation and treatment. The sequelae of an ocular burn can be severe and particularly challenging to manage”[1,7]. “Ocular chemical injuries can occur under diverse circumstances and in such varied locations as the home, the workplace, and school”[1,8]. “In a retrospective study done by R.Kuckelkorn et.al they found inage analysis that the greatest at-risk population were the 20-40year-old patients. 73.8% were industrial accidents, 30% happened to builders and labourers, 20% in the chemical industry and 20% in machine factories. At home most of the injuries were caused by lime and drain cleaners. Sodium and potassium hydroxide produced more extended and deeper damages than lime due to their rapid penetration through the ocular tissues”[2].

“Recent studies put the incidence of ocular burns of the eye at 7.7-18% of all ocular traumas. The majorly of victims are young and exposure occurs at home, work and in association with criminal assaults. Alkali injuries occur more frequently than acidic injuries”[1,7]

## PATHOPHYSIOLOGY

Acid burns- Acids have lower than normal pH values of the human eye (7.4) they precipitate tissue protein, creating a barrier to further ocular penetration. Due to this fact acid injuries tend to be less severe than alkali injuries. One exception to this is hydrofluoric acid, which may rapidly pass through cell membranes and enter anterior chamber of the eye.

“It reacts with collagen resulting in shortening of collagen fibres which cause a rapid increase in intraocular pressure (IOP). After severe acid burns with ciliary body damage, decrease in levels of aqueous ascorbate has been demonstrated”[1]

Alkali burns

“Alkali burns cause corneal damage by pH change, ulceration, proteolyzes and collagen synthesis defects. Alkali substances are lipophilic and penetrate the eye more rapidly than acids. The basic substance can quickly deposit within the tissues of the ocular surface causing saponification reaction within those cells. The damaged tissue secrete proteolytic enzymes as part of an inflammatory response which leads to further damage. Alkali substances can penetrate into the anterior chamber causing cataract formation, damage to the ciliary body and damage to the trabecular meshwork”[1,10].

There is acute rise in intraocular pressure in early phase to shrinkage of cornea and sclera[6].

**CLASSIFICATION:**

Initial classification was given by Ballen[3] and then modified by Roper-Hall[4].

Table 1.ROPER-HALL CLASSIFICATION[4]

<b>GRADE</b>	<b>CORNEA</b>	<b>LIMBUS</b>	<b>PROGNOSIS</b>
<b>1</b>	Corneal epithelial damage	No limbal ischemia	Good
<b>2</b>	Corneal haze, iris details visible	<1/3 <sup>rd</sup> limbal ischemia	Good
<b>3</b>	Stromal haze, iris details obscured	1/3-1/2 limbal ischemia	Guarded
<b>4</b>	Opaque cornea, iris and pupil obscured	>1/2 limbal ischemia	Poor

Dua et.al later gave recent classification based on limbal clock hour involvement and percentage of bulbar conjunctiva involved[5].

Table 2.DUA CLASSIFICATION[5]

<b>GRADE</b>	<b>PROGNOSIS</b>	<b>CLOCK HOURS OF LIMBAL INVOLVEMENT</b>	<b>CONJUNCTIVAL INVOLVEMENT</b>	<b>ANALOGUE SCALE</b>
<b>I.</b>	Very good	0	0%	0/0%
<b>II.</b>	Good	<=3	<30%	0.1-3/1-30%
<b>III.</b>	Good	3-6	30-50%	3-6/30-50%
<b>IV.</b>	Good to guarded	7-9	51-75%	6.1-9/51-75%
<b>V.</b>	Guarded to poor	9-11	76-99%	9.1-11/76-99%

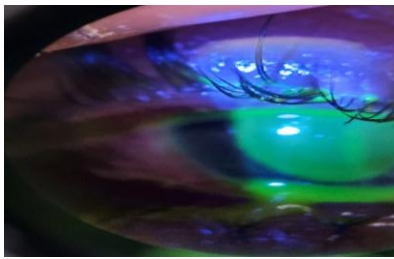
## CASE SERIES:-

### CASE 1

A 31Y female patient presented to OPD after injury with Holi color (acidic injury). Her complaints were-

- Severe pain in both eyes since 1 day
- Pricking sensation since 1 day
- Watering both eyes since 1 day
- Sudden diminution of vision both eyes since 1day

As per history given by patient, her children accidentally spilled Holi color in her both eyes while she was doing her household chores.



**Fig. 1 Right eye with diffuse fluorescence stain positive**

**Fig.2 left eye with congestion**

Table 3. Variation in parameters among right and left eye (Case 1)

	RIGHT EYE	LEFT EYE
VISUAL ACUITY	5/60 No improvement with pinhole or glasses	6/36 6/12 with pinhole -0.75spherical 6/6
EXTERNAL	Both upper and lower lid edema	Both upper and lower lid edema
CONJUNCTIVA	Grade 2 chemosis with circumcorneal congestion with limbal ischemia from 9-12 o'clock	Conjunctival congestion
CORNEA	Pan cornea epithelial defect sparing temporal 3mm and inferior 1mm of limbus.	transparent
ANTERIOR CHAMBER	Normal in depth	Normal in depth and content
IRIS/PUPIL	Iris details faintly visible, pupil sluggishly reactive	Iris structure well defined, pupil brisk reactive to light

LENS	transparent	Transparent
FUNDUS	Media hazy, disc appear normal, rest details not clear	0.3 cup disc ratio, blood vessels and background appear normal, foveal reflex present

After copious saline irrigation for 30minutes, systemic and local treatment was started. Detailed treatment is being described subsequently.

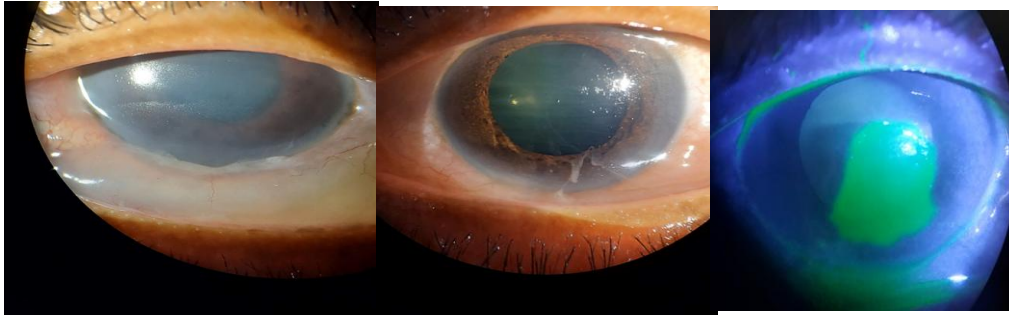
**CASE 2:-**

A female patient, 57y old visited with acid thrown into her both eyes at home by her husband, with complaints as under:

- 1) Sudden diminution of vision both eyes since 2days
- 2) Pain with burning sensation both eyes since 2days
- 3) Inability to open both eyes since 2 days
- 4) Watering both eyes since 2 days.

	<b>RIGHT EYE</b>	<b>LEFT EYE</b>
VISUAL ACUITY	Finger counting 1metre No improvement with glasses.	4/60 No improvement with glasses.
EXTERNAL	Both upper and lower lid edema	Both upper and lower lid edema
CONJUNCTIVA	Chemosis grade 2 with 360degree limbal ischemia	Circumcorneal congestion
CORNEA	Diffuse stromal edema with mild visibility if iris details	Epithelial defect around 6*5mm vertically
ANTERIOR CHAMBER	Depth normal	Depth normal, content clear
IRIS/PUPIL	3mm pupil, sluggishly reactive to light	Normal brisk reactive to light
LENS	Early immature senile cataract	Early immature senile cataract
FUNDUS	Media hazy, details not clear	Media hazy, disc appear normal, rest details not clear

Table 4. Variation in parameters among right and left eye (Case 2)



**Fig 3. Right eye**

**fig.4.Left eye**

### **EXAMINATION AND MANAGEMENT:-**

#### **Case 1**

**Right eye suffered grade 3 and left eye suffered grade 1 chemical injury.**

Copious saline irrigation done to neutralize the pH to around 7.2 for around 30 min. Lid eversion was done to remove the residual particles.

Systemic medication started in form of-

1. Tab Doxycycline 100mg BD
2. Tab Vitamin C 500mg QID
3. Tab Ciprofloxacin 500mg BD
4. Tab Pantoprazole 40mg OD

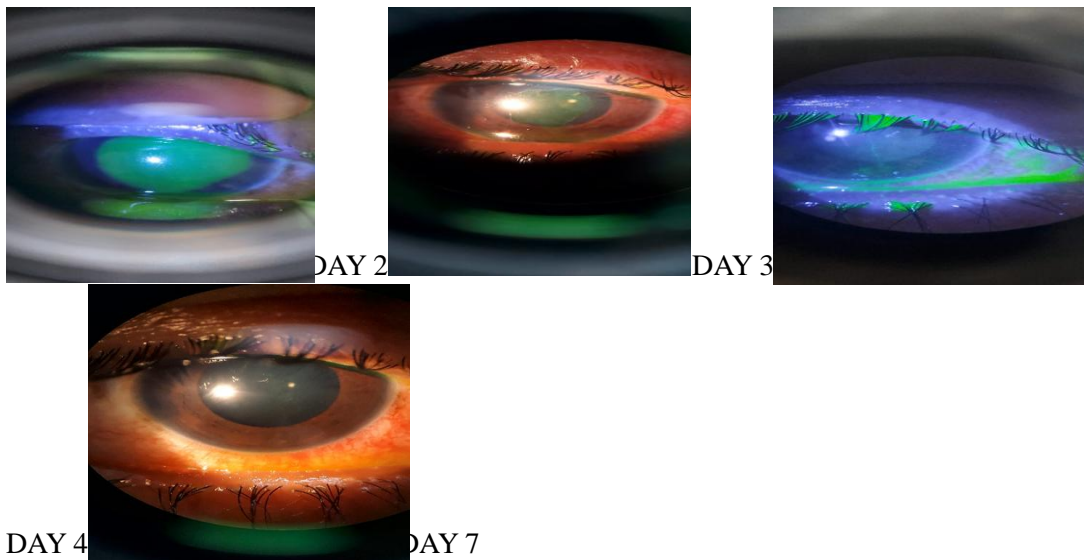
#### Locally (RE)

Patching done by eye ointment Atropine+ ocupol (Chloramphenicol + Polymixin B) + lubricant ointment.

#### Locally (LE)

1. Eyedrop Prednisolone Acetate 1% 6/ day
2. Eyedrop Moxifloxacin 0.5% 1hrly
3. Eyedrop Homatropine 2% TDS
4. Eyedrop Brimonidine+ Timolol BD
5. Eyedrop Sodium Hyaluronate 2hrly
6. Eyedrop Carboxymethylcellulose 1% 6/day

#### **Figure 5. RIGHT EYE**



**Table 5. ocular examination after 1week**

	Right eye	Left eye
VISUAL ACUITY	6/36 with pinhole correction 6/18 BCVA- -0.75 DSP 6/6	6/36 with pinhole correction 6/12 BCVA -0.75DSP 6/6
LIDS	Both upper and lower lid edema (decreased than earlier)	Normal lid and cilia
CONJUNCTIVA	Mild congestion with regaining of vasculature around limbus.	Clear
CORNEA	Nebulomacular opacity as thin line from center to 6 o clock position, fluorescence stain negative	transparent
ANTERIOR CHAMBER	Depth normal, content clear	Depth normal, content clear
IRIS	Brown in color, normal in pattern	Brown in color, normal in pattern
PUPIL	Dilated, fixed, nonreactive under effect of atropine.	Semidilated under mydriatics
LENS	Transparent	transparent

FUNDUS (BE)

Media Clear, CDR 0.3, Disc margin well defined, Blood vessels & Background appear Normal, Foveal reflex present.

### **Case 2-**

**Right eye suffered grade 4 chemical injury and left eye sustained grade 2 chemical injury.**

1. Copious irrigation with normal saline was done at the time of presentation for 30min. Lid eversion was done to remove and wash the residual acid.
2. Injectable antibiotics (Ceftriaxone 1g I.V. twice a day)
3. Tab. Doxycycline 100mg twice a day
4. Tab. Vitamin C 500mg QID with plenty of water
5. Tab. Acetazolamide 250mg BD

#### **Locally (right eye)-**

Eye drop Prednisolone acetate 1% 1hrly

Eyedrop moxifloxacin 0.5% 1hrly

Eyedrop 1% atropine TDS

Eyedrop Brimonidine+ Timolol BD

Eyedrop sodium hyaluronate 2hrly

#### **Locally (left eye)-**

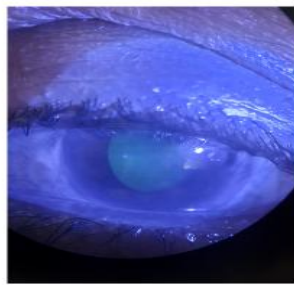
Overnight Patching with eye ointment ocupol (chloramphenicol with polymyxin-B) with atropine ointment and panthegel.

**FIGURE 6- Eye morphology (2 days after initiation)**

2 days after the initiation of treatment:-



Reduction in both eyes lid edema with improvement in eye opening



Healed epithelial defect of left eye

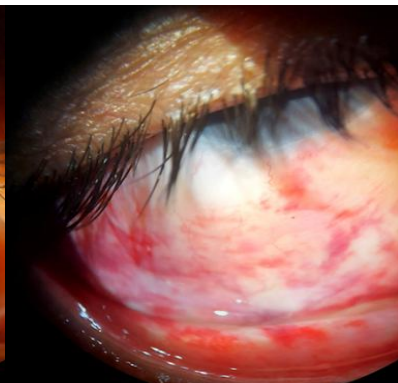


Significant improvement in corneal edema and chemosis

**FIGURE 7- Eye morphology (2 weeks after initiation)**

RIGHT EYE (after 2weeks)

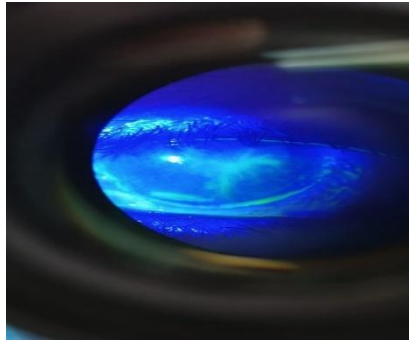
RIGHT EYE (after 2weeks)



**RE (after 2weeks)**

1. Anterior Segment Ischemia with maximum visual acuity of 6/60.
2. Perilimbal ischaemia with conjunctival necrosis with multiple petechial subconjunctival haemorrhage.
3. Diffuse corneal edema with Descemet's folds

After 4 weeks, patient developed right eye dendritic keratitis for which tablet Acyclovir 400mg BD and eye ointment acyclovir 5/day was started and eyedrop Prednisolone was stopped.



**FIGURE 8. showing right eye dendritic ulcer**

PATIENT LATER DEVELOPED RIGHT EYE SECONDARY GLAUCOMA .

#### DISCUSSION:-

“Chemical injuries to the eye are ophthalmic emergencies that require immediate management. Delay in care can result in deeper penetration of the chemical agent resulting in more widespread injury. Long term sequelae of chemical ocular burns include secondary glaucoma, limbal stem cell deficiency, and permanent vision loss”[11].“Pathophysiological events which may influence the final visual prognosis and which are amenable to therapeutic modulation include 1) ocular surface injury, repair, and differentiation, 2) corneal stromal matrix injury, repair and/or ulceration, and 3) corneal and stromal inflammation. Immediately following chemical injury, it is important to estimate and clinically grade the severity of limbal stem cell injury (by assessing the degree of limbal, conjunctival, and scleral ischemia and necrosis) and intraocular penetration of the noxious agent (by assessing clarity of the corneal stroma and anterior segment abnormalities)”[12].

“Immediate therapy is directed toward prompt irrigation and removal of any remaining reservoir of chemical contact with the eye. Initial medical therapy is directed promoting re-epithelialization and transdifferentiation of the ocular surface, augmenting corneal repair by supporting keratocyte collagen production and minimizing ulceration related to collagenase activity, and controlling inflammation”[12].

#### INITIAL MANAGEMENT:-

“Before any workup or in-depth examination occurs, it is critical that the patient receives immediate and copious amounts of irrigation to the affected eyes in order to neutralize the pH as quickly as possible”. [23] While there may be some advantage to using Cederroth Eye Wash[13] or amphoteric solutions such as Diphoterine[14] , the most important factor is reducing time to treatment [12]. “Eversion of the eyelids and sweeping of the fornices is important for identifying and removing any hidden precipitates. Lime, in particular, forms calcium soaps that can be lodged in the superior fornix and cause severe damage if not removed in a timely fashion” [12].

#### MANAGEMENT:-

“The next steps in management focus on closing the epithelium, controlling inflammation, and supporting corneal repair” [10 ,17]. “Treatment should be based on the initial estimate of the extent of limbal stem cell injury. For injuries with any suspected stem cell injury aggressive medical treatment is recommended”[12,15]. “When an absence of limbal ischemia (Roper-Hall I) suggests no limbal stem cell injury, liberal use of preservative free lubrication is usually sufficient to promote re-epithelialization. Re-epithelialization in eyes with stem cell injury (Roper-Hall II or greater) may be

delayed. Full medical support, and sometimes surgical intervention, is required to facilitate expedited epithelial recovery”. [23]

“Reducing inflammation is important when treating chemical ocular injuries as collagenases and degranulating polymorphonuclear leukocytes (PMN) can slow down epithelial regeneration, and lead to persistent stromal inflammation and sterile ulceration” [12]. “Current therapeutic strategies for controlling inflammation include prompt debridement of any necrotic conjunctival tissue and use of anti-inflammatory medications. These include topical corticosteroids, topical citrate, topical medroxyprogesterone, and, in some cases, systemic corticosteroids”[16-18].

“Tetracyclines and ascorbic acid have been found to decrease the risk of ulceration by reducing collagenolysis and promoting repair, respectively. In addition to its antimicrobial actions, tetracyclines reduce collagenase activity, inhibit PMN activity, suppress alpha-1-antitrypsin degradation, and scavenge reactive oxidative species” [16]. “Oral doxycycline is the most potent tetracycline collagenase inhibitor and is the treatment of choice based on its documented clinical and experimental efficacy” [16]. “Ascorbic acid supports stromal repair by serving as a cofactor in the formation of stable triple helix collagen molecules in the stromal matrix as well as through its antioxidant properties” [17,18].

**LONG TERM SEQUELE-** Chronic complications of chemical ocular injuries include limbal stem cell deficiency, vision-limiting corneal scarring, secondary glaucoma, entropion, and cicatrization of the conjunctiva with symblepharon formation.

In a retrospective study done by Daniel J L Bunker et.al in 2014 “the records of 39 patients who presented with chemical-related injury were assessed, 12 of whom had confirmed alkali burns involving the cornea. The most commonly implicated agent was sodium hydroxide, usually in the context of otherwise trivial domestic accidents”. “Acute medical management included copious irrigation and the use of analgesics, cycloplegics, and topical antibiotics. In half the cases, steroid drops and oral vitamin C were also used. Ten of the 12 patients (83%) had return to pre-morbid visual acuity. Complications included cicatricial ectropion, pseudoexfoliative syndrome, and symblepharon”[19]. This study also specified the importance of early acute management of ocular chemical injury to prevent long term complications.

In a study done by Anchal Arora et.al in 2023 “medical records of 15 eyes of 14 patients with ocular injuries caused while using carbide guns, visiting the Institute, from January 2021 to January 2022, were retrospectively reviewed”. “According to the Dua classification, 5 eyes (33.3%) had Grade I-II ocular surface burns, 3 eyes (20%) had grade III burns, and 7 eyes (46.6%) had grade IV-VI burns. Presenting visual acuity ranged between hand movements to 20/50, and in 6 eyes (40%), the visual acuity was  $\leq 20/200$ . Five eyes were managed medically alone, and 10 (66.6%) eyes needed surgical intervention (Amniotic Membrane Transplantation). After a mean follow-up of  $14.23 \pm 11.92$  weeks, complete epithelization was seen in 10 eyes (66.6%). Partial limbal stem cell deficiency and its sequelae such as conjunctivalization of the cornea were noted in 7 eyes (46.6%)”[20].

In a case report by Mayur Anil Patil et al in 2022 at Dr.D.Y.Patil Medical College, Hospital and research centre, Pimpri, they reported about a 11-year-old male patient came to ophthalmology OPD with complaints of loss of vision in the left eye for 5 days. The patient gave a history of trauma to the left eye by a chemical explosive from a carbide gun, after which the patient developed diminution of vision in the left eye for 5 days. The patient was diagnosed with grade 4 chemical injury in her left eye with total epithelial defect, corneal haze with 270degree limbal ischemia[21].

In a rare case report on ocular chemical injury by Deeksha Rani et al in 2020, they reported about A 5-year-old boy with the history of lime falling into the left eye.It was a severe ocular

chemical burn that was graded as Grade IV burn as per Roper-Hall Classification and Grade VI as per Dua's Classification[22].

### **CONCLUSION:-**

Ocular chemical injuries are one of the most important ocular emergencies, constituting a significant proportion of all traumas. To minimize sequelae, prompt and accurate treatment in the early period and successful management of complications in the long term are essential. Chemical ocular injuries have significant psychological, physical, and economic effects, especially since serious injuries can cause permanent blindness. Therefore early and prompt recognition of extent of damage and early initial treatment can help in preventing the potentially blinding condition and the complications.

### **Ethical Approval:**

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

### **Consent**

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

### **REFERENCES-**

1)Singh P, Tyagi M, Kumar Y, Gupta KK, Sharma PD. Ocular chemical injuries and their management. Oman J Ophthalmol. 2013 May;6(2):83-6. doi: 10.4103/0974-620X.116624. PMID: 24082664; PMCID: PMC3779420.

2)Kuckelkorn R, Makropoulos W, Kottek A, Reim M. Retrospektive Betrachtung von schweren Alkaliverätzungen der Augen [Retrospective study of severe alkali burns of the eyes]. Klin Monbl Augenheilkd. 1993 Dec;203(6):397-402. German. doi: 10.1055/s-2008-1045695. PMID: 8145483.

3)BALLEN PH. TREATMENT OF CHEMICAL BURNS OF THE EYE. Eye Ear Nose Throat Mon. 1964 Jan;43:57-61. PMID: 14116244.

4)Roper-Hall MJ. Thermal and chemical burns. Trans Ophthalmol Soc U K (1962). 1965;85:631-53. PMID: 5227208.

5)Dua HS, King AJ, Joseph A. A new classification of ocular surface burns. Br J Ophthalmol. 2001 Nov;85(11):1379-83. doi: 10.1136/bjo.85.11.1379. PMID: 11673310; PMCID: PMC1723789.

- 6) Paterson CA, Pfister RR. Intraocular pressure changes after alkali burns. *Arch Ophthalmol*. 1974 Mar;91(3):211-8. doi: 10.1001/archopht.1974.03900060219014. PMID: 4814971.
- 7) Dua HS, Ting DSJ, Al Saadi A, Said DG. Chemical eye injury: pathophysiology, assessment and management. *Eye (Lond)*. 2020 Nov;34(11):2001-2019. doi: 10.1038/s41433-020-1026-6. Epub 2020 Jun 22. PMID: 32572184; PMCID: PMC7784957.
- 8) Quesada JM, Lloves JM, Delgado DV. Ocular chemical burns in the workplace: Epidemiological characteristics. *Burns*. 2020 Aug;46(5):1212-1218. doi: 10.1016/j.burns.2019.11.007. Epub 2019 Nov 30. PMID: 31791857.
- 9) Beare JD. Eye injuries from assault with chemicals. *Br J Ophthalmol*. 1990 Sep;74(9):514-8. doi: 10.1136/bjo.74.9.514. PMID: 2393641; PMCID: PMC1042196.
- 10) GRANT WM, KERN HL. Action of alkalies on the corneal stroma. *AMA Arch Ophthalmol*. 1955 Dec;54(6):931-9. doi: 10.1001/archopht.1955.00930020937019. PMID: 13268149.
- 11) Cabalag MS, Wasiak J, Syed Q, Paul E, Hall AJ, Cleland H. Early and late complications of ocular burn injuries. *J Plast Reconstr Aesthet Surg*. 2015 Mar;68(3):356-61. doi: 10.1016/j.bjps.2014.10.031. Epub 2014 Nov 5. PMID: 25465150.
- 12) Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. *Surv Ophthalmol*. 1997 Jan-Feb;41(4):275-313. doi: 10.1016/s0039-6257(96)00007-0. PMID: 9104767.
- 13) Rihawi S, Frenzt M, Reim M, Schrage NF. Rinsing with isotonic saline solution for eye burns should be avoided. *Burns*. 2008 Nov;34(7):1027-32. doi: 10.1016/j.burns.2008.01.017. Epub 2008 May 15. PMID: 18485603.
- 14) Schrage NF, Kompa S, Haller W, Langefeld S. Use of an amphoteric lavage solution for emergency treatment of eye burns. First animal type experimental clinical considerations. *Burns*. 2002 Dec;28(8):782-6. doi: 10.1016/s0305-4179(02)00194-8. PMID: 12464478.
- 15) Eslani M, Baradaran-Rafii A, Movahedan A, Djalilian AR. The ocular surface chemical burns. *J Ophthalmol*. 2014;2014:196827. doi: 10.1155/2014/196827. Epub 2014 Jul 1. PMID: 25105018; PMCID: PMC4106115.
- 16) Perry HD, Hodes LW, Seedor JA, Donnenfeld ED, McNamara TF, Golub LM. Effect of doxycycline hyclate on corneal epithelial wound healing in the rabbit alkali-burn model. Preliminary observations. *Cornea*. 1993 Sep;12(5):379-82. doi: 10.1097/00003226-199309000-00002. PMID: 8306657.
- 17) Pfister RR, Paterson CA, Hayes SA. Topical ascorbate decreases the incidence of corneal ulceration after experimental alkali burns. *Invest Ophthalmol Vis Sci* 1978;17(10):1019-1024
- 18) Pfister RR, Paterson CA. Ascorbic acid in the treatment of alkali burns of the eye. *Ophthalmology*. 1980 Oct;87(10):1050-7. doi: 10.1016/s0161-6420(80)35126-9. PMID: 7243199.

19)Bunker DJ, George RJ, Kleinschmidt A, Kumar RJ, Maitz P. Alkali-related ocular burns: a case series and review. *J Burn Care Res.* 2014 May-Jun;35(3):261-8. doi: 10.1097/BCR.0b013e31829b0037. PMID: 23877138.

20)Arora A, Priyadarshini SR, Das S, Mohanty A, Shanbhag SS, Sahu SK. Carbide Gun-Related Ocular Injuries: A Case Series. *Cornea.* 2023 Jun 1;42(6):726-730. doi: 10.1097/ICO.0000000000003095. Epub 2022 Jul 6. PMID: 35867658.

21)Patil MA, Alapati A, Paranjape R, Kilari S, Bora S, Garlapati AG, Palimar MP, Singh VD, Naik KS, Chaudhary NS. A case of ocular chemical injury. *Int J Med Rev Case Rep.* 2022; 6(11): 84-88. [doi:10.5455/IJMRCR.172-1646767808](https://doi.org/10.5455/IJMRCR.172-1646767808)

22)Rani D, Sharma N, Sinha R, Bafna RK. A rare presentation of ocular lime injury. *BMJ Case Rep.* 2020 Sep 7;13(9):e235889. doi: 10.1136/bcr-2020-235889. PMID: 32900734; PMCID: PMC7477973.

23) Caroline Y, Diel RJ, Lai Jiang MD, Greiner MA. Chemical Eye Injury: A Case Report and Tutorial.