CORNEAL ANATOMY AND PHYSIOLOGY

A. Anterior portion of the fibrous tunic
   a. Composed of dense connective tissue arranged in a regular lamellar pattern.
   b. Avascular
   c. Normal corneal thickness in cats and dogs is approximately 0.55-0.6 mm and thickness increases with age and weight of the animal.
   d. Horizontal corneal diameter: canine 13-17 mm, feline 17 mm
   e. Vertical corneal diameter: canine 12-16 mm, feline 16 mm
   f. Sensory innervation by ophthalmic branch of trigeminal nerve in epithelium and anterior stroma
   g. Transparency maintained by the following mechanisms:
      1. Regular lamellar pattern
      2. Corneal dehydration (deturgescence) maintained by a) physiological pump of the posterior endothelium and b) mechanical barriers of the epithelium and endothelium
      3. Non-keratinized anterior surface epithelium
      4. Small diameter of the collagen fibrils
   h. Corneal function: most powerful refractive surface of the eye and bends light in order to focus the image onto the retina.

B. **Four Corneal Layers**: Epithelium, Stroma, Descemet’s membrane, Endothelium.
   a. **Epithelium** consists of 8-15 cell layers of non-keratinized squamous epithelial cells that are contiguous with the conjunctival epithelium. Basal cells lie on a thin basement membrane and underlie several layers of polyhedral or wing cells. Two to three layers of nonkeratinized superficial cells are continuous with the conjunctival epithelium. Turnover rate from basal to superficial cells is 7 days.
      i. Epithelium is lipophilic/hydrophobic, therefore impedes passage of fluid and hydrophilic drugs.
b. **Stroma** (substantia propria) forms the bulk of the cornea (90%)  
   i. Extracellular matrix and parallel lamellar arrangement of collagen fibrils separated by less than a wavelength of light.  
   ii. Keratocytes interwoven between the collagen fibrils and extracellular matrix.  
      1. Possess cellular extensions that help to maintain the integrity of the stromal lamellae.  
      2. After deep corneal injury, keratocytes differentiate into fibroblasts and contribute to scar formation.  
   iii. Stroma is hydrophilic so absorbs water and fluorescein dye.

c. **Descemet’s membrane** - located on the posterior surface of the cornea, is an acellular basement membrane that functions as a protective boundary within the cornea and thickens as it is produced throughout life by the corneal endothelium.

d. **Corneal endothelium** is a monolayer of flattened polygonal cells lining the posterior aspect of the cornea.  
   i. Metabolic (sodium-potassium-ATPase) pumps function to maintain a state of deturgescence (relative dehydration) in the corneal stroma to maintain the optical clarity.  
   ii. In the adult, the endothelium rarely undergoes mitosis and there is an age-dependent loss of endothelial cells. Cells enlarge and spread to compensate for loss of neighboring cells.  
   iii. Endothelium also serves as a mechanical barrier to fluid absorption by stroma

C. Corneal response to injury and disease

1. **Corneal ulceration** usually occurs secondary to trauma and will result in edema.  
2. **Focal edema** is usually due to an epithelial defect and **diffuse edema** is usually due to an endothelial defect.  
3. **Vascularization** is a nonspecific change. Cornea will vascularize at the depth of the lesion.  
   a. Superficial vascularization = branching vessels that cross the limbus  
   b. Deep vessels = straight brush border appearance and do not cross the limbus  
   c. Vascularization begins 24-72 hours after an insult or injury and then continues at a maximum of one millimeter per day thereafter.
4. **Pigmentation** is a nonspecific change that is due to chronic irritation, but can be a result of specific diseases such as chronic superficial keratitis (pannus).

5. **Scarring and granulation tissue** are usually due to injuries that penetrate the stroma.

**D. Sequelae of corneal disease**

1. The axonal and oculopupillary reflexes result in conjunctival hyperemia, miosis (pupil constriction), ciliary muscle spasm (seen as blepharospasm and photophobia), increased protein levels in the aqueous humor (aqueous flare).

2. Topical atropine alleviates some of the discomfort by paralyzing the iris and ciliary body muscles.

**E. Normal corneal wound healing**

1. With **superficial epithelial defects**, basal cells migrate to cover the defect within one hour of the injury, and within 24 to 48 hours mitosis begins to replenish cell loss.

2. **Uncomplicated corneal ulcers** will heal within 5 to 7 days unless the underlying etiology persists, the ulcer is infected or it has a basement membrane defect (indolent ulcer).

3. **Stromal wound healing** is slower and involves epithelial sliding and stromal replacement. Initially there is edema at the site of epithelial loss until the defect has re-epithelialized. Fibroblast proliferation provides structural support for the stromal defect.

4. Defects resulting from **full thickness ulcers or lacerations** are sealed initially with fibrin from blood-aqueous-barrier breakdown. The surrounding epithelium begins to replicate. Then, neutrophils infiltrate the wound. Keratocytes are converted to fibroblasts that produce collagen and invade the fibrin plug in three to six days. Collagen and glycosaminoglycan synthesis and collagenase simultaneously remodel the cornea. Lost stromal lamellae are replaced by fibrous scar tissue that may not be transparent.

5. **Corneal strength** is not regained for months-years following stromal injury.

6. **Corneal endothelial loss** is addressed by hypertrophy (increase in cell size) of the endothelial cells to retain their barrier effect. These cells are slowly lost with age and they do not undergo mitosis in the adult animal. If a significant number of endothelial cells are lost, diffuse corneal edema will develop.

7. **Corneal opacity** in response to disease or injury can be classified based on color.

   a. **White** opacities include edema, cellular infiltrate, calcium and lipid infiltrates and scars.
b. **Brown** opacities include pigmentation (all species) and sequestrum formation (cats only).

c. **Red** opacities include vascularization and intrastromal hemorrhage.

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**DIAGNOSIS AND TREATMENT OF CANINE ULCERATIVE CORNEAL DISEASE**

1. **Causes of corneal ulceration:**
   a. **Dogs:** corneal ulcers are usually the result of trauma, keratoconjunctivitis sicca (KCS aka. “Dry Eye”), corneal degeneration, bullous keratopathy secondary to endothelial dystrophy or degeneration or indolent ulcers.
   b. **Cats:** corneal ulcers are usually caused by feline Herpesvirus-1 (FHV-1) infections

2. **Four types/degrees of ulcerative keratitis in small animals:** superficial, midstromal, deep (descemetocele), and melting corneal ulcers. Indolent ulcers are a type of superficial ulcer.

3. **Diagnosis:**
   a. **Initial evaluation requires determining the extent of corneal involvement and then performing the appropriate diagnostics and therapy.**
   b. **A Schirmer tear test** should be performed to rule out KCS in all cases of non-traumatic/non-perforated corneal ulceration.
      i. Normal values: ≥15 mm/minute
      ii. Greater than normal STT values are expected in cases of corneal ulceration/ocular pain due to increased reflex tearing; therefore, a normal STT in an eye with an ulcer may indicate KCS.
   c. **Corneal culture with susceptibility** is indicated in all cases where corneal infection is suspected based on clinical appearance and/or chronicity and in all cases of stromal involvement.
      i. Corneal culture may not be feasible due to risk of perforation in ulcers with greater than mid-stromal involvement or descemetoceles.
      ii. Corneal culture may not be feasible due to risk of wound leakage in perforated corneal ulcers.
      iii. Culture is best performed using a moistened culturette and before application of topical anesthetics to increase yield and viability of organisms.
   d. **Fluorescein stain** to determine the size of the ulcer and/or if the epithelium is intact.
      i. Best done before application of topical anesthetic
ii. Helpful to diagnose a descemetocoele (see below).

e. **Corneal cytology** is indicated in all cases where corneal infection is suspected based on clinical appearance and/or chronicity and in all cases of stromal involvement.
   i. May be performed after application of topical anesthetic.

4. **Superficial corneal ulcers** are caused by trauma to the epithelium due to injury, pre-existing eyelash disorders (distichiasis or ectopic cilia), lagophthalmos, KCS and trichiasis.
   a. Superficial ulcers are painful and clinical signs include blepharospasm, epiphora, and pawing at the eye.
   b. **Clinical appearance**: break in epithelium only, fluorescein positive, focal corneal edema, no divot or defect in corneal stroma.
      i. **Infected superficial ulcer**: same as above except increased pain, evidence of anterior uveitis, and white-yellow cellular infiltrate
   c. They generally heal within 72 hours unless the underlying etiology is not removed, the ulcer has become infected, or it is indolent.
   d. Therapy for uncomplicated corneal ulcers is broad-spectrum topical antibiotic solution or ointment (triple antibiotic QID) and topical atropine (SID to BID).
   e. **Topical corticosteroids are always contraindicated with corneal ulcers**!!

5. **Indolent/refractory ulcers** (a.k.a. Boxer ulcer, recurrent erosion) are a specific type of superficial corneal ulcer that result from failure of the epithelium to adhere to the underlying basement membrane.
   a. The Boxer breed is predisposed as are middle-aged to older dogs of any breed.
   b. Routine corneal ulcer therapy alone is not sufficient for these ulcers; they require debridement of the ulcer’s redundant epithelial edges and a multiple grid keratotomy (MGK) or diamond burr debridement +/- bandage soft contact lens placement.
      i. **Debridement and MGK Procedure**: Quiet room, good animal restraint, topical anesthetic, dry cotton-tipped swabs to debride epithelium, 25-gauge needle using beveled edge held at a 30 degree angle to the cornea to create the grid lines completely covering the ulcer and extending a small distance into the normal surrounding epithelium.
      ii. **Diamond burr debridement**: Quiet room, good animal restraint, topical anesthetic, dry cotton-tipped swabs to debride epithelium, Algerbrush II with
3.5 mm burr (fine or medium grit) gently rolled over entire ulcerated surface and margins of ulcer until no more loose epithelium can be removed.

iii. Place bandage soft contact lens to improve comfort and speed rate of healing.
   1. AcriVet – sized for variety of canine corneal diameters and curvature (expensive but since made for dogs, more likely to be retained throughout healing process)
   2. Bausch and Lomb Plano – 14 mm, 8.4 base curve (less expensive option but less likely to be retained throughout healing period)

iv. **Warn owner that dog’s eye may be much more uncomfortable** for 24-48 hours after the debridement/MGK procedure due to irritation of superficial corneal nerve endings.

v. Dogs with secondary entropion or severe blepharospasm will benefit from a temporary contact lens (do not leave in without cleaning for greater than 5 days).

vi. Topical antibiotic (Terramycin® ointment or triple antibiotic solution) q 8 hours and atropine (q 24-72 hours) are used to prevent secondary bacterial infection and control pain, respectively.
   1. Use of tetracyclines topically and/or orally has been associated with increased rate of healing of indolent/refractory ulcers *in-vitro* and in retrospective and prospective evaluation *in-vivo*
   2. May use oral doxycycline – 5 mg/kg PO q 12 hours until healed.

vii. Carprofen – 2.2 mg/kg PO q 12 hours x 5-7 days to improve comfort

c. Recheck ulcer every 5-7 days until healed. ≥ 90% are healed within 14 days.

d. Don’t repeat debridement and MGK for at least 2 weeks.

e. **Topical corticosteroids are always contraindicated with corneal ulcers**!!

6. **Midstromal corneal ulcers** are usually infected, painful, and have associated uveitis.
   a. **Clinical appearance:** visible divot or defect in stroma, usually more pronounced and extensive corneal edema, fluorescein-positive, white-yellow cellular infiltrate may be visible, anterior uveitis (miosis, refractory to pharmacologic mydriasis, aqueous flare)
   b. These ulcers should be evaluated by a **culture/sensitivity and cytology** performed.
      i. Diff-Quik to screen for neutrophils, rods and/or cocci bacteria
ii. If organisms found on cytology, culture and susceptibility should be submitted
iii. If cytology is negative for PMNs and organisms and no corneal cellular infiltrate is observed, culture is optional.
c. Ulcer must be monitored closely for progression to deeper ulcer or perforation –
   **Hospitalize patient or recheck at least every 24-48 hours!**
d. Initial topical antibiotic therapy should include **triple antibiotic** solution or ointment every 4 hours for 24 hours then q 4-6 hours until healed.
e. If this fails to stop progression, then change to a stronger antibiotic such as topical fluoroquinolone such as levofloxacin (Quixin®) or ofloxacin (Ocuflox®), ciprofloxacin (Ciloxan®) every 4-6 hours.
f. Topical atropine (q 24-72 hours) should be used to relieve pain
g. Tear replacement therapy if needed (TID to QID).
h. Most infected ulcers are caused either by opportunistic gram-positive organisms or *Pseudomonas* species.
i. The fluoroquinolones (see **Summary Canine Ulcer Treatment**) have excellent efficacy against *Pseudomonas* spp. as well as good gram-positive spectrums and are superior to gentamicin and tobramycin.
   i. Gentamicin has poor efficacy again gram-positive flora and is only ~85% effective again *Pseudomonas aeruginosa*; therefore, it is a poor choice for treatment of most corneal ulcers.
j. **Surgical management** is indicated for ulcers that have progressed beyond the mid-stroma. Surgery commonly involves debridement of affected stroma and placement of a conjunctival pedicle graft.

7. A **descemetocoele** is the deepest type of corneal ulcer and perforation is imminent.
a. **Clinical appearance:** Exposed Descemet’s membrane does not retain fluorescein and since it is basement membrane, it does not absorb fluid like the stroma and remains clear relative to the adjacent edematous stroma. There is always a visible divot around the descemetocoele.
   i. **Clinical hint:** Descemet’s membrane rarely bulges forward. Most often, observed bulging of the cornea is due to keratomalacia, edema or fibrin clot/uveal prolapse after corneal perforation.
b. Deep ulcers, descemetoceles and perforated corneal ulcers are ophthalmic emergencies that require immediate surgical intervention.

c. Conjunctival pedicle flaps with or without a tectonic (support) graft or corneoconjunctival transposition are the treatments of choice depending on the case.

d. **Antibiotic therapy:**
   i. Triple antibiotic solution is used first. If this is not effective as evidenced by progression of the ulcer or by cytology results, then changing to a fluoroquinolone solution is indicated until culture results are available.
   ii. Systemic antibiotics are indicated when a perforated corneal ulcer is present.
   iii. Topical atropine therapy is indicated for cycloplegia and ciliary spasm.
   iv. Ophthalmic **solutions** should be used in cases of perforation or impending perforation because ointments are extremely irritating to the intraocular structures.
   v. Third eyelid flaps are **contraindicated** for this or any other ocular disease in dogs and cats.

8. **Melting corneal ulcers** are rapidly progressive ulcers caused by enzymatic degradation of the stroma, usually due to bacterial infection with *Pseudomonas aeruginosa* or beta-hemolytic *Streptococcus* species or excessive collagenase activity induced by neutrophilic infiltrate.

   a. **Clinical appearance:** Severe corneal edema, variable degrees of ocular pain, pronounced swelling and malacia of the cornea resulting in a drooping or bulging appearance. Melting may commonly manifest in dogs as “drilled out” defects in the stroma rather than stromal swelling and bulging. Cellular infiltrate may or may not be apparent. Usually considerable anterior uveitis (aqueous flare, miosis, hypopyon).

   b. Medical therapy includes topical antibiotic therapy as described above, and topical atropine.

   c. Topical **anticollagenolytic agents** should be used (tetracycline family member topically or orally). The tetracycline family has been shown to chelate zinc and calcium, inhibit matrixmetalloproteinases and stabilizes collagenolytic activity.
      i. In dogs, can use oral doxycycline (5 mg/kg PO BID) in lieu of or in addition to topical tetracycline.
d. Surgery is often indicated and includes debridement of affected stroma and placement of a conjunctival pedicle graft. This condition is also an ophthalmic emergency.

9. **Corneal perforation** occurs after full-thickness progression of an ulcer through Descemet’s membrane and the endothelium or corneal laceration.
   
a. **Clinical appearance:** Deep corneal defect surrounding a protruding area of clotted fibrin/aqueous (usually tan-yellow +/- blood) or uveal tissue (tan to brown or black +/- hemorrhage) plugging the defect.
   
i. May visualize leakage of aqueous humor and/or blood through the defect.
   
ii. The anterior chamber usually appears shallow or collapsed.
   
iii. Dyscoria may be observed if the defect is plugged by an iris prolapse and the chamber is reformed.
   
iv. Siedel test: fluorescein dye is applied to the corneal surface. Dye penetrates into the anterior chamber making the aqueous green or clear aqueous leaking from the defect can be seen flowing in the green tear film.
   
b. **Prognostic factors** to consider before determining treatment:
   
i. Size of ulcer
      
1. Better prognosis with smaller defect to repair
   
ii. Health of surrounding cornea
      
1. Need some healthy surrounding corneal tissue to which to suture corneal and/or conjunctival graft
   
iii. Size of uveal prolapse
      
1. Smaller more acute prolapse generally has better prognosis than larger more chronic prolapse
   
iv. Anterior chamber
      
1. Formed, deeper AC indicates that the defect is not leaking and generally has a better prognosis
2. Hyphema, when extensive, indicates significant intraocular injury and therefore has a poorer prognosis
3. Fibrin in the AC is a normal response to trauma and leaking and helps to plug the defect. Extensive fibrin formation may be associated with a worse prognosis due to sequelae of synechia, cataract, etc.
4. Hypopyon suggests corneal +/- intraocular infection and may indicate a poorer prognosis.

5. When opacity of the anterior chamber such as blood, fibrin precludes examination of the lens and fundus, ocular ultrasonography is indicated.

v. Lens involvement
1. Surface visible, no evidence of capsule laceration or cataract – better prognosis
2. Surface not visible due to fibrin, blood – risk of capsule laceration
3. Lens capsule laceration/rupture – lens extraction is required to prevent cataract, severe lens-induced uveitis and secondary glaucoma

vi. Posterior segment health/prognosis for vision
1. Menace, dazzle (light perception) and pupillary light reflexes should always be evaluated.
   a. Absent menace may be due to pupil occlusion or opacity of the ocular media
   b. Absent direct and consensual PLR and dazzle reflex indicates severe damage to the retina +/- optic nerve; therefore prognosis is poor
   c. Presence of PLR and dazzle indicates at least 2-5% of the retina is functioning but is non-specific in predicting long-term quality of vision
2. Ocular ultrasonography – indicated when opacity of the ocular media precludes examination of the fundus
   a. Vitreal hemorrhage and/or retinal detachment – poor prognosis

vii. Patient/client factors – age/health of patient, financial ability of client, etc.

c. Treatment:
   i. Emergency referral for surgical therapy is recommended
   ii. Recommend placing an Elizabethan collar to prevent further ocular trauma
   iii. Ophthalmic solutions should be used in cases of perforation because ointments are extremely irritating to the intraocular structures.
iv. Systemic antibiotics and non-steroidal anti-inflammatories are indicated
v. Surgical options include corneoconjunctival transposition, corneal transplantation and conjunctival pedical grafting
vi. For cases with a poor prognosis for vision or globe salvage, enucleation must be considered.

**DIAGNOSIS AND TREATMENT OF NON-ULCERATIVE CORNEAL DISEASE**

1. **Chronic Superficial Keratitis** (a.k.a. Pannus) is a progressive immune-mediated disease with lymphocytic-plasmacytic cellular infiltrate, pigmentation and vascularization.
   a. German shepherd dogs and Greyhounds are predisposed but any dog can be affected.
   b. Pannus begins at the temporal or inferotemporal limbus and progresses centrally.
   c. If not treated appropriately, it can infiltrate the entire cornea.
   d. In some dogs the third eyelid can also be involved (called “plasmoma”).
      1. Clinical signs of plasmoma are hyperemia, depigmentation +/- irregularity of leading edge of nictitans and grey-pink lymphopasmacytic nodules.
   e. The clinical signs, signalment and cytology specimens that yield lymphocytes and plasma cells will confirm the diagnosis.
   f. **Therapy:**
      1. Topical corticosteroids initially three to four times daily (0.1% dexamethasone or 1% prednisolone acetate) then weaned over 4-8 weeks to treatment q 48 hours, then eventually discontinued.
      2. Maintained with topical 0.2% cyclosporine (Optimmune®, Schering Plough), 2% cyclosporine compounded in oil or 0.02% tacrolimus compounded in solution or ointment twice daily.
      3. Consider canine sunglasses, Doggles®, to reduce UV light exposure to the cornea ([www.doggles.com](http://www.doggles.com))
      4. Rarely, topical medication is not effective and subconjunctival corticosteroids, oral corticosteroids (prednisone at the anti-inflammatory dose) or beta irradiation (i.e. strontium 90) may be necessary.
   g. Pannus is more difficult to treat in dogs living at higher altitudes due to increased UV light exposure.
   h. Disease is **not curable** but can be adequately controlled.

2. **Nodular granulomatous episclerokeratitis** (NGEK)
a. Immune-mediated lymphoplasmacytic and histiocytic infiltration of episclera and cornea
b. Collie and Cocker spaniels predisposed
c. Clinical signs: episcleral injection and thickening +/- nodular appearance, peripheral corneal vascularization and edema
d. Treatment: topical corticosteroid such as 0.1% dexamethasone or 1% prednisolone acetate q 6-8 hours, then wean in addition to Optimmune or 1% or 2% cyclosporine or 0.2% tacrolimus q 12 hours for long-term maintenance therapy.
e. Prognosis is good for control but usually requires life-long therapy

3. Pigmentary keratitis

a. Progressive pigmentation, vascularization and fibrosis of the cornea originating at the medial limbus
b. Pugs
c. Occurs due to medial canthal entropion/trichiasis
d. Treatment: Optimmune, 1% or 1% or 2% cyclosporine or 0.2% tacrolimus q 12-24 hours for long-term maintenance therapy
e. Severe cases require medial canthoplasty to eliminate the trichiasis
f. Prognosis is good for control but usually requires life-long therapy

4. Corneal dystrophy

a. Inherited lipid deposits, usually bilateral, axial to paraxial, round to oval
b. Non-progressive
c. No treatment indicated

5. Corneal degeneration

a. Lipid and calcium forms
b. Causes: systemic hyperlipidemia or hypercholesterolemia, corneal disease
c. Treatment: EDTA may bind calcium and improve calcium degeneration; tear supplements may improve both forms
CANINE ULCER MANAGEMENT SUMMARY

Diagnostics:

1) Preliminary examination
   a. Determine depth of ulcer: epithelial, stromal, desmetocele or rupture/full-thickness
   b. Evaluate for signs of infection: yellow-white cellular infiltrate, excessive pain, uveitis (miotic pupil), stromal loss or malacia (melting), active corneal vessels
   c. Rule out conjunctival foreign body or other predisposing causes of mechanical trauma (distichia, ectopic cilia, entropion, etc.)

2) Culture and susceptibility for aerobic bacteria (moisten sterile swab w/ sterile saline first to improve yield of organisms or break vial at bottom of prepared culturette w/ Stewart’s transport medium).

3) Schirmer tear test to rule out keratoconjunctivitis sicca
   a. Normal ≥ 15 mm/minute
   b. In the presence of an ulcer/ocular pain, reflex tearing should result in an increased STT in a normal eye.

4) Fluorescein stain
   a. To determine size of the ulcer and/or if the epithelium is intact
   b. Also useful to diagnose a desmetocele—Descemet’s membrane is basement membrane and does not stain while corneal stroma does. Descemet’s membrane is clear and does not imbibe the stain while exposed stroma is cloudy/edematous and stains green.

5) Topical anesthetic then cytology to look for bacteria and fungi (Diff-Quik)

Antibacterials:

1) Triple antibiotic ophthalmic ointment or solution – Sig: 1/8” q 4-6 hours
   Cost ≥ $15-30
   **Broad-spectrum – good 1st choice for infected ulcer or prophylactic therapy

2) Ciprofloxacin ophthalmic oint or soln. (Ciloxan) – Sig: 1/8” (or 1 drop) q 4-6 hours
   Cost ≥ $50-55 per 5 ml; $45-50 per 5 gm tube ointment
   **Penetrates intact epithelium therefore used to treat stromal abscesses

3) Ofloxacin ophthalmic soln. (Ocuflox) – Sig: 1 drop q 4-6 hours
   Cost ≥ $30 per 5 ml
   **Penetrates intact epithelium and reaches therapeutic levels in aqueous humor better than Ciloxan

4) Levofloxacin soln. (Quixin) – Sig: 1 drop q 6 hours
   Cost ≥ $45
   **Penetrates corneal epithelium 6X better than Ciloxan
Anticollagenases:

1) Autogenous serum – Sig: 0.1 ml q 4 hours (discard after 48-72 hours)

2) Oxytetracycline oint (Terramycin) – Sig: 1/8” q 6-8 hours

   **Has anti-collagenase and immune modulating properties**

3) Doxycycline – Sig: 5 mg/kg PO BID until melting resolves

Anti-inflammatories

1) Carprofen (Rimadyl) - Sig: 2 mg/kg orally q 12-24 hours

2) Diclofenac sodium (Voltaren) – Sig: 1 drop q 8-12 hours

Mydriatic:

1) Atropine 1% solution or ointment

   Sig: 1/8” or 1 drop q 8-12 hours initially or as needed to keep pupil dilated.

   **Not recommended to use more frequently than q 8-12 hours
   **If uveitis is well controlled, pupil may stay dilated ≥48 hours from a single application.

Antifungals (Fungal ulcers in dogs and cats are RARE):

1) Miconazole 1% or 2% ointment - Sig: 1/8” q 6 hours

   Cost $15-20

   **Avail: Wedgewood Pharmacy 800-331-8272 or other compounding pharmacies

2) Voriconazole 1% solution (V-FEND) – Sig: 1 drop q 6 hours

   Cost $180 / 20 ml

   Reconstitute a 200 mg vial of powder for IV injection with 19 ml sterile water.

   Does not need to be refrigerated. Broad spectrum of efficacy.

   **Avail: Wedgewood Pharmacy 800-331-8272 or other compounding pharmacies

Candidates for Surgical Therapy:

-Depth of ulcer near or greater than 50% stromal depth

-Desmetoceles

-Perforated ulcers

-Melting ulcers

-Corneal lacerations

-Stromal abscesses, especially in axial cornea, no vessels approaching and/or if refractory to therapy

A. Metzler 10/14