Management of chemical burns of the canine cornea

Richard Christmas

Abstract

Significant clinical signs and general principles of treatment for chemical burns of the canine cornea are presented using three typical case studies for illustration. Alkali burns are more common in dogs than acid burns. The sources of alkali in this study were soap, cement, and mortar dust. Common signs of chemical burns are ocular pain, corneal ulceration, tear film inadequacy, corneal edema, and marked corneal neovascularity. Successful treatment requires thorough ocular lavage, treatment for corneal ulceration, and adequate anti-inflammatory therapy when the corneal epithelium becomes intact.

Résumé

Approche thérapeutique lors de brûlures chimiques de la cornée chez le chien

Les auteurs présentent les principes généraux du traitement pour les brûlures chimiques de la cornée chez le chien ainsi que les signes cliniques les plus importants en illustrant trois cas cliniques représentatifs. Les brûlures alcalines sont plus fréquentes que les brûlures acides chez le chien. Les produits alcalins identifiés dans cette étude provenaient de savons, du ciment et de poussière de mortier. Les signes cliniques communément rencontrés lors de brûlures chimiques sont une douleur oculaire, une ulcération, un oedème, une néovascularisation marquée de la cornée et une production de larmes inadéquate. Le succès du traitement est basé sur un lavage oculaire complet, le traitement de l'ulcère cornéen et d'une thérapie anti-inflammatoire adéquate lorsque l'épithélium cornéen est intact. *(Traduit par Dr Thérèse Lanthier)*

Can Vet J 1991; 32: 608-612

Introduction

Chemical burns affecting the cornea can be among the most severe ocular emergencies in dogs. It is rare for owners to recognize a chemical burn in their dog's eye. As a result, owners will not normally be able to provide the appropriate history, and diagnosis will be difficult unless the veterinarian is familiar with the clinical signs and appearance of the cornea after it has been affected by an acid or an alkali. In my experience, the most common presentation is that of a patient in which the chemical insult has occurred some days previously.

My purposes in this paper are to highlight the typical clinical signs of chemical burns of the cornea in the dog, describe treatments available, and summarize current thoughts on emergency treatment. Three cases that best illustrate typical presentations have been selected.

17th Avenue Animal Hospital, 233 17th Avenue SW, Calgary, Alberta T2S 0A4

Case studies

Case 1

A two-year-old Lhasa Apso bitch was presented (Day 1) because of severe ocular pain and blepharospasm of both eyes. The patient required sedation with meperidine (Demerol, Winthrop, Aurora, Ontario) and acepromazine maleate (Ayerst, Montreal, Quebec) before it would allow an adequate ocular exam. I found bilateral severe complete circumcorneal superficial neovascularity extending 1-2 mm into the cornea from the limbus. Each eye exhibited a moderate degree of corneal edema that was worse centrally. Anterior uveitis was present in each eye. The right eye had a superficial corneal ulcer, mucopurulent conjunctivitis and a Schirmer tear test value of 8 mm. The left eye also had mucopurulent conjunctivitis, and a Schirmer value of 5 mm. The owner had noticed the animal having ocular problems the night before but could not think of any noxious insult that the eyes had been exposed to. Further questioning revealed that the dog had been bathed by a family member several days before. A presumptive diagnosis of alkaline corneal burn was made.

General anesthesia was required to treat the eyes because of the severity of ocular pain. The eyes were washed thoroughly for several minutes with a balanced salt solution (Eye Stream, Alcon Canada Inc., Mississauga, Ontario). Eight mg of methylprednisolone acetate 20 mg/mL (Depomedrol, Tuco Products, Orangeville, Ontario) were injected into the superior subconjunctival area of the left eye. The subconjunctival route was used to reduce the need for topical drug application which was going to be difficult for the owner due to the dog's severe ocular pain. Steroids are contraindicated in the canine cornea when corneal integrity is affected; cortisone was not used on the right eye because of the ulcer. A mixture of 5 mL chloramphenicol ophthalmic drops (Maurry Biologicals, Los Angeles, California, USA), 5 mL 20% acetylcysteine (Mucomyst, Bristol Labs, Belleville, Ontario), and 15 mL artificial tears (Hypotears, Iolab, Johnson and Johnson, Peterborough, Ontario) was made and is hereinafter referred to as "ulcer mix". The ulcer mix was used topically in each eye q2h. Atropine 1% (Alcon Canada Inc., Mississauga, Ontario) was used q1h in each eye until the pupils were dilated and then once daily. The corneal ulcer on the right eye was debrided with a #15 scalpel blade and 10 mg of gentamicin sulfate (Gentocin, Schering Canada Inc., Pointe Claire, Quebec) were injected subconjunctivally.

To treat the severe keratitis and the uveitis and yet not interfere with the healing of the ulcer on the right eye, a nonsteroidal anti-inflammatory drug (0.25 mL of flunixin meglumine 50 mg/mL; Banamine, Schering Canada Inc., Pointe Claire, Quebec) was injected IV. On day 2 the treatments were repeated and the animal sent home with a cervical fixation cone (McCarthy and

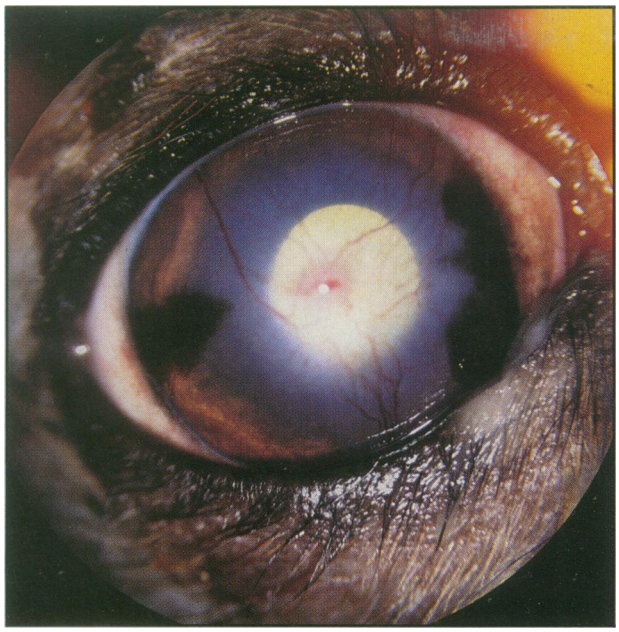


Figure 1. Lhasa Apso right eye three weeks postchemical burn. Note the neovascularity of the cornea with a vascular plaque centrally and pigmentary keratitis medially and laterally. The neovascularity resolved following topical steroid therapy but the pigmentary keratitis remained.

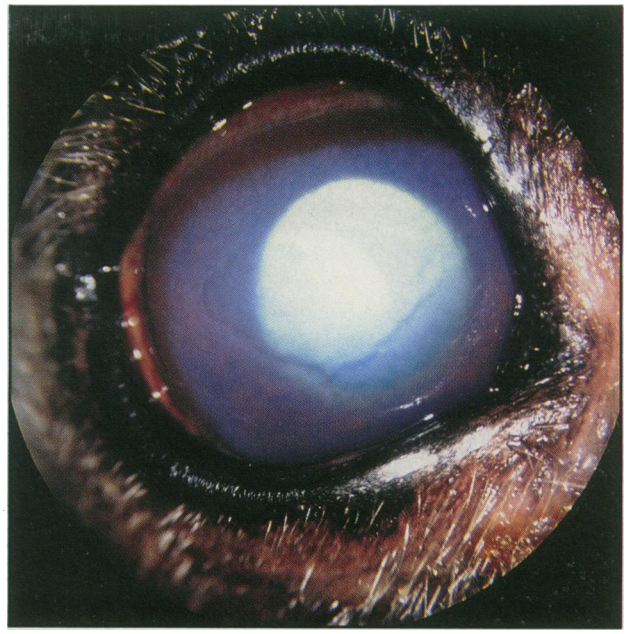


Figure 2. West Highland White terrier, chemical burn of the right cornea. Note the prominent neovascularity of the cornea, large superficial corneal ulcer, diffuse corneal edema, cloudy anterior chamber. Pupil has been dilated with atropine.

Sons, Calgary, Alberta) and with instructions for the owner to give the patient 80 mg of acetylsalicylic acid (Baby aspirin, Sterling Drugs, Aurora, Ontario) per os q12h. On day 4 the owner returned the patient because the patient would not allow treatment of the right eye. The ulcer of the right eye was a little deeper probably because the owner had been unable to administer the medications. Under general anesthetic the third eyelid was sutured over the right eye. To stimulate tear secretion, two drops of 2% pilocarpine (Alcon Canada) were placed in food and given per os q12h. Because there was no evidence of corneal epithelium breakdown on the left eye, topical dexamethasone phosphate (Maurry Biologicals) one drop q6h was begun for the left eye only.

Three weeks following the initial presentation the third eyelid flap was removed from the right eye. The ulcer had healed but there was significant neovascularity of the cornea, pigment deposition in the medial and lateral aspects of the cornea, and a diffuse hazy appearance to the cornea (Figure 1). The left eye had improved dramatically and was normal in appearance as seen with the unaided eye but ghost vessels were evident in the cornea with slit lamp examination. The Schirmer tear test had increased to 10 mm for each eye. Dexamethasone phosphate ophthalmic ointment (Maxitrol, Alcon Canada) was dispensed to be used q6h in each eye. Three months after the initial presentation the Schirmer tear test was 12 mm in each eye. There was pigmentary keratitis of the left eye but the most prominent corneal vessels had resolved to small diameter vessels on the superior aspect of the right cornea that were visible only by slit lamp examination. Much of the corneal haze had disappeared. The owner observed that the dog's vision appeared normal from both eyes. The oral pilocarpine had been discontinued by the owner some weeks before. The owner was warned to watch for further signs of keratoconjunctivitis and was dispensed an ophthalmic ointment (Vetropolycin HC, MTC Pharmaceuticals, Mississauga, Ontario) to be used q12h in the right eye for another 30 days.

Case 2

The second case involved a nine-year-old West Highland White terrier. The owners' home was being renovated and mortar dust had accidentally blown into the dog's right eye. The dog was presented to an emergency clinic because of acute ocular pain several days following the accident and referred to me the next day (Day 1). There was severe blepharospasm and a mucopurulent discharge from the right eye. Other ocular findings were superficial perilimbal neovascularity, diffuse mild corneal edema, superficial central corneal ulcer, and severe anterior uveitis (Figure 2). The left eye was normal. Schirmer values were 2 mm in the right eye and 12 mm in the left eye. At the emergency service the animal had been treated with dexamethasone phosphate (Azium, Schering Canada) 3 mg IM, prednisolone 10 mg per os q12h, 1% atropine ophthalmic drops (Alcon Canada) q12h topically, gentamicin sulfate ophthalmic drops (Gentocin, Schering Canada) 1 drop q2h topically, and a cervical fixation cone applied.

Following initial examination, a topical anesthetic was applied to the right eye (proparacaine, Maurry Biologicals) and the eye flushed with Eyestream. The corneal ulcer was debrided with a #15 scalpel blade. Ulcer mix was prepared and used q2h, chloramphenicol 250 mg was given per os q12h, and flunixin meglumine 0.25 mL IV. Cyclosporin A (Sandimmune, Sandoz, Montreal, Quebec) was mixed to a 2% solu-

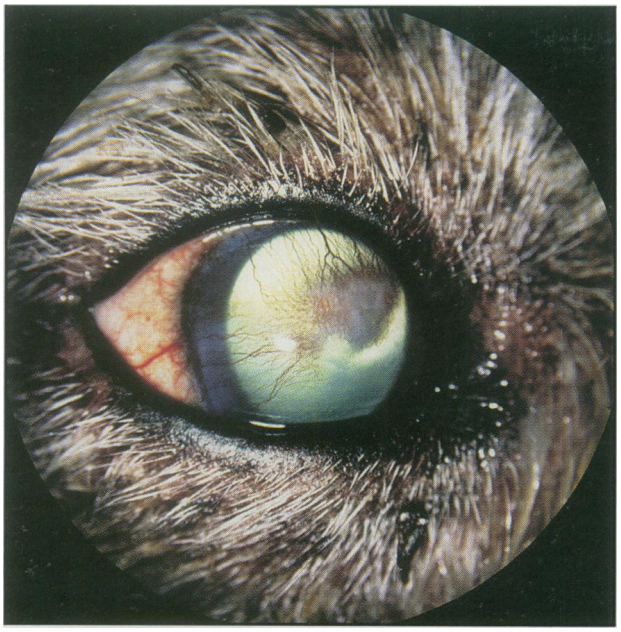


Figure 3. Same dog as Figure 2 three weeks following treatment and before topical steroids were started.

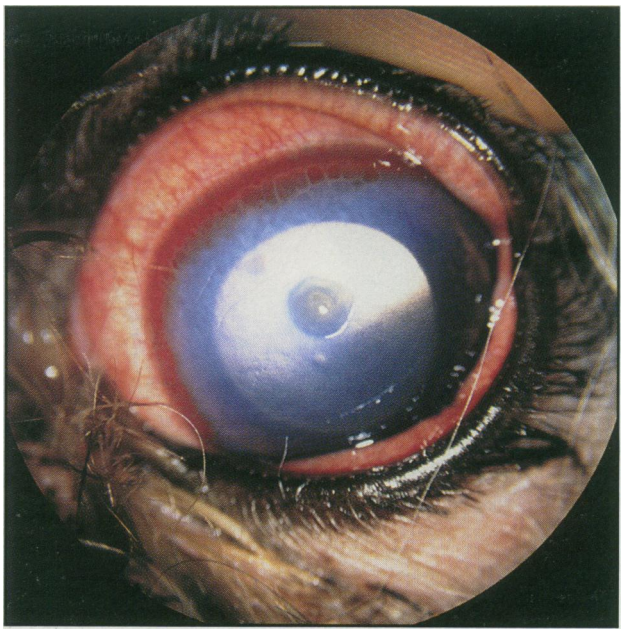


Figure 4. Chemical burn of the right cornea in a Shih Tzu. Note severe perilimbal neovascularity, mild corneal edema, mid-stromal central corneal ulcer. The pupil has been dilated with atropine.

tion with olive oil and applied topically q12h to stimulate tear production. Although cyclosporin is used mainly to treat keratoconjunctivitis sicca of suspected immune etiology because of its T cell inhibiting property, there is some evidence that it also has a tear-stimulating property of undefined mechanism (1). The dog was discharged from the hospital with ulcer mix, atropine, cyclosporin to be used topically as described, and chloramphenicol orally. The owners were asked to administer 100 mg acetylsalicylic acid q12h per os for its antiprostaglandin effect.

On day 6 the uveitis and blepharospasm had resolved but the corneal ulcer was not yet epithelialized and there was significant neovascularity present. Atropine frequency was reduced to one drop every three days, but the other medications were continued. On day 10 the corneal ulcer was almost healed, the Schirmer tear values were 13 mm for the right eye and 17 mm for the left eye. The chloramphenicol and atropine were stopped. On day 19 the ulcer had healed but there was a raised white inflammatory plaque where the ulcer had been surrounded by corneal vascularization (Figure 3). Flumethasone (Anaprime, Syntex Agribusiness, Mississauga, Ontario) was dispensed to be used q6h to reduce the neovascularization. By day 29 the eye had a normal appearance to the unassisted eye but there was still evidence of neovascularity by slit lamp examination. Fluoromethalone (Flarex, Alcon Canada) was prescribed to be used q6h for one week, q8h one week, q12h one week, and once a day for one week. Because the Schirmer tear values were 17 mm in each eye, the cyclosporin was discontinued. Vision was normal by client observation. The right eye appears normal, there has been no recurrence of the keratoconjunctivitis sicca, and Schirmer values for both eyes remain the same three years later.

Case 3

A six-month-old Shih Tzu dog was presented because of moderate ocular pain and blepharospasm of the left eye. Examination revealed corneal edema, superficial corneal neovascularity, a central mid-stromal corneal ulcer, and mild anterior uveitis (Figure 4). The Schirmer tear values were 8 mm in the affected left eye and 15 mm in the unaffected right eye. Although the owner was unaware of trauma of the eye it had been noted that the animal had been near workmen who had been mixing powdered cement several days before the painful eye was noticed. A presumptive diagnosis of alkaline corneal burn was made. Because the eye was painful and the animal uncooperative, sedation with atropine, demerol and acepromazine followed by halothane anesthesia was used for treatment. The eye was lavaged thoroughly with Eyestream, the corneal ulcer debrided with a #15 scalpel blade, gentamicin sulfate 10 mg was injected subconjunctivally, and a third eyelid flap was used to protect the eye. Ulcer mix as described above was prepared for use q2h. Atropine 1% was used topically once a day. The third eyelid flap was removed on day 10. The corneal ulcer had healed but superficial neovascularity of the cornea, especially superiorly, was severe. The Schirmer value was 15 mm. Flumethasone drops (Anaprime) were dispensed to be used q8h for 10 days. When examined three months later, the left eye appeared normal.

Discussion

Chemical burns of the cornea are among the most critically urgent ocular emergencies. Chemical burns are either acid or alkaline in nature. Alkaline materials penetrate more rapidly into the cornea and anterior chamber and are generally more serious than acids (2–5). Alkalis combine with the lipids of the cell

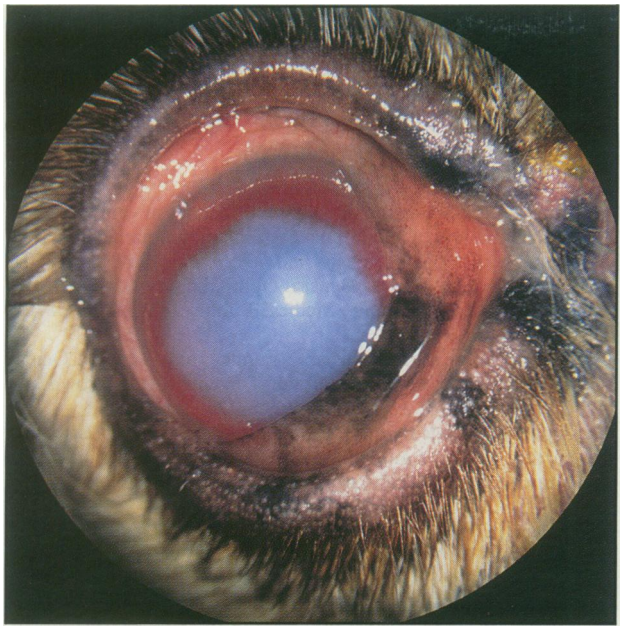


Figure 5. Chemical burn of the right cornea in an American Cocker Spaniel. Note the severe perilimbal vascular response, diffuse corneal edema and the miotic pupil.

membranes and destroy glycosaminoglycan ground substance thereby disrupting cells and causing softening of the tissue and devitalization of the keratocytes. This allows additional alkali to penetrate and causes further damage; this process may continue for several days (2–4). The degree of alkalinity will determine the extent of the damage (3). Effects alkali may have on the eye are: ischemia and necrosis of the sclera and conjunctiva, corneal ulceration and perforation of the globe, an immediate increase in intraocular pressure due to scleral contraction, destruction of the trabecular meshwork which can cause glaucoma, damage to lacrimal glands and their ducts leading to temporary or permanent keratoconjunctivitis sicca, cataract formation, and anterior uveitis.

Soaps are mild alkalis and are the most common source of chemical burns in dogs and cats. Other common sources of alkalis are: lye (NaOH, KOH), and lime (CaO), which are found in materials like fresh mortar, plaster, whitewash, or line-marking powders on sport fields; ammonia and magnesium hydroxide, which are used in fireworks and sparklers.

Acids cause damage in the first few hours after exposure. Acids quickly precipitate tissue proteins which set up physical barriers against further penetration and limit damage to the area of contact (4). In addition the tissues themselves have a buffering and neutralizing action on the acid (4). In the home the most common sources of acid are battery acid and cleaning agents like muriatic or glacial acetic acids (3). Fortunately, most pets are not exposed to industrial or laboratory acids such as hydrofluoric acid or those containing heavy metals that are capable of penetrating the cornea and anterior chamber damaging the endothelium and causing intraocular scarring and membrane formation (4).

The examples chosen for this paper illustrate some of the common features of chemical burns of the canine cornea. Usually the owner is not aware that chemical contact has been made. Breeds with abundant periorbital facial hair (Shih Tzu, Lhasa Apso, Old English Sheepdog) are more predisposed to problems because they require more cleaning of these areas and because it is harder for the owner to see eye lesions if the eyes are obscured by hair. Patients are often presented with severe blepharospasm, tear inadequacy, corneal neovascularity, corneal ulcers, and uveitis. Most uncomplicated corneal ulcers caused by trauma in an otherwise healthy eye heal in seven days, but ulcers caused by chemical burns frequently heal more slowly because of damage to the keratocytes and the tear film.

An essential part of treatment is irrigation. In an emergency out of the hospital, use copious amounts of tap water while the lids are held open as much as the patient will allow. In the clinic, topical ophthalmic anesthetic should be used to ameliorate the pain. In some cases general anesthetic will be required even for irrigation. Warm sterile saline or an eye wash or balanced salt solution are desirable. Washing needs to continue for as long as 20–30 minutes in severe cases. Remember to wash out the fornices and under the third eyelid as well as the cornea. The pH section of a urinalysis test strip may be useful to determine when irrigation is sufficient or to determine whether the chemical is acid or alkaline. Normal pH of conjunctiva ranges from 7.3–7.7 (2). It may be necessary to determine the pH 5 min after neutral pH is achieved to make sure the pH is not changing.

The use of neutralizing agents is controversial. Some advocate that simple dilution with water or saline is preferred (4), others feel that neutralizing agents are dangerous because there is the possibility of heat generated by a chemical reaction causing further damage (2), or that valuable time will be lost in searching for antidotes (5). Because of the fact that neutralizing solutions are rarely available in most clinics and because of the reports of possible harm from these, I suggest that immediate and thorough irrigation with water, saline or balanced salt solutions is the most practical. The neutralizing solutions advocated by others are: 3% sodium bicarbonate for acids, and 2% acetic acid (dilute vinegar) or 10% neutral ammonium tartrate in distilled water for alkali burns (6). If the epithelium has been denuded, some authors advocate the use of sodium edatate (EDTA) if there are discrete particles embedded in the tissues (2,6).

Once irrigation is complete, adjunctive treatments should begin. Topical atropine is necessary if there is pain from iridospasm as a result of an ulcer or anterior uveitis. Atropine should be used to effect only. Once cycloplegia is accomplished, the frequency of atropine should be reduced in my opinion. There is evidence that atropine does cause a temporary drop in tear production (7) and, since most patients with chemical burns have compromised lacrimal function, it is wise to use atropine only to the extent needed to maintain cycloplegia.

It has been observed by Pfister and coworkers that in the rabbit eye following a chemical burn there is a lack of vitamin C in the aqueous humor (8). They suggest that 10% citrate every 30 min for 14 h might be an effective treatment for corneal ulcers resulting from

alkali burns to the human eye. Anticollagenase drugs such as acetylcysteine (Mucomyst 20%), sodium edatate, or serum are valuable to combat the collagenase released by damaged corneal and conjunctival epithelial cells (2,3,5,6).

As a result of inflammation or because of the entry of hydrogen ions into the anterior chamber in an acid burn, there is significant release of prostaglandins in the eye (4). Systemic antiprostaglandins such as flunixin meglumine or acetylsalicylic acid or topical antiprostaglandins such as indomethacin (Indocid, MSD, Pointe Claire, Quebec) or flurbiprofen sodium (Ocufen, Allergan, Markham, Ontario) should be used (9,10). If there is a corneal ulcer, topical antiprostaglandins or steroids must be used with caution. There is usually significant neovascularity of the cornea following a chemical burn but use of topical steroids is contraindicated if the cornea is ulcerated. However, systemic steroids may be used and are indicated if there is uveitis. Steroids may be used in conjunction with antiprostaglandins since they inhibit the inflammation cascade at different points (9,10).

Soft therapeutic contact lenses may be used to support the cornea, act as a bandage, and add to patient comfort. Contact lenses help reduce the chances for symblepharon (11). Contact lenses must be meticulously clean and changed twice weekly as a minimum. Collagen corneal shields which last up to 72 hours before dissolving have recently become available. The shield comes in a dehydrated state and may be rehydrated with artificial tears or an antibiotic to provide high levels of drug in the cornea (12,13).

Topical and or systemic antibiotics should be used when there has been chemical damage to the cornea and the conjunctiva because the host defense system is compromised and secondary bacterial infection must be prevented. Chloramphenicol remains the best choice not only because of its ability to penetrate intraocularly, which may be less important if the epithelium is not intact, but also because some feel that drugs such as gentamicin, neomycin, and tobramycin are toxic to the epithelial cells (4).

Surgical procedures such as the third eyelid flap and bulbar conjunctival flaps are useful adjuncts to therapy. For superficial ulcers I prefer to leave the eye exposed to be able to see the cornea. For an ulcer that is mid-stromal in depth or where the patient is uncomfortable, the flap procedures are needed. If an ulcer is deeper than mid-stromal, a complete bulbar conjunctival flap should be used since it provides much better support to the cornea and allows the cornea to come in contact with a blood supply that will deliver systemic drugs, products of the immune system, and serum, which has an anticollagenase effect. In addition, for the eye with minimal tear production, covering the eye will keep the cornea moist and protected.

If there is reduced tear production and the eye is not surgically flapped then artificial tears are useful and necessary in my opinion. I prefer to use tears that are hypotonic. Lacrimal stimulants such as pilocarpine per os or topical cyclosporin are worth trying.

Encouraging owners and groomers to place mineral oil or an ophthalmic ointment in the eye prior to bathing is worthwhile client education.

Acknowledgments

I gratefully acknowledge Alcon Canada for its generosity in providing financial support for the publication of the photographs in color. CVJ

References

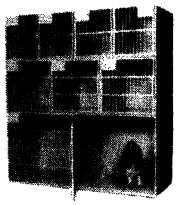
1. Kaswan RL, Salisbury MA. Current therapy of canine keratoconjunctivitis sicca and rationale for change in treating the causal factor with topical cyclosporine. Vet Focus 1989; 1: 98-104.
2. Vaughan D, Asbury T. General Ophthalmology. 11th ed. Los Altos, California: Lange Medical Publications, 1986: 58-62, 99, 340.
3. Pavan-Langston D. Burns and trauma. In: Manual of Ocular Diagnosis and Therapy. 2nd ed. Toronto: Little Brown and Company, 1986: 31-34, 109, 367.
4. American Academy of Ophthalmology. External diseases and cornea. In: Basic and Clinical Science Course. Section 7. San Francisco: American Academy of Ophthalmology, 1987: 130-133.
5. Deustch TA, Feller DB. Management of Ocular Injuries. Philadelphia: WB Saunders, 1985: 93-99.
6. Blogg RJ. The Eye in Veterinary Practice. Vol. 3. Eye injuries. Malvern, Australia: Chilcote Publishing, 1987: 59-61.
7. Peiffer RL, Stowe CM. Veterinary ophthalmic pharmacology. In: Gelatt K, ed. Textbook of Veterinary Ophthalmology. Philadelphia: Lea & Febiger, 1981: 362-363.
8. 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: 1110-1115.
9. Wilkie DA. Control of ocular inflammation. In: Millichamp NJ, Dziezyc J, eds. Vet Clin North Am [Small Anim Pract] 1990; 3: 693-715.
10. Wilkie D. Topical corticosteroidal and non-steroidal ophthalmic antiinflammatory agents. Vet Focus 1989; 1: 105-107.
11. Dice P, Cooley P. Use of contact lenses to treat corneal diseases in small animals. Semin Vet Med Surg 1988; 3: 46-51.
12. Shofner RS, Kaufman HE, Hill JM. New horizons in ocular drug delivery. In: Zimmerman TJ, Kooner KS, eds. Ophthalmol Clin North Am 1989; 2: 15-24.
13. Miller T. The effects of collagen shields on the reepithelialization of the normal canine cornea. Proc Am Coll Vet Ophthalmol Meet Phoenix, Arizona 1990: 26-28.

Need Cages?

Save 33 1/3%

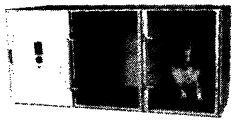
Clark Cages

- Formica Lined
- Quiet & Warm
- Sensibly Priced
- Durable
- Attractive
- Easy To Assemble



Clark Cage Dryer

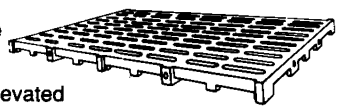
- Heavy-duty construction
- Fully assembled



- Virtually maintenance-free
- Dries animal quickly and safely
- Space-saver design

Thrifty Cage and Run Floors

- Lightweight polypropylene plastic panels
- Easy-to-clean
- Cage floors elevated on 3/4" legs
- 2' x 2' panels snap together for large run floor
- Available in custom sizes



For a free colour brochure and price list contact:

Clark Cages Inc. *Committed To Excellence*

Canada (705) 474-8303
USA Texas (214) 744-5954
Penn. (215) 922-5564
Calif. (213) 689-9324