Keratomalacia

Keratomalacia, commonly called “corneal melting,” is a process of degeneration and liquefaction of the corneal stoma. Keratomalacia frequently onsets following establishment of corneal infection and aggressive neutrophilic response to that infection. Certain bacteria organisms, mainly *Strep* and *Psuedomonas* species have the ability to secrete degenerative proteases and/or elastases that attack the collagen and glycosaminoglycan structural components of the cornea. These enzymes also have the ability to break-down intrinsic inhibitors of the enzymes present within the cornea. Large quantities of degenerative enzymes are also secreted by responding neutrophils as a response to the attacking bacterial organisms. This process sets-up a chain reaction in which corneal structural components are damaged at the same time remaining inhibitors of the process are eliminated and as such the degeneration accelerates. Keratomalacia is often a true ophthalmic emergency as the corneal stroma can be damaged by this process so quickly that a seemingly superficial ulcer can rapidly increase in depth to a descemetocele or even overt corneal rupture in a matter of hours.

The keys to treatment involve identifying the appearance of this process and conditions that develop the risk for onset. Corneal cytology is a key diagnostic element as it allows detection of substantial neutrophilic infiltrates and possibly large colonies of bacterial organisms. These findings should raise the “red flag” of alarm that risk for keratomalcia developing is imminent. Bacterial culture and sensitivity should be taken at the same time as cytology. Even though it will be a number of days until the results are back, the susceptibility pattern of the organisms
grown will be critical for successful management, especially in the case of poor or incomplete response to your initial empirically chosen antibiotic regimen.

Treatment typically involves frequent (q2-4h) application of broad spectrum topical antibiotics. Many ophthalmologists use a combination of at least two topical antibiotics to ensure capturing both the Gram+ coccal (Strep) and Gram– rod (Pseudomonas) groups of organisms. An inhibitor of the lytic enzymatic stromal breakdown is also critical. Perhaps one of the easiest and most readily available sources is the animal’s own steriley collected blood serum. Serum contains a number of molecules such as alpha-macroglobulin with activity against protease, collagenase, and elastase. Other inhibitor substitutes have been described including: acetylcysteine, EDTA, and tetracycline drugs. High-frequency application (q1-2h) is recommended for at least 48–72 hours or the keratomalacia process is detected to be abating. With improvement, slow tapering of medication application can be considered. Topical steroids are contraindicated due to local immunesuppression and inhibition of appropriate neutrophil activity. Topical NSAID application is controversial. Clinical research in human patients suggests NSAID may actually potentiate the degenerative process in some cases and advise against use. However, some veterinary ophthalmologists believe an overly aggressive inflammatory response within the cornea establishes the environment that allows onset of keratomalacia and; therefore, believe the small risk of potentiation of degeneration is actually outweighed by the tempering of the intrasotrmal inflammation provided by topical NSAID.

Patients with keratomalacia should be monitored very closely (i.e., q12-24h) due to the possibility of rapid progression. If progression occurs in the face of appropriate and aggressive medical treatment, the risk of corneal rupture dramatically increases. At that point, surgical intervention with keratectomy to debride inflammatory material and subsequent pedicle conjunctival grafting to provide tectonic and vascular support may become essential.

**Glaucoma**

Glaucoma is a disease resulting in progressive vision loss due to the dysfunction and death of retinal ganglion cells, precipitated by an abnormal elevation of intraocular pressure. Multiple conditions, both primary and secondary, can result in the onset of glaucoma. However, all have a common mechanism of onset, obstruction of the outflow of aqueous humor from the eye. Ongoing production of aqueous humor in the face of impaired outflow results in elevation of intraocular pressure. During periods of elevated intraocular pressure, blood flow within retinal and choroidal vessels is greatly reduced. Similarly axoplasmic flow within ganglion cell axons in the retinal nerve fiber layer and optic nerve head is severely impaired. It is thought that a combination of ischemia due to diminished blood supply and accumulation of toxic metabolites from restricted axoplasmic flow result in initial ganglion cell dysfunction and acute vision loss.
If intraocular pressure remains elevated over time, irreversible injury results in ganglion cell death. Ganglion cell demise contributes to uncontrolled release of potent neurotransmitters and other sequestered intracellular chemicals. Nonspecific release and diffusion of these toxic agents can establish a devastating chain reaction in which adjacent retinal cells are damaged and subsequently release further toxic substances. This self-perpetuating amplification process leads to the progressive retinal degeneration and permanent vision loss seen in chronic glaucoma patients. Time is; therefore, of utmost importance when managing animals with glaucoma. Emergency reduction of elevated intraocular pressure can mean the difference between restoration of visual function and irreversible blindness.

Primary

Primary glaucoma results from a congenital abnormality in formation of the aqueous humor outflow pathways and may or may not be heritable. Malformation of the outflow pathways occurs bilaterally almost invariably; however, onset of clinical signs is commonly asymmetric with one eye developing elevated intraocular pressure a few months to even a few years prior to the contralateral eye. Primary glaucoma occurs without evidence of additional acquired disease (i.e., inflammation or neoplasia). Primary glaucoma is most often noted in the dog; it is much more rare in the cat or horse. Interestingly, although outflow pathway abnormalities occur congenitally, clinical signs of glaucoma in the dog are not typically seen at birth, developing rather in middle-age. By far, the most common etiology of primary glaucoma in dogs is goniodysgenesis. Goniodysgenesis manifests as a grossly apparent (via gonioscopy) congenital malformation or narrowing of structures important in the conventional aqueous humor outflow pathway. Animals that develop primary glaucoma due to goniodysgenesis commonly present with an acute onset of markedly elevated intraocular pressure and blindness. The vision loss can quickly become permanent if immediate therapy is not pursued. Long-term management of these dogs can be difficult, with a majority ultimately developing permanent blindness, even in the face of aggressive medical and/or surgical intervention. Although a significant cause of blindness in the adult human population of the United States, primary open-angle (non-goniodysgenic) glaucoma is rare in other species. With the open-angle form of primary glaucoma, elevation of intraocular pressure occurs over an extended period of time and vision-loss is more insidious.

Specific breeds (i.e., American Cocker Spaniel, Basset hound, Chow chow, Shar pei, Boston Terrier, Wire Fox Terrier, Norwegian Elkhound, Siberian Husky, Cairn Terrier, Miniature Poodle, and many others) show significant and likely heritable predisposition to the development of primary glaucoma.
Secondary

Secondary glaucoma occurs in association with or following an antecedent ocular disease or traumatic injury. With secondary glaucoma the aqueous humor outflow pathways are functioning adequately prior to onset of the inciting event. However, the inciting etiology results in compromised function of the outflow pathways and subsequent elevation of intraocular pressure. Secondary glaucoma is a significant cause of blindness in all species. Examples of common inciting causes include:

1) Iridocyclitis: Chronic anterior uveal inflammation can lead to secondary glaucoma via two separate mechanisms. Firstly, congestion of the aqueous humor outflow pathways can occur through swelling of adjacent tissue, deposition of inflammatory debris and cells within the trabecular meshwork of the ciliary cleft, and extension of pre-iridal fibrovascular membranes over the pectinate ligament. Secondly, formation of 360° posterior synechiae (iris to lens adhesions) interrupts flow of aqueous humor through the pupillary aperture into the anterior chamber. The entrapped aqueous humor distends the iris forward resulting in iris bombé and eventual elevation of intraocular pressure. Iridocyclitis is likely the commonest inciting cause of secondary glaucoma across species.

2) Neoplasia: Tumors can cause secondary glaucoma via direct extension into the aqueous humor outflow pathways or by inducing inflammation and tissue adhesions.

3) Anterior lens luxation: An anteriorly displaced lens typically fills the anterior chamber, resulting in pupillary blockade. Aqueous humor accumulates posterior to the iris, leading to elevation of intraocular pressure.

4) Hyphema: Red blood cells that have hemorrhaged into the anterior chamber must exit the eye through the ciliary cleft. An excessive number of cells can accumulate within and occlude the trabecular meshwork; thereby, impairing outflow of aqueous humor.

5) Retinal detachment: Release of inflammatory mediators and neovascularization factors from a detached retina can induce formation of pre-iridal fibrovascular membranes. As these membranes grow across the pectinate ligament, they prevent entrance of aqueous humor into the ciliary cleft.

Medical Management of Glaucoma

Once the diagnosis of glaucoma is made rapid, decisive treatment is essential for restoration and preservation of vision. Unfortunately, early clinical signs and visual disturbances often go unnoticed by owners when the disease is easiest to treat. This can make subsequent medical intervention even more difficult. It is important to have a candid discussion early on with the owner about expectations and prognosis for the animal’s long-term visual outcome. Due to recent advances in pharmacologic technology, topical drugs have become the mainstay in both
emergency reduction of intraocular pressure and also long-term maintenance of controlled intraocular pressure. Systemically administered medications are often fraught with significant side effects that can easily be avoided with topical administration. Research in humans has shown that once glaucoma begins, the retina becomes susceptible to progression of retinal degeneration at lower intraocular pressures. This translates to mean simply striving to reduce intraocular pressure back to within the normal range may not be enough to preserve vision over the long-term. Rather the goal should be to maintain intraocular pressure at the lowest value possible. In people, this is typically achieved through combination therapy with multiple classes of glaucoma medications. Comparable observations will likely prove true in our veterinary patients. Unfortunately many of the best glaucoma medications are currently sold only as trademark products still under patent. Due to the expense, the importance of multiple drug therapy can be lost on the owner. We eagerly anticipate the arrival of efficacious generic drugs. Another important concept to grasp is prophylactic intervention for the contralateral eye of a dog diagnosed with primary glaucoma. An excellent study showed following initial diagnosis of primary glaucoma, the average time to onset of glaucoma in the opposite eye when left untreated was approximately 8 months. Whereas the average time to onset of glaucoma was increased to approximately 30 months when the opposite eye was started on a topical glaucoma medication prophylactically.

**Emergency**

Acute onset of glaucoma must be managed as an emergency until the intraocular pressure is well controlled. The eye must be carefully and frequently monitored to ensure response to the prescribed medications. DO NOT attempt to treat acute glaucoma by sending the animal home on medications for 1 week with a plan to refer to an ophthalmologist if the intraocular pressure remains elevated. In a majority of cases handled this way, vision is permanently lost by the time of referral. If you are not comfortable treating, call your local ophthalmologist and send the animal to the referral center immediately. There are only a few medications effective in lowering the dramatically elevated intraocular pressure commonly observed in animals with primary glaucoma.

1) Prostaglandin analogs: facilitate non-conventional or uveoscleral outflow of aqueous humor. The current drugs on the market do not appear to be efficacious in cats and efficacy is controversial in horses. Ocular side effects: significant miosis and exacerbation of any underlying uveitis. Systemic side effects: minimal. Contraindications: severe uveitis.

   Travoprost 0.004% OS (Travatan): 1 gtt to affected eye, intraocular pressure should decrease over the next hour. Can be repeated; however, if no affect on
intraocular pressure is noted at this point then an osmotic diuretic should be employed. Note: this medication does not require refrigeration.

Latanoprost 0.005 % OS (Xalatan): 1 gtt to affected eye, intraocular pressure should decrease over the next hour. Can be repeated; however, if no affect on intraocular pressure is noted at this point then an osmotic diuretic should be employed. Note: this medication requires refrigeration.

2) Osmotic diuretics: establish an osmotic gradient between the plasma and the intraocular space. As fluid flows across this gradient into the bloodstream, dehydration of the vitreous humor results in a rapid reduction of intraocular pressure. Water must be withheld from the patient for 1–2 hours to ensure maintenance of the osmotic effect. The osmotic diuretics require an intact blood-ocular barrier for maximal efficacy. Ocular side effects: minimal. Systemic side effects: fluid and electrolyte imbalances, pulmonary edema, congestive heart failure. Contraindications: cardiopulmonary or renal dysfunction, intracranial hemorrhage.

Mannitol 20% IV (generic): 1 g/kg IV infused through micro-pore filter over 15–20 minutes. Intraocular pressure should decrease over the next hour. Can be repeated; however, if no affect on intraocular pressure is noted at this point then referral for emergency surgery is indicated.

Glycerol 50% oral solution: 1 g/kg PO. Intraocular pressure should decrease over the next hour. Can be repeated; however, if no affect on intraocular pressure is noted at this point then referral for emergency surgery is indicated. Vomiting is a common side effect. Note: this drug should not be administered to diabetic animals

Maintenance

There are a number medication classes that are effective to help maintain lower intraocular pressure over time once it has been reduced in an acute crisis. Generally the topical medications are currently preferred due to reduced systemic side effect profile. The most commonly utilized medications will be discussed below.

1) Prostaglandin analogs: facilitate non-conventional or uveoscleral outflow of aqueous humor. The current drugs on the market do not appear to be efficacious in cats and efficacy is controversial in horses. Ocular side effects: significant miosis and exacerbation of any underlying uveitis. Systemic side effects: minimal. Contraindications: severe uveitis.
Travoprost 0.004% OS (Travatan): 1 gtt q12-24h. Note: this medication does not require refrigeration.

Latanoprost 0.005% OS (Xalatan): 1 gtt q12-24h. Note: this medication requires refrigeration.

2) Carbonic anhydrase inhibitors: decrease aqueous humor formation by reducing the availability of bicarbonate and hydrogen ions free in the ciliary body for transport with sodium and chloride. Ocular side effects: minimal. Systemic side effects: (topical) minimal; (oral) vomiting, diarrhea, metabolic acidosis. Contraindications: hepatic disease, renal or adrenocortical insufficiency, electrolyte imbalance.

Dorzolamide HCl 2% OS (Trusopt): 1 gtt q8h. Conjunctival irritation and a burning sensation have been occasionally reported. Note: this is a good drug for prophylactic management of the contralateral eye in cases of primary glaucoma.

Brinzolamide 1% OS (Azopt): 1 gtt q8h. Note: this is a good drug for prophylactic management of the contralateral eye in cases of primary glaucoma.

Methazolamide 25–50 mg tablets (generic): 2.5–5 mg/kg PO q12h

3) Beta-blockers: decrease aqueous humor formation, although the mechanism is not entirely understood. In veterinary medicine these drugs have a limited affect on intraocular pressure when used alone, but have an additive affect when used in combination therapy. Ocular side effects: minimal. Systemic side effects: bradycardia, hypotension, bronchospasm. Contraindications: cardiopulmonary dysfunction.

Timolol maleate 0.5% OS (generic): 1 gtt q12h.

Timolol maleate/dorzolamide HCl OS (Cosopt): 1 gtt q12-8h. Note: this is a good drug combination for prophylactic management of the contralateral eye in cases of primary glaucoma.

Currently at Michigan State University, combination topical therapy with a prostaglandin analog, carbonic anhydrase inhibitor, and beta-blocker is considered the gold standard for medical management of controlled primary glaucoma in the dog. This combination is thought to have the greatest capacity to maintain intraocular pressure at the lowest possible value. In
cases of secondary glaucoma topical prostaglandin analogs are used sparingly due to the risk of exacerbating any uveitis that may be present. Since prostaglandin analogs appear less effective in cats and horses, combination topical treatment with a carbonic anhydrase inhibitor and beta-blocker seems to be the best medical management protocol.

Other drugs

The following groups of drugs are available for direct purchase or through a compounding pharmacy. Many of these medications have indications in human medicine; however, in veterinary medicine they are either less efficacious or have a greater potential for side effects.

1) Parasympathomimetics: facilitate aqueous humor outflow through the conventional pathway. Both direct-acting cholinergic agonists and indirect-acting cholinesterase inhibitors are available. Ocular side effects: miosis. Systemic side effects: parasympathetic affects: ptalism, lacrimation, urination, defecation, vomiting. Contraindications: may interfere with prostaglandin analog efficacy, pupillary obstruction glaucoma, may have additive effects with cholinesterase inhibitor flea control products

   Pilocarpine HCl 2% OS (generic): 1 gtt q8h. Direct-acting
   Demecarium bromide 0.125% OS (compounded): 1 gtt q12-48h. Indirect-acting
   Note: this drug has been described for use in the prophylactic management of the contralateral eye in cases of primary glaucoma


   Apraclonidine HCl 0.5 % OS (Iopidine): 1 gtt q8h.


   Dipivefrin HCl 0.1% (Propine): 1 gtt q8-12h
Surgical Management of Glaucoma

Surgical intervention is often required to control intraocular pressure. It is important to realize, even with the new classes of topical drugs available, medical management of glaucoma is usually only temporarily effective. Over time most veterinary patients will go blind with medical management alone. Detailed client communication concerning the expected disease course and prognosis for vision is essential when an animal is originally diagnosed with glaucoma. Surgery is most successful when the retina is not irreversibly damaged by chronic elevation in intraocular pressure. If the owner elects to pursue surgical intervention, referral to a veterinary ophthalmologist should occur on an emergency basis or as soon as possible once the intraocular pressure is controlled. The owner needs to be aware, rarely will surgery eliminate the need for glaucoma medications. Aggressive medical management in combination with surgery provides the best chance for long-term vision in the glaucoma patient. Surgery should not be pursued simply as an alternative for an owner who is non-compliant with drug administration. Once permanent vision loss occurs, the primary objective of surgery becomes palliation of the animal and reduction of medication application in the blind eye.

Surgery for sighted eyes

1) Cyclophotoablation: this procedure utilizes laser energy (diode is most common) to damage a portion of the ciliary epithelium in order to decrease aqueous humor production. Two delivery methods are currently being utilized in veterinary medicine. Most veterinary ophthalmologists use a transcleral laser probe positioned on the outside of the eye to direct the laser energy to the ciliary body. The probe is repositioned at multiple spots on the globe to deliver a predetermined quantity of light energy. More recently, some ophthalmologists have invested in endolaser probes. The probe is placed inside the globe through a sclerotomy site. The advantage of this approach is both the ciliary body and the resulting laser-induced damage can be directly visualized. A cryo-probe can also be used; however, the inflammation induced by the thermal energy traveling though the adjacent tissue is significantly greater than that generated by light energy. Cryoabaltion techniques are not typically recommended in sighted eyes. A major complication of all these ciliary ablation techniques is regeneration of the ciliary epithelium over time. It is common that intraocular pressure will begin to rise as the epithelium regrows and cyclophotoablation will have to be repeated.

2) Anterior chamber shunts: provide an alternate drainage pathway for aqueous humor to exit the eye. One end of a length of silicone tubing is inserted through a sclerotomy site into the anterior chamber. The alternate end is placed into the subconjunctival connective tissue or into the frontal sinus of the skull (other sites have been described). The
advantage of this procedure, if successful, normal intraocular pressure may be achieved indefinitely. However, both the uveal tract and connective tissue in various locations are exceeding adept at resisting the flow of aqueous humor. Frequently fibrinolytics or chemotherapeutic agents are used repetitively to maintain patency of the shunt. Obviously, intraocular pressure rises rapidly if the shunt becomes permanently occluded. The shunt may also provide an entrance route for pathogens causing endophthalmitis.

Surgery for blind eyes

1) Enucleation: is a straightforward procedure with minimal complications that provides globe and periocular glandular material extraction with apposition of the eyelid margins. A silicone orbital prosthesis may be placed to reduce the “sunken” appearance of eyelid tissue distortion into the orbital space over time.

2) Evisceration with intrasceral prosthesis: is a more complicated procedure that results in extraction of the uveal tract, lens, vitreous, and retina from the outer corneoscleral shell. A silicone prosthesis can be inserted into the corneoscleral shell to maintain the approximate original size of the globe. This procedure is appealing to owners who can not bear the thought of a pet absent an eye. However, it is important to realize the complication rate for evisceration is higher than that for enucleation. The eyelid margins, conjunctiva, and cornea remain intact and these sites may be affected by adnexal and corneal disease processes over time.

3) Chemical ciliary ablation: involves intravitreal injection of a pharmacologic agent that is toxic to the ciliary epithelium. The most commonly utilized agent is gentamicin, which is also toxic to the retina and lens epithelium. This technique is designed for carefully selected glaucoma cases with irreversibly blind eyes in which a definitive cause has been determined and neoplasia or endophthalmitis are not present. Typically an empirical dose of gentamicin is used. The procedure may need to be repeated if the intraocular pressure remains elevated or phthisis bulbi may result if all of the ciliary epithelium is destroyed. It is recommended that this technique be performed under general anesthesia due to the intense pain experienced by the animal immediately after injection. Chemical ablation should not be performed in cats as risk for subsequent intraocular sarcomas has been described. Disease processes affecting any ocular or periocular structure remain a concern over time.
Eosinophilic Keratitis

Eosinophilic keratitis (EK) is an inflammatory disease of the feline cornea (a similar condition is described in the horse; however, we will focus on the cat here). The inciting etiology for EK remains unknown; however, it frequently occurs in cats with a chronic history of ocular and respiratory signs consistent with repetitive shedding of Feline Herpes Virus type 1 (FHV-1). It is hypothesized that an accumulation of large quantities of viral antigen within the cornea perpetuates an immune reaction to the non-host antigen. Alternate theories have also been put forward.

Typically EK falls into the category of a “non-ulcerative” keratitis, yet when the condition is aggressive, chronic non-healing corneal ulceration can develop. Most commonly EK manifests as vascularization and cellular infiltrates adjacent to the limbus. This appears similar to granulation tissue, but often has a plaque-like white to yellow surface “crust”. Frequently conjunctival injection, epiphora, squinting and elevation of the nictitans accompany the onset of EK. Left untreated, the inflammatory lesions can migrate axially and laterally to include large areas of the corneal surface. In rare instances these infiltrates may even significantly affect vision in a compromised eye due to opacification of the cornea. EK often begins unilaterally, but development of bilateral disease remains a risk in affected cats.

Although a strong clinical suspicion may arise due to appearance of lesions in a presenting cat patient, confirmation is achieved through cytology captured from the “crust” of the invading inflammatory infiltrate. Cytology specimens usually consist of mixed inflammatory cell populations: lymphocytes, plasma-cells, non-degenerate neutrophils, and epithelial cells; while frequently not the most abundant cells on the slide the disease takes its name from a regular appearance of eosinophils within the sample. Eosinophils are commonly seen at a density of 1 cell per 2–4 high-power fields (100× objective); mast cells may also be observed at a similar cellular density. Note as intracellular granules do not always stain appropriately in “Diff-Quick”-type stains, sometimes careful screening of cells with eccentrically place nuclei will allow detection of their presence even if the granule color is quite pale.

Once an EK diagnosis is confirmed, it typically responds quickly to application of topical anti-inflammatory agents. Classically topical steroids such as neomycin-polymixin-dexamethasone or prednisolone acetate ophthalmic preparations at frequencies of q12-6h (based on severity of lesions) were employed, and these are still very effective. More recently use of topical cyclosporine has been described for management of EK at frequencies of q12-8h. Subjectively I find that topical steroids often control the inflammatory infiltrates more quickly (2–3 weeks) than topical cyclosporine (4–5 weeks), yet I lack any controlled clinical trial data to confirm this observation. When the infiltrates are more severe, I sometimes prescribe both a topical steroid and topical cyclosporine and taper the topical steroid in a few weeks as the lesions begin to
regress. While EK is readily controllable, it is infrequently curable once a cat has had an initial diagnosis. Yet frequency of “flare-ups” can vary widely among cats from once every few years to every few months. In newly diagnosed cases, I often begin to taper medication administration slowly until I am using topical cyclosporine every other day or a few times weekly. If inflammatory lesions remain “at bay” and the cat has had no intervening instances of upper respiratory signs, I give the owner the option to discontinue use of the topical anti-inflammatory medications. Then we assess the interval between the next reoccurrence. When recurrence occurs more frequently (2–3 times yearly), I typically recommend maintaining the cat on topical cyclosporine indefinitely and tailor the frequency of administration to the lowest frequency required to prevent “flare-ups” (usually EOD). When recurrence occurs less frequently (once per year or less), I give the owner the option of treating indefinitely as list above or returning when a suspected “flare-up” is occurring (confirming with cytology that the diagnosis is again correct) and reinstating 4–6 weeks of tapering therapy.

Systemic megestrol acetate administration has previously been described for control of EK. However, with the efficacy of topically administered anti-inflammatory agents and the possible side-effects associated with megestrol use, I do not commonly recommend use of this therapeutic option. As mentioned above with more aggressive forms of EK (frequently recurring, poorly responsive to initial medications, rapidly progressive, etc) simultaneous corneal ulceration is a possible presenting complication. Cats may also have concurrent upper respiratory signs. In these cases, I tend to have initial reluctance to “diving” right for the topical steroids and/or cyclosporine due to the concern of local immunosuppression that may exacerbate or prolong a FHV-1 shedding event and slow corneal re-epithelialization of the ulcerative lesion. Based on the severity of the upper respiratory disease or ocular signs in the contralateral eye consistent with FHV-1, a course of topical and/or systemic antiviral agents may be indicated. In these cases I have had success using the topical mast-cell stabilizer lodoxamide for management of the EK in the face of corneal ulceration which is managed with a topical antibiotic. Unfortunately lodoxamide is expensive and I tend to reserve it for these special cases and defer to the cheaper topical steroids or topical cyclosporine in more straightforward cases. When EK is excessively persistent or frequently recurrent a discussion about stress management for the cat is often helpful at decreasing the severity of events and increasing the frequency between “flare-ups.” This seems to be especially the case in multi-cat households where frequent dominance behavior utilized to maintain the “proper pecking order” can keep less aggressive cats in a constant state of physiological stress. Providing “safe-space” for these cats to eat, drink, use the litter-box and rest without risk of “bullying” by the dominant cat can go a long way to reducing aggressive flare-ups of EK.