

+ CORNEAL ULCERS IN DOGS

DIAGNOSE AND TREAT CORNEAL ULCERS

UPTAKE OF FLUORESCEIN TO THE CORNEA

UNCOMPLICATED ULCER

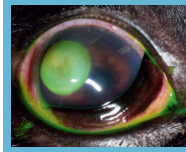
Superficial defects should heal within hours to days with appropriate treatment!

Treatment consists of supportive medical therapy:

1. **Topical antimicrobial therapy**
 - a. Broad spectrum; minimal epithelial cell toxicity is best
2. **Ocular Lubricant**
 - a. **Oculenis™ BioHance™ Ocular Repair Gel:** 1 to 2 drops BID (or as needed based on clinical appearance/response).
3. **Analgesia**
 - a. Oral NSAIDs if tolerated +/- gabapentin or opioids PRN
 - b. Topical atropine 1% if iridial spasm present – use no more than BID for 1-2 days. Aim for mid-dilated pupil size. Take care in small patients due to systemic side effects. Take care in patients with KCS (will reduce tear production).
 - c. Avoid topical anesthetics (epitheliotoxic) and corticosteroids due to delayed healing/corneal melting (from induction of host collagenolytic enzymes)
4. **Hard plastic Elizabethan collar to prevent self-trauma**

CORNEAL ULCER

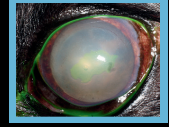
- Clinical signs - lacrimation, photophobia, blepharospasm, conjunctival erythema, corneal opacity (due to corneal edema), vascularization of cornea +/- anterior uveitis (miotic pupil, aqueous flare, iris synechiae, change in iris color, decrease in IOP initially)



Etiology:

- **Primary** - SCCED (spontaneous chronic corneal epithelial defect), corneal degeneration, punctate keratitis
- **Secondary** - Structural (distichiasis, trichiasis, ectopic cilia, lagophthalmos, other eyelid abnormalities), foreign body, trauma, viral (FHV-1), quantitative or qualitative tear film deficiencies (dry eye disease), corneal irritants (heat, smoke, acid, alkalis), neurological disease (neurotrophic keratopathy, facial nerve paralysis)
- Bacterial and fungal keratitis likely requires corneal damage to allow colonization

UNDERRUNNING OF FLUORESCEIN UNDER EPITHELIUM



SCCED ULCER

(SPONTANEOUS CHRONIC CORNEAL EPITHELIAL DEFECT)

- AKA boxer/indolent/chronic superficial/refractory ulcer or canine recurrent erosions
- Clinical signs: see 'corneal ulcer' - ocular discomfort is variable, loose edges of epithelium surrounding denuded corneal stroma (shown by underrunning of fluorescein stain)
- Can persist for weeks to months
- More common in middle-aged to older dogs
- Boxers are over-represented

Treatment

1. **Debridement of loose epithelium**
 - a. Instill topical local anesthetic (e.g.: proparacaine)
 - b. Use dry cotton tip applicator to debride loose edges
 - c. Grid keratotomy* **OR** diamond burr debridement
2. **Appropriate topical antimicrobial therapy as indicated on a case-to-case basis**
3. **Consider use of hyaluronic acid ocular lubricant or bandage contact lens**
 - a. Provides comfort and reduces disruption of migrating corneal epithelial cells by blinking
 - b. **Oculenis™ BioHance™ Ocular Repair Gel:** 1 to 2 drops BID (or as needed based on clinical appearance/response).
4. **Analgesia** (see uncomplicated ulcer)
5. **Hard plastic Elizabethan collar to prevent self-trauma if indicated**

*Not in cats (risk of forming corneal sequestrum)

PROGRESSION IN SIZE/DEPTH

NOT HEALED WITHIN EXPECTED TIME FRAME

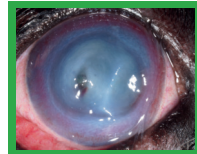
SCCED? RECHECK WITH FLUORESCEIN

COMPLICATED ULCER

- Check for persistent underlying etiology and treat as appropriate (see box above)
- Resistant bacterial infection? Swab for C&S and cytology
- Deep or melting ulcer

MELTING ULCER

- Clinical appearance - opaque/gelatinous/irregular cornea, stromal loss, variable pain, as sensation may have been lost with significant destruction of cornea.
- Stromal collagenolysis caused by excess of host and bacterial matrix metalloproteinases (i.e., collagenase and proteinase enzymes)
- If bacterial infection is present, *Pseudomonas* and *Streptococcus* spp. are commonly implicated (Note - ulcers can degrade even if sterile!)
- Descemetocoele - lack of fluorescein uptake at deepest part of ulcer but often see deep stromal stain uptake around that area of no uptake centrally. The Descemet's membrane of the corneal endothelium does not take up stain and is the remaining layer of cells before perforation!



Aggressive Medical treatment (ideally done in a hospital setting; consider referral)

1. **Anti-collagenases**
 - a. Serum eye drops/EDTA/tetracyclines - typically applied q1-2h and then decreased as melting stops
2. **Aggressive topical antimicrobial therapy***
 - a. In-house corneal cytology can be useful (rods vs cocci) in guiding initial antibiotic choice while awaiting C&S
 - b. Consider adding in systemic antibiotics with good ocular penetration if there is risk for corneal perforation (e.g.: fluoroquinolones)
3. **Analgesia*** (see uncomplicated ulcer)
4. **Hard plastic Elizabethan collar to prevent self-trauma**
5. **Consider use of hyaluronic acid ocular lubricant**
 - a. Provides comfort and supports the tear film
 - b. **Oculenis™ BioHance™ Ocular Repair Gel:** 1 to 2 drops BID (or as needed based on clinical appearance/response).

*Note: Consider avoiding ointment in eyes at risk of perforating - avoid if perforated!

Surgical intervention

Referral for surgical management should be initiated when:

- Any ulcer ≥ 50% of stromal depth
- Descemetocoele
- Area of keratomalacia that enlarges/deepens despite aggressive medical management
- Rupture of the globe

MELTING ULCER: RISK FACTORS

Keratomalacia (corneal melting) develops as a complication of an existing corneal ulcer and can occur at any time point following diagnosis. Keratomalacia should be considered in any ulcer that has progressed in size or depth! Secondary bacterial infection is commonly (but not always) implicated.

Risk factors for the development of keratomalacia include:^{1,2}

- Brachycephalic conformation: up to 64% of melting ulcers in dogs are found in brachycephalic breeds
- Pre-existing ocular surface disease (e.g., KCS, lipid degeneration, SCCED, endothelial degeneration, trichiasis)
- Use of topical steroids on the cornea
- Recent general anesthesia
- Concurrent systemic disease (diabetes mellitus, hypoadrenocorticism, hyperadrenocorticism, hypothyroidism, chronic skin diseases)
- Recent ocular surgery (e.g., keratotomy)
- Chemical injury to the cornea

Further considerations:

- Monitoring at-risk ulcers should be done more frequently (q24-48 hours)
- Corneal cytology can be useful to identify microscopic evidence of keratomalacia (neutrophils, rods, cocci)

NOTE: Oculenis™ BioHance™ Ocular Repair Gel contains a cross-linked HA matrix that has been shown to support accelerated healing. It does *not* contain an antibiotic. Antibiotics are required if an infection is present and should be used for infection prophylaxis as needed. Oculenis™ is *not* a one-for-one substitute for serum. Serum contains factors that may help neutralize the destruction of peptide bonds from collagenases; collagenases contribute to the breakdown of extracellular structures in bacterial pathogenesis. In complex cases of infected ulcers, serum still may be required - treat each case individually and use clinical judgement. Always consider referral of ophthalmic cases due to the possibility of rapid progression.

For more information, please visit domespharma.us/learnmore



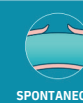
CORNEAL ULCERATIVE DISEASE



CORNEAL EROSION



EPITHELIAL ULCER



SPONTANEOUS CHRONIC CORNEAL EPITHELIAL DEFECT



SUPERFICIAL STROMAL ULCER



DEEP STROMAL ULCER



DESCMETOCOELE



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References: 1. Tsvetanova, A, Powell, R. M., Tsvetanov, K. A., Smith, K. M., & Gould, D. J. (2021). Melting corneal ulcers (keratomalacia) in dogs: A 5-year clinical and microbiological study (2014-2018). *Veterinary ophthalmology*, 24(3), 265-278 2. Guyonnet, A., Desquilbet, L., Faure, J., Bourguet, A., Donzel, E. and Chahory, S. (2020). Outcome of medical therapy for keratomalacia in dogs. *J Small Anim Pract*, 61: 253-258