An accidental ocular alkali injury - a case report on effective management and successful outcome

Dr. Dipali P. Parmar[1*], Dr. Nikhil B. Adroja[2], Dr. Pradnya K. Bhole[1]

[1]Assistant professor, [2]II[nd] year Resident Doctor, M & J institute of Ophthalmology (Government eye hospital), B. J. Medical College, Ahmedabad.

Corresponding Author: Dr. Dipali P Parmar

Email: dipaliparmar@ymail.com

Abstract

Ocular surface burns require timely interventions to prevent any blinding sequelae. We report a case of an accidental alkali injury successfully managed in terms of eye integrity and visual rehabilitation. A 28-years-old male factory worker presented to the emergency eye care department with accidental spill of caustic soda in left eye before two hours. On presentation, patient was managed with immediate eye irrigation. Ocular examination revealed LE Dua's grade IV injury and visual acuity of counting fingers at 3 meters. Patient was managed with medical treatment and amniotic membrane transplant with tenonplasty for delayed epithelization due to limbal ischemia. Complete ocular surface epithelization was achieved with visual acuity improvement to 6/6 with glasses in LE. Ocular surface burns with alkali agents can cause severe damage to an eye. Immediate eye wash and appropriate medical therapy with timely surgical interventions can prevent blinding sequelae and reduce ocular surface co-morbidities.

Keywords: Ocular surface burns, alkali injury, amniotic membrane transplant, tenonplasty

Introduction

Acute ocular surface burns (OSB) are an ophthalmic emergency requiring prompt treatment to minimize ocular damage. These are the second most common occupational eye injury (12.68%) following that due to ocular foreign bodies (43.42%) and affect males predominantly (68.54% of cases of ocular chemical burns).[1] The factors which determine the severity and outcome of OSB are mainly the chemical and physical characteristics of an offending agent (particularly the pH), concentration, volume, temperature, impact force and duration of contact with the ocular surface, including clinical grading on presentation.[2,3] Management of an acute OSB consists of immediate ocular surface irrigation with appropriate medical therapy to control inflammation and intraocular pressure. Surgical interventions include debridement of the particulate matter and/or necrotic tissue, amniotic membrane transplant (AMT), tenonplasty, limbal stem cell transplant, autologous conjunctival grafts and lamellar and penetrating corneal grafting as and when indicated according to nature and severity of OSB.[2,3]

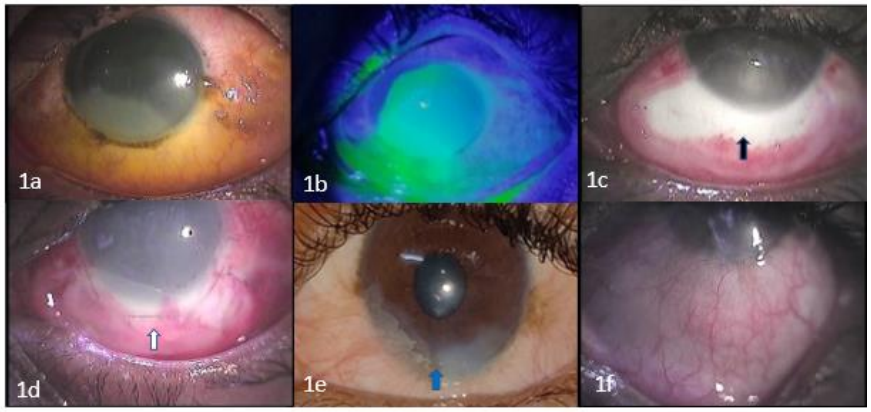
Here, we report a case of an accidental alkali injury successfully managed with medical and timely surgical intervention.

Case report

A 28-year-old male patient presented to the emergency department with complaints of watering, redness and pain in the left eye (LE) for 2 hours following spill of caustic soda powder (concentration not

known) in LE, while working as a color assistant operator at a chemical factory. An immediate copious irrigation with normal saline (2 liters for 30 to 40 minutes) was given under topical anesthesia in supine position, with double eversion of the lids to check any retained particulate chemical agent.[2,3] Patient had no history of use of protective eye gear during the accident. There was'nt any chemical exposure in the contralateral eye nor skin or respiratory tract involvement; hence following emergency left eye care, he was subjected to complete ocular examination. Visual acuity (V/A) post normal saline wash was counting fingers at 3 meters in LE. On slit lamp examination, Dua's grade IV chemical injury was found in LE (Large corneal epithelial defect with stromal edema, Limbal staining from 1 o'clock to 8 o'clock area with approximately 60% involvement of inferior nasal and inferior temporal quadrant of conjunctiva on fluorescein strip staining).[3] Iris and lens details were seen faintly due to corneal cloudiness (Image 1a and 1b). Digital tonometry was normal for LE. Right eye anterior segment was normal with V/A of 6/6. Patient was primarily managed with topical Moxifloxacin (0.5%) eye drops QID, Prednisolone Acetate (1%) eye drops 1 hourly, Carboxy Methyl Cellulose (0.5%) eye drops 1 hourly, Atropine (1%) eye drops TID and ascorbic acid eye-drops (10%) 1 hourly. Systemic therapy included Cap. Doxycycline (100 mg) BD, Tab. ascorbic acid (500mg) BD, Tab. Ibuprofen (400 mg) BD and Tab. Famotidine (40 mg) OD before meal. In subsequent follow up, LE showed re-epithelization of bulbar conjunctiva and superior part of cornea. By 2 weeks, there was delayed epithelization of lower half of cornea and failure of epithelization over adjacent inferior sclera due to limbal ischemia clinically [Image 1c]. Patient was subjected to LE tenonplasty with wet human amniotic membrane transplant (AMT) with fibrin glue and sutures, under local anesthesia for the same [Image 1d]. Frequency of topical prednisolone eyedrops was reduced to QID with tapering as per clinical response with the rest of topical therapy.[2,3] A close follow up was maintained for 3 months till complete epithelization of cornea and conjunctiva was achieved. Patient had successfully achieved corneal and conjunctival re-epithelization with peripheral inferior corneal scarring (<2 mm) and vascularization due to partial limbal stem cell deficiency, without any symblepharon formation [Image 1e & 1f].

Image 1a to 1f: LE slit-lamp images showing corneal & conjunctival staining (Dua's grade IV injury (1a & 1b), inferior limbal and scleral blanching due to ischemia (black arrow, 1c), AMT with tenonplasty and anchoring sutures in situ (white arrow, 1d), inferior conjunctivalization of cornea sparing visual axis due to partial limbal stem cell deficiency (blue arrow, 1e), lower fornix without symblepharon (1f).



Lens was clear with normal intraocular pressure and posterior segment findings. Visual acuity was achieved to 6/6 on Snellen's chart with -2.5 diopter of cylinder at 10 degrees in LE. Patient did not have

any cosmetic concern for pannus and till 6 months of follow up, ocular surface was stable with no further progression of corneal scarring or vascularization.

Discussion

The main offending agents for ocular surface burns are alkalis, acids and irritants like alcohols.The present case had a LE Dua's grade IV alkali injury with good to guarded prognosis (Table 1).[3]

Table 1. Dua classification of chemical eye injury

Grade	Prognosis	Clinical Findings (Limbal involvement)	Conjunctival Involvement	Analogue Scale
I	Very good	0 clock hours	0%	0/0%
II	Good	≤3 clock hours	≤30%	0.1-3/1-29.9%
III	Good	>3-6 clock hours	>30-50%	3.1-6/31-50%
IV	Good-Guarded	>6-9 clock hours	>50-75%	6.1-9/51-75%
V	Guarded-Poor	>9- <12clock hours	>75- <100%	9.1-11.9/75.1-99.9%
VI	Very poor	Total limbus (12 clock hours)	100%	12/100%

Being hydrophilic and lipophilic in nature, alkalis cause deeper penetration in the tissue compared to acidic agents.[2,3] Sodium Hydroxide (NaOH, Synonyms: caustic soda, lye, soda lye, and sodium hydrate) is a strong alkali and very corrosive in nature. It is widely used in various industries (soap, metal, food, drain cleaners etc.). It rapidly causes corneal cloudiness other than irritation upon contact with the eyes, as seen in the present case.[4] Sharma et al. reported moderate to severe injury with sodium hydroxide with severe exposure and delay in eye care resulting in residual limbal stem cell deficiency (6 o'clock hours) with complete epithelization over 3.5 months in their study.[5] The present case was managed with immediate eye irrigation on presentation followed by medical treatment and AMT with tenonplasty for delayed epithelization due to limbal ischemia. Timely irrigation of the eye to remove offending chemical agent has been observed as the most important intervention in managing such cases to reduce disease severity and to improve visual outcome.[6] Topical corticosteroids like dexamethasone 0.1% and prednisolone acetate 1% are the mainstay of medical treatment, especially in the first week of acute phase of moderate to severe OSB to control the ocular surface inflammation. Frequency of topical steroid agents needs to be adjusted as per the severity of OSB and response to the treatment. Anti-proteases agents like doxycycline and sodium citrate as topical and systemic preparations help to reduce inflammation and/or ulceration; as adjuncts to other topical therapy like intraocular pressure reducing agents and cycloplegics.[2,3] AMT is an effective adjunctive modality in the management of acute ocular chemical burns to support epithelialization and restore ocular surface integrity with a potential to improve vision like in the present case.[7] Tenonplasty combined with AMT has shown successful results in terms of ocular surface re-epithelization by providing vascular supply to ischemic area and thereby preventing further melting in severe grades of ocular surface burns as in the present case.[8,9] Eyes with moderate to severe grades of OSB are at risk of losing integrity and vision due to ocular surface melting in acute phase if not addressed timely. Among the topical treatment modalities for alkali burns, it was observed that prolonged treatment with topical steroids when used in conjunction with topical vitamin C is not associated with corneoscleral melting.[10] Lid and adnexa deformity, secondary glaucoma, cataract, limbal stem cell deficiency, corneal scarring and dry eyes require a multidisciplinary approach, later in the chronic phase, for visual and cosmetic rehabilitation.[2,3] Hence,close follow up is recommended by authors in moderate to severe grades of ocular surface burns irrespective of nature of chemical agent for timely surgical intervention.Since chemical injuries are mainly accidental domestic or occupational injuries as in the present case, emphasis should be placed on prevention of such injuries by use of appropriate protective eye gears, especially in the factory workers. The site supervisors/workers in the

workplaces involving such agents must be sensitized about the injuries and trained to give primary care in the form of adequate eye wash to reduce the severe effects of offending agent on the ocular surface. Immediate and adequate eye irrigation must be considered by the primary attending physician for all the patients presenting with ocular surface burns; irrespective of type or nature of the agent and amount of wash given at the site of accident, to prevent ongoing damage by the offending agent.

Conclusion

Ocular surface burns with alkali agents like caustic soda can cause severe damage to an eye. An immediate eye wash followed by appropriate medical therapy and timely surgical intervention can prevent blinding sequelae or reduce ocular surface co-morbidities like symblepharon with lid deformities, limbal stem cell deficiency, corneal scarring and dry eyes.

Declaration of patient's consent

Consent has been obtained from the patient for the images and other clinical information to be reported in the journal. The patient understands that due efforts will be made to conceal his identity.

Conflicts of interest: None

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