

# Topical steroid use in the treatment of ocular alkali burns

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## Abstract

**Background**—Ocular alkali burns can be associated with a poor visual outcome. The release of collagenases and proteases after the injury leads to corneoscleral melting. The role of topical steroids in such patients is controversial as they have been postulated to exacerbate corneoscleral melting.

**Methods**—30 patients were reviewed retrospectively after admission to King's College Hospital with alkali burns between 1990 and 1993. All patients were treated with an intense and prolonged regimen of topical steroids and topical and systemic vitamin C.

**Results**—22 patients had mild injuries and eight had severe injuries as estimated by the Roper-Hall grading system. 23 patients were treated with topical steroids for > 10 days and 22 patients were treated with topical vitamin C for more than 10 days. One patient with a severe injury developed corneoscleral melting.

**Conclusion**—Prolonged treatment with topical steroids when used in conjunction with topical vitamin C is not associated with corneoscleral melting.

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Ocular alkali injuries remain a difficult therapeutic challenge. After an ocular alkali burn there is a release of collagenases and proteases which can lead to corneal stromal thinning and perforation.<sup>1</sup> Collagen synthesis is impaired by lower aqueous concentrations of ascorbate<sup>2</sup> and topical ascorbate and citrate have been shown to prevent corneal ulceration and perforation when started immediately after the injury, ascorbate acting by stimulating collagen production by fibroblasts, and citrate acting by inhibiting polymorphonucleocytes (PMN).<sup>3</sup>

It has been suggested that the continued use of topical steroids after the first 10 days following injury may contribute to an increased risk of corneoscleral melting and lead to the development of corneal perforation.<sup>4</sup> Since the introduction of vitamin C therapy, it has been our clinical impression that prolonged steroid therapy has not adversely affected outcome. One of the actions of steroids in the inflammatory response is to block infiltration by PMNs<sup>5</sup> and thus it could be postulated that their use may have a beneficial effect by preventing accumulation of collagenases in an ocular alkali burn.

We reviewed 30 patients with mild to severe ocular alkali burns admitted to King's College Hospital between 1990 and 1993 with reference to our policy of using a combination of intensive topical vitamin C and topical steroid treatment.

## Materials and method

The notes of all patients admitted with alkali burns between 1990 and 1993 were reviewed, the patients having been identified using the computerised hospital activity system. The following information was recorded: demographic details, date and cause of injury, conjunctival pH, initial visual acuity (VA), severity of conjunctival and corneal injury using the Roper-Hall (RHG) grading system.<sup>6</sup> A mild injury was defined as a RHG score of 1 or 2, a severe injury was defined a RHG score of 3 or 4. The presence of lid and facial burns and degree of anterior chamber (AC) activity were recorded.

All patients had the affected eye irrigated copiously at presentation with normal saline 0.9% until the conjunctival pH was neutralised. Patients were admitted after alkali injury if they were found to have conjunctival ischaemia or a corneal epithelial defect and had their injury graded during the first 24 hours after presentation. All patients with a grade 1 injury or greater were treated with a standard regimen which included hourly topical vitamin C (potassium ascorbate 10% in an aqueous vehicle; pH 5.5-6.5, vitamin C) and topical steroid (prednisolone 0.5%). These treatments were reduced slowly, but continued until epithelial healing had occurred and ocular inflammation resolved. Patients also received systemic vitamin C 1 g/day, topical chloramphenicol (drops four times daily), and a mydriatic (cyclopentolate 1%, drops thrice daily).

Outcome measures recorded were final VA and the presence of ocular complications. Only details from one eye from each patient were recorded for the study. There were eight patients in which both eyes were affected. The more severely injured eye was included in the study according to the RHG.

The data were analysed using Minitab statistical package. Probability analysis was performed using Fisher's exact test for categorical data.

## Results

The notes of 31 patients admitted during the study period were reviewed. One patient aged 6 months was excluded as an accurate assessment of her RHG was not possible. The

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Table 1 Ocular alkali burns

Patient number	Age	Sex	Cause of injury	Time to irrigation	Initial VA	RHG conjunctiva	RHG cornea	AC activity	Facial burns	Steroids (days)	Vitamin C (days)	Postop vitamin C (days)	Combined steroids/vitamin C	Final VA	Complications
1	57	M	Assault	unknown	6/18	4	4	3	No	205	80	0	80	6/18	Mild epiphora
2	51	M	Assault	9 hours	CF	4	2	2	No	25	24	24	24	6/9	Alcoholic
3	33	M	Assault	unknown	6/9	4	1	0	No	17	17	10	17	6/9	Central SPK
4	42	M	Assault	48 hours	HM	4	4	3	No	58	52	35	52	CF	Central tarsorrhaphy
5	72	M	Domestic	unknown	6/60	4	3	2	Yes	177	78	78	78	evisceration	Corneal melting
6	29	M	Assault	20 hours	6/24	3	1	0	No	5	5	5	5	6/6	None
7	63	M	Assault	2.5 hours	CF	3	2	0	No	49	45	17	45	6/24	None
8	70	M	Assault	4.5 hours	6/24	2	3	0	No	23	24	0	23	6/12	amblyope
9	56	M	Assault	4 days	CF	2	2	1	No	61	49	28	61	6/12	PSCO
10	30	M	Assault	48 hours	6/6	2	1	0	No	2	2	2	2	6/6	Conj flap
11	35	M	Assault	2 minutes	6/18	2	1	0	No	20	20	20	20	6/6	Absconded
12	52	M	Assault	unknown	6/24	2	1	0	No	12	8	8	8	6/5	None
13	19	M	Assault	45 minutes	6/9	2	1	0	No	16	30	7	30	6/5	None
14	21	M	Assault	2.5 hours	6/6	2	1	0	No	8	8	4	8	6/5	Corneal opacity
15	38	M	Assault	24 hours	6/9	2	1	0	No	19	19	6	19	6/6	None
16	39	F	Domestic	unknown	6/9	2	0	0	No	22	22	3	22	6/6	Corneal opacity
17	60	M	Work related	24 hours	CF	0	2	0	No	16	16	2	16	6/24	None
18	16	M	Assault	unknown	6/9	1	1	0	No	18	18	11	18	6/9	Corneal nebulae
19	67	F	Domestic	unknown	6/9	1	1	0	No	39	11	0	11	6/9	None
20	56	M	Assault	10 days	6/36	1	1	0	No	74	37	14	37	6/6	Entropion
21	18	M	Assault	unknown	6/9	1	1	0	No	1	1	1	1	6/9	None
22	24	M	Assault	2 hours	6/12	1	1	1	No	24	24	4	24	6/9	None
23	40	M	Assault	3 hours	6/9	1	1	0	No	19	12	0	12	6/6	Corneal nebulae
24	45	M	Assault	3.5 hours	6/9	1	1	0	No	9	10	9	9	6/9	None
25	21	M	Assault	unknown	6/5	1	0	0	No	16	16	16	16	6/5	None
26	37	M	Assault	unknown	6/5	0	1	0	No	24	24	21	24	6/4	None
27	17	M	Assault	30 minutes	6/9	0	1	0	No	5	5	5	5	6/5	None
28	32	M	Assault	unknown	6/9	0	1	0	No	28	14	5	14	6/5	None
29	31	M	Assault	2 hours	6/6	0	1	0	No	1	1	1	1	6/6	None
30	65	F	Unknown	unknown	6/24	0	0	1	No	21	0	0	0	6/12	None

median age was 38.5 years (interquartile range (IQR) being 27.75 to 56.25 years). There were 26 men and four women. The majority of patients were seen in 1992 (n = 19). The cause of injury was assault in 24 patients, domestic accidents in five patients, and one work related incident in a plasterer. The median time to presentation was 3.5 hours with an IQR of 1–24 hours. One patient did not attend until 2 days after the injury. For eight patients the time of injury to presentation was unknown. The median follow up time was 23.5 days with an IQR of 9.5 to 80 days.

In those patients in which both eyes were affected (n = 8), the less severely injured eye had a mild injury in all cases. One patient required treatment for 15 days and seven for < 10 days in the less affected eye. In all cases the less affected eye had no long term ocular sequelae.

Table 1 shows the presenting features, treatment and outcome of all the patients. A total of eight patients had a severe injury (RHG 3 or 4 for conjunctiva and/or cornea). Three patients had severe conjunctival and corneal injuries, four patients had severe conjunctival injuries only, and one a severe corneal injury only. There was no significant correlation found between initial VA and conjunctiva RHG score (p = 0.19, relative risk = 1.43, 95% CI +0.9 to +2.3). However, in patients with a corneal RHG score of 3 or 4 none had a VA of > 6/9 at presentation (p = 0.03, relative risk = 1.45, 95% CI +1.0 to +2.1).

Treatment with topical steroids and topical/oral vitamin C was continued in all patients until the epithelial defect had healed and ocular inflammation resolved. Thus, 23 patients were treated with topical steroids for more than 10 days in total and 21 patients were treated with topical vitamin C for more than 10 days.

Eighty per cent of the patients (24/30) had a good visual outcome (VA > 6/12) with minimal complications. This included 21 patients with mild injuries (RHG 1 or 2) for conjunctiva and cornea and three with severe injuries. Of the six patients who did not achieve 6/12 vision, five had severe injuries. The reasons for poor visual acuity were corneal scarring (n = 2), evisceration for a perforated cornea (n = 1), non-healing epithelial defect (n = 1), and phthisis bulbi (n = 1). There was one amblyopic eye.

The patient who developed corneoscleral melting presented with RHG scores of 4 for both conjunctiva and cornea. He developed a corneal perforation and eventually required evisceration 6 months after the injury. There was one case of phthisis bulbi in an alcoholic patient who presented with a grade 4 injury for both conjunctiva and cornea. He absconded from the ophthalmic ward during the first week of treatment and returned only sporadically thereafter. One patient, with a RHG of 2, required a conjunctival flap and one, with a RHG 4, needed central tarsorrhaphy for a non-healing epithelial defect.

Other complications were that one patient required lid surgery for bilateral upper lid ectropion as a result of extensive facial burns, one eye developed a posterior subcapsular

cataract 2 years after presentation, and five had mild corneal scarring (VA 6/5 to 6/9). No patient developed long term glaucoma. No patient with a mild injury (RHG 1 or 2) developed corneoscleral melting despite topical steroid treatment of more than 10 days.

### Discussion

This study was undertaken to assess the effect of an intense treatment regimen in the management of ocular alkali burns. The patient group is similar in distribution to that reported by Beare<sup>7</sup> in that the majority of our patients (80%) sustained their injuries as a result of an assault.

At presentation 16/30 (53.3%) of our patients had a RHG score of 1 or 2 for conjunctiva and 21/30 (70%) had a RHG score of 1 or 2 for cornea. These patients showed rapid re-epithelialisation. As would be expected from their RHG score they had a good visual outcome.<sup>6</sup>

Topical steroids if used for more than 10 days after injury have been suggested to lead to corneoscleral melting.<sup>6</sup> In our study there were eight patients with severe conjunctival and/or severe corneal injuries as indicated by an RHG score of 3 or 4, only one patient developed corneal melting. Three patients had a visual outcome of >6/12.

The role of topical steroids in the management of ocular alkali burns remains controversial. In an animal study Donshick *et al*<sup>4</sup> found that topical steroids if used from day 6 to day 21 following the injury were associated with an increase in number and severity of corneal ulceration. However, in Donshick's study, in which no adjunctive vitamin C was given, 50% of both the treatment and control groups had severe ulceration when steroid treatment was limited to only the first 6 days after injury. In contrast, in our study only one patient had a corneal perforation, despite 77% having topical steroid treatment for more than 10 days. Enhancement of collagenase has been postulated to be responsible for the possible effect of steroid on corneoscleral ulceration.<sup>8</sup> However, no parallel increase in collagenase activity was found by Donshick *et al* in their animal study.<sup>4</sup> Equally, collagenase activity is maximal 9 hours after alkaline injury and steroids might thus be expected to cause early rather than late ulceration.<sup>9</sup> Interestingly, our patient developed corneal perforation 6 months after the injury.

Pahlitzsch and Sinha have postulated that the accumulation of the active form of collagenase maybe linked to a lack of glutamyl transpeptidase, necessary for its breakdown, in the damaged cornea.<sup>10</sup> This enzyme is also

necessary for the breakdown of factors (5-HETE and leukotriene B<sub>4</sub>) in the prostaglandin pathway. These factors are powerful chemotactic agents and could be linked to a persistent infiltration with PMNs. One of the actions of corticosteroids is to block the prostaglandin pathway by stimulating production of lipomodulin<sup>3</sup> which indirectly prevents the initial release of arachidonic acid. Thus, steroids by this mechanism may have an indirect effect on reducing collagenase activity by reducing PMN infiltration.

Pfister and Paterson,<sup>11</sup> using animal studies, showed that there is an acute fall in ascorbate levels in the aqueous following an alkali burn by as much as two thirds. This low level is sustained for 3 days in a moderate injury, but persists for at least 30 days in severe burns. It has been our policy to treat all patients with intense topical vitamin C until epithelial healing has occurred. Twenty two patients had topical vitamin C for a total of more than 10 days. Thus Donshick *et al*'s observation<sup>4</sup> that prolonged steroids cause corneal ulceration may rather be a reflection of the prolonged decrease in aqueous concentration of vitamin C rather than a direct action of steroids. In our study despite prolonged topical steroids in the majority of patients (77%) only one patient had a corneal perforation.

These results suggest that a regimen of intense topical steroids combined with topical vitamin C does not cause corneoscleral perforation, although further prospective work is needed to test this observation.

The authors have no proprietary interest in any of the treatments mentioned.

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- 1 Reim M, Bahrke C, Kuckelkorn R, Kuwert T. Investigation of enzyme activities in severe burns of the anterior segment. *Graefes Arch Clin Exp Ophthalmol* 1993;231:308-12.
- 2 Pfister R. Chemical injuries of the eye. *Ophthalmology* 1983; 90:1246-53.
- 3 Pfister R, Haddox J, Lank K. Citrate or ascorbate/citrate treatment of established corneal ulcers in the alkali-injured rabbit eye. *Invest Ophthalmol Vis Sci* 1988;29:1110-5.
- 4 Donshick P, Berman M, Dohlman C, Gage J, Rose J. Effect of topical steroids on ulceration in alkali burned corneas. *Arch Ophthalmol* 1978;98:2117-9.
- 5 Katzung BG. Prostaglandins and other eicosonoids. In: Katzung BG, ed. *Clinical pharmacology*. California: Lange, 1984:217-8.
- 6 Roper-Hall T, Eagling E. Burns to the eye and periorbital tissue. In: Roper-Hall T, Eagling E, eds. *Eye injuries*. London: Gower, 1986:4.2-4.25.
- 7 Beare J. Eye injuries from assault from chemicals. *Br J Ophthalmol* 1990;74:514-8.
- 8 Brown S, Weller C, Vibrich A. Effect of steroids on corneal collagenase of rabbits. *Am J Ophthalmol* 1970;
- 9 Parrish CM, Chandler JW. Corneal trauma. In: Kaufman HE, McDonald MB, Walkman SR, Barrew BA, eds. *Cornea*. London: Churchill Livingstone, 1988:59-647.
- 10 Pahlitzsch T, Sinha P. The alkali burned cornea: electron microscopic, enzyme histochemical, and biochemical observations. *Graefes Arch Clin Exp Ophthalmol* 1985;223: 27-286.
- 11 Pfister R, Paterson C. Ascorbic acid in the treatment of alkali burns of the eye. *Ophthalmology* 1980;87:1050-7.



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