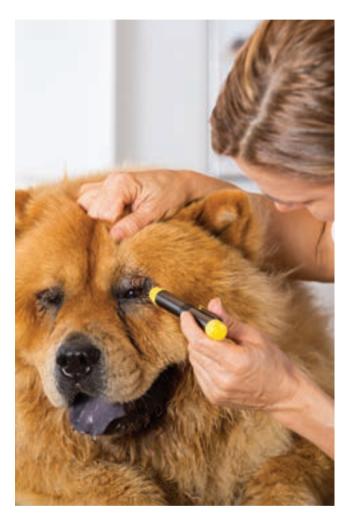
Acute ocular chemical injuries: recognition and treatment

Professor Sheila Crispin MA VetMB BSc PhD DVA DVOphthal DECVO FRCVS, Visiting Fellow, Centre for Comparative and Clinical Anatomy, University of Bristol, UK, outlines how to recognise and treat acute ocular chemical injuries



Chemical injuries can occur in any species, but guard dogs and police dogs are at increased risk in the UK. The causative agent is less likely to be identified when criminal activities are involved, but for domestic accidents the owner should be asked to bring the chemical as well as the animal. Irrespective of the causal chemical the single most important action is immediate irrigation with readily available tap water. The recommended range for the pH of drinking water in the US and UK is 6.5 to 8.5. Drinking water in the UK is usually between seven (neutral) and eight (slightly alkaline). The immediate response to chemical injury often determines the final outcome.

ACIDS

Acids precipitate protein and unless they are particularly strong (ie. pH 2.5 or less), they do not penetrate beyond the corneal epithelium. Examples of strong acids include

hydrofluoric acid, which may pass rapidly through cell membranes and enter the anterior chamber and sulphuric acid

ALKALIS

Alkalis are lipophilic and react with fats to form soaps, which damage cell membranes; they thus have the capacity to penetrate the eye, so that their damaging effects can be widespread (conjunctiva, cornea, limbus, anterior chamber, iris, ciliary body, lens zonules, lens and drainage angle). The speed of penetration from fastest to slowest is ammonium hydroxide, sodium hydroxide, potassium hydroxide and calcium hydroxide. The latter, which is found in plaster and mortar, does not penetrate well as the calcium soaps formed upon saponification are relatively insoluble and precipitate out, so forming a barrier to further penetration. Conjunctival damage can result in perilimbal ischaemia, symblepharon formation, eyelid deformities such as cicatricial entropion and ectropion, tear film problems because of damage to the conjunctival goblet cells and other complications of extensive scarring. If there is loss of pluripotential limbal stem cells longer term treatment is complex and specialist help should be sought for the management of most alkali burns.

CLINICAL SIGNS OF ACUTE CHEMICAL INJURY

Conjunctival changes range from mild hyperaemia and chemosis to disastrous limbal ischaemia. Corneal changes range from focal or diffuse corneal clouding, with or without punctate epithelial erosion, to pancorneal opacification, ulceration and, occasionally, perforation. For most alkalis and some strong acids, the stromal changes are essentially similar to those of liquefactive stromal necrosis.

Other anterior segment changes range from mild aqueous flare to frank uveitis, or more generalised anterior segment inflammation. Lens luxation can follow damage to the lens

The intraocular pressure may rise soon after the injury. Chemical burns involving the eyelids are similar to those of skin, but because the subcutaneous tissue is areolar, there may be marked swelling. Skin damage from chemicals is classified as first-degree (epidermis only), second-degree (epidermis and dermis) and third-degree (epidermis, dermis and subcutaneous tissue). The management of eyelid chemical injury is based on a similar classification.

EMERGENCY TREATMENT

As an emergency measure, advise the owner to irrigate the eye with copious quantities of tap water and to bring the

SMALL ANIMAL I CONTINUING EDUCATION

animal without delay for veterinary assessment. When the animal arrives, apply topical anaesthetic (proparacaine hydrochloride), check pH of conjunctival sac if possible and ensure that irrigation is continued until a near neutral conjunctival sac pH has been restored (normal pH of conjunctival sac is about 7.4, the alkaline side of neutral); tap water can be replaced by sterile isotonic saline, balanced salt solution (BSS) or, better, balanced salt solution plus (BSS plus) which is enriched with bicarbonate, dextrose and glutathione.

The irrigation solution must contact the ocular surface and is usually delivered by hand via the intravenous tubing. Irrigation should be continued until the pH of the ocular surface is neutralised to a range of 7.0-7.5. Usually 1-2L of fluid will be required. Ensure irrigation for 20-30 minutes and monitor the pH at 15-minute intervals up to and after stabilisation. Once irrigation has achieved a near normal pH, which is maintained, a complete examination of the eye and adnexa should be performed. When pain and ocular damage are severe, examination under general anaesthesia may be required, in addition to the topical local anaesthetic previously applied. Particular attention should be paid to the ocular surface, the fornices, under the eyelids (upper, lower and third eyelid) for residual particles and what can be seen of intraocular structures. If possible identify the causal chemical and its pH.

ACUTE PHASE TREATMENT OF CHEMICAL INJURIES

For mild chemical injuries with no intraocular involvement, topical antibiotic ointment and systemic analgesic is usually

sufficient. Preservative-free tear substitutes can reduce ocular discomfort when there is epithelial erosion. When there is involvement deeper than the corneal epithelium, treatment may require specialist help and the regime adopted may be similar to that used for liquefactive stromal necrosis (melting ulcer).

Control pain with systemic drugs and ifthere are any indications of intraocular involvement (for example iridocyclitis or glaucoma) a short-acting cyloplegic should be applied topically and antiglaucoma therapy initiated, in addition to the suggested regime for ocular surface injuries. Additional drugs may be useful to support ocular healing - for example using ascorbate orally (500mg four times daily in the dog) and topically as a 10% solution formulated in artificial tears. Ascorbate plays a fundamental role in collagen remodelling, leading to an improvement in corneal healing. It may also prevent or delay corneal ulceration. It is available in topical and oral forms. Oral tetracycline may also decrease the risk of corneal melting through inhibition of matrix metalloproteinases. Controlling inflammation with topical steroids can help break this inflammatory cycle. Prednisolone acetate 1% should be used four times daily for one week in a mild chemical burn. This should be increased to hourly dosing in more severe burns. Steroids should be discontinued or tapered rapidly by 10-14 days to avoid corneal melting.

REFERENCES

1. Singh P, Tyagi M, Kumar Y et al. Ocular Chemical Injuries and their management 2013: 6(2): 83-86

READER QUESTIONS AND ANSWERS

- 1. WHAT IMMEDIATE ACTION SHOULD BE TAKEN BY THE VETERINARIAN IF ACUTE OCULAR CHEMICAL INJURY IS SUSPECTED?
- A Detailed ocular examination
- B Measurement of intraocular pressure (usually after topical local anaesthetic has been applied and the pH of the conjunctival sac has been measured)
- C Copious and continuous topical irrigation with balanced salt solution or similar irrigating fluid, including water (usually after topical local anaesthetic has been applied and the pH of the conjunctival sac has been measured)
- D Topical application of preservative-free tear replacement solution (usually after topical local anaesthetic has been applied)
- 2. WHY ARE ALKALIS GENERALLY MORE DAMAGING TO THE EYE THAN ACIDS?
- A They precipitate tissue proteins in the corneal epithelium and anterior stroma
- B They are lipophilic and can penetrate the eye, causing saponifiation and lysis of cell membranes
- C They cause immediate corneal ulceration
- D They produce immediate eyelid adhesions

- 3. AT WHAT PH DO ACIDS PENETRATE BEYOND THE CORNEAL EPITHELIUM?
- A pH 6.5 or less
- B pH 4.5 or less
- C pH 3.5 or less
- D pH 2.5 or less
- 4. WHAT EFFECT ON INTRAOCULAR PRESSURE FOLLOWS SEVERE CHEMICAL INJURY TO THE CORNEA AND SCLERA?
- A No effect
- B An immediate rise
- C An immediate fall
- D An increase 24 hours after the injury
- 5. WHICH OF THE ALKALIS LISTED BELOW HAS THE FASTEST SPEED OF OCULAR PENETRATION?
- A Sodium hydroxide
- B Potassium hydroxide
- C Ammonium hydroxide (ammonia)
- D Calcium hydroxide

VARMERS: 1:C, 2:B, 3:D, 4:B, 5:C