

# **Ocular Chemical Burns from Accidental Exposure to Sulphuric Acid due to Car Battery Explosion**

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**ABSTRACT:**

**Introduction:** Chemical burns represent potentially blinding ocular injuries and constitute a true ocular emergency requiring immediate assessment and initiation of treatment. The majority of victims are young and exposure occurs at home, work place and in association with criminal assaults. Alkali injuries occur more frequently than acid injuries. Chemical injuries of the eye produce extensive damage to the ocular surface epithelium, cornea, anterior segment and limbal stem cells resulting in permanent unilateral or bilateral visual impairment. Proper management in the acute setting as well as follow-up by an ophthalmologist is crucial in limiting adverse effects of ocular tissue damage secondary to the chemicals.

**Methods:** This case study describes the presentation and the emergency management of an adult male who presented to the eye department of CMH, Dhaka, with grade III bilateral ocular injury. **Conclusion:** Patient coming with chemical ocular injury need a thorough and immediate evaluation and intensive treatment. Advances in understanding of the pathophysiology of the injury have led to improvement in treatment such as use of topical ascorbate and citrate, as well as surgical treatment. The goal of treatment is restoration of the normal ocular surface anatomy and lid position, control of glaucoma and restoration of corneal clarity.

**Keywords:** Acid, Alkali, Ocular injury

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**INTRODUCTION:**

Chemical injuries to the eye are common and represent one of the “true “ophthalmic emergencies. Practically any chemical can cause ocular irritation. Severe ocular damage is most commonly associated with strong alkaline or acidic compounds. Chemical burns may be induced by means of vapor, solid, or liquid.<sup>1,2</sup> Such injuries produce extensive damage to the ocular surface epithelium, cornea, ciliary body, iris & lens, resulting in permanent unilateral or bilateral visual impairment. They are more prevalent among young

males between 20- 40 years of age, working in factories, chemical industries & laboratories or as a result of an accident.<sup>3,4</sup>

Alkalis tend to penetrate more deeply than acids, as the latter coagulate surface proteins, forming a protective barrier; the most commonly involved alkalis are ammonia, sodium hydroxide and lime. Hydrofluoric acid used in glass etching and cleaning also tends to rapidly penetrate the ocular tissues, while sulphuric acid may be complicated by thermal effects and high velocity impacts associated with car battery explosion.<sup>5</sup>

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The vision impairment or blindness has important health, socioeconomic & quality of life implications. Specially devastating are the alkali injuries because of their inherent ability to lyse cell membranes & penetrate intraocular structures. The severity of chemical injury is related to the type of chemical, the volume of direct exposure, the pH of the solution, and the duration of exposure.<sup>6,7,8</sup>

Acute chemical injuries are graded to plan appropriate treatment and afford an indication of likely ultimate prognosis. Grading is performed on the basis of corneal clarity and severity of limbal ischaemia (Roper-Hall system); it is assessed by observing the patency of the deep and superficial vessels at the limbus.<sup>5,9,10</sup>

**Table: Roper – Hall Grading System of Ocular Chemical Injury**

Grade	Prognosis	Corneal appearance	Limbal ischaemia
I	Good	Epithelial damage	None
II	Good	Haze but iris details visible	Less than 1/3
III	Guarded	Total epithelial loss with haze that obscures iris detail	1/3 to 1/2
IV	Poor	Cornea opaque with iris and pupil obscured	More than 1/2

Alkali burns are the most common cause of ocular burn and also tend to cause the most ocular damage. Regardless of the type of chemical, the treatment modalities are similar with an initial immediate and extensive ocular irrigation for best patient outcomes.<sup>5,9,11</sup>

In this paper, we submit the case of a patient that presented to our hospital with chemical injury in both eyes due to sulphuric acid after an explosion of car battery.

**Case Presentation:** A 52 years old male patient presented to us with accidental chemical injury in both eyes leading to burning sensation and loss of vision. He was a driver and working with a car battery. The battery was explored and both eyes were severely injured with sulphuric acid within it. He was immediately irrigated with a health care practitioner and referred to our hospital. Further irrigation was done in our hospital, sweeping of fornices was done with ciprofloxacin eye ointment. On examination, there was a cut mark on the nose and left side of face. Vision was counting finger, conjunctival congestion, limbal ischaemia less than 180 degree at the lower half, cornea was cloudy, total epithelial loss with severe stromal oedema, iris detail is obscured and anterior chamber is not visible in both eyes, Roper Hall Grading III. No symblepharon is noted. Ocular motility –Full in all gage.



**Fig: Total epithelial loss with haze that obscures iris detail**

The patient was admitted in eye ward and treated with Cap Doxycycline 100 mg bd, Tab Vitamin C 1tab tds, E/D Moxifloxacin 1drop 1 hourly,

Hypromellose eye drop 1 drop 6 times daily, Atropine eye drop 1 drop tds, Prednisolone acetate eye drop 1 drop 1 hourly in both eyes. Bandage contact lens was applied. He was followed up daily.

**By day 2**, corneal oedema reduced and iris details were visible. Visual acuity was improved.

**By day 7**, epithelial healing was completed, Bandage contact lens was removed. Cornea was little bit hazy; Prednisolone eye drop was tapered.

**By day 12**, visual acuity was 6/12 both eyes unaided. No conjunctival congestion, cornea was clear, no limbal ischaemia, anterior chamber showed no abnormalities, lental opacity was found in both eyes. Cap Doxycycline for 2 weeks, Tab Vitamin C for 2 weeks, Moxifloxacin eye drop for 2 weeks, Hypromellose eye drop for 4 weeks was given. He was discharged and advised to report again after 15 days.

**By day 30**, his visual acuity was 6/6, ocular adnexae was normal, cornea clear, iris detail well visualized, No symblepharon was noted. He was advised to apply artificial tear tds both eyes for 1 month.



**Fig: 1 month after the injury**

## **DISCUSSION:**

Chemical injury of the eye is an emergency requiring a good history, prompt clinical evaluation and treatment. The severity of the injury relates directly to the duration, the type of the chemical substances. Most patients with mild to moderate chemical injuries can achieve a stable ocular surface, if the immediate treatment & acute phase is optimal. Usually the surface gets epithelized completely without any sequelae. However severe chemical injuries have a very unfavorable

prognosis. In our case, the patient sustained an explosive chemical injury by sulphuric acid due to explosion of a car battery. It was graded as grade III burn with guarded visual prognosis. Rapid irrigation and dilution of the chemical with a neutralizing solution, preferably distilled water, or even tap water, is the immediate first step of treatment in order to reduce tissue damage and preserve vision. Our patient had got it within time.

The slit lamp examination with the use of fluorescein staining is essential to determine the extent of the injury, the corneal endothelium and into anterior segment structures, such as the iris, lens, and ciliary body.

Application of topical antibiotic eye drop is necessary till the epithelialization of ocular surface. Cycloplegic agents, such as atropine 1%, were administered thrice daily to prevent ciliary spasm.<sup>12</sup>

The corneal stromal oedema and inflammation was treated with steroid for initial 7 days. Steroids reduce inflammation and neutrophil infiltration, and address anterior uveitis. However, they also impair stromal healing by reducing collagen synthesis and inhibiting fibroblast migration. For this reason, topical steroids may be used initially but must be tailed off after 7–10 days.

Tetracyclines are effective collagenase inhibitors and also inhibit neutrophil activity and reduce ulceration. They should be considered if there is significant corneal melting and can be administered both topically and systemically.

Ascorbic acid reverses a localized tissue scorbutic state and improves wound healing, promoting the synthesis of mature collagen by corneal fibroblasts.<sup>5,13</sup>

Symblepharon formation should be prevented as necessary by lysis of developing adhesions with a sterile glass rod or damp cotton bud. IOP should be monitored, with treatment if necessary.

Since severe ocular eye burns are difficult to treat and the course of healing often takes several months, all cases of ocular chemical injuries should be closely monitored, especially within 24 hours of

the injury, in order to monitor progress and identify any sign of complications.<sup>5,14</sup>

### CONCLUSION:

Patient coming with chemical ocular injury need a through and immediate evaluation and intensive treatment. Advances in understanding of the pathophysiology of the injury have led to improvement in treatment such as use of topical ascorbate and citrate, as well as surgical treatment. The goal of treatment is restoration of the normal ocular surface anatomy and lid position, control of glaucoma and restoration of corneal clarity.

### REFERENCES:

1. Singh P, Tyagi M, Kumar Y. "Ocular chemical injuries and their management," *Oman Journal of Ophthalmology*. 2013; 6(2):83–86.
2. Kuckelkorn R, Kottek A, Schrage N & Reim M. Poor prognosis of severe chemical and thermal burns. The need for adequate emergency care and primary prevention. *Int Arch Occup Environ Health*. 1995; 67:281–284.
3. Edwards RS. Ophthalmic emergencies in a district general hospital causality department. *Br J Ophthalmol*. 1987;71(12):938-42.
4. Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. *Surv Ophthalmol*. 1997; 41(4):275–313.
5. Kanski, Jack J. *Clinical Ophthalmology: A Systematic Approach*. Edinburgh: Butterworth-Heinemann/Elsevier, 2015: 881-885.
6. Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. *Surv Ophthalmol*. 1997; 41: 275-313.
7. Hughes WF. Alkali burns of the cornea. Review of the literature and summary of present knowledge. *Arch Ophthalmol*. 1946; 35:423-426
8. Augsburger J, Taylor A. Chapter 19: Ocular and Orbital Trauma. Riordan Eva P, Whitcher JP: Vaughan & Asbury's General Ophthalmology, 17th Edition, The McGraw-Hill Companies, Inc. 2008: 527-533.
9. Jay H. Krachmer, Mark J Mannis, Edward J Holland, CORNEA, Elsevier Health Sciences, 2010: 2665-2684.
10. Roper-Hall MJ. Thermal and chemical burns. *Trans Ophthalmol Soc UK*. 1965; 85:631–653.
11. Burns FR, Paterson CA. Prompt irrigation of chemical eye injuries may avert severe damage. *Occup Health Safety*. 1989; 58: 33–36.
12. Kuckelkorn R, Schrage N, Keller G, Redbrake C. Emergency treatment of chemical and thermal eye burns. *Acta Ophthalmol Scand*. 2002; 80(1): 4-10.
13. Hong J, Qiu T, Wei A, et al. Clinical characteristics and visual outcome of severe ocular chemical injuries in Shanghai. *Ophthalmology*. 2010; 117:2268–2272.
14. Gicquel JJ. Management of ocular surface chemical burns. *Br J Ophthalmol*. 2011; 95:159–161