THERAPY OF SUPERFICIAL CORNEAL ULCERS IN THE HORSE

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Equine corneal ulceration is very common in horses and is a sight threatening disease requiring early clinical diagnosis, laboratory confirmation, and appropriate medical and surgical therapy. Ulcers can range from simple, superficial breaks or **abrasions** in the corneal epithelium, to full-thickness corneal perforations with iris prolapse. The prominent eye of the horse may predispose to traumatic corneal injury. Both bacterial and fungal keratitis in horses may present with a mild, early clinical course, but require prompt therapy if serious ocular complications are to be avoided.


Superficial ulcer

Corneal ulcers in horses should be aggressively treated no matter how small or superficial they may be. Corneal infection and iridocyclitis are always major concerns for even the slightest corneal ulcerations. Iridocyclitis or uveitis is present in all types of corneal ulcers and must be treated in order to preserve vision. Globe rupture, phthisis bulbi, and blindness are possible sequelae to corneal ulceration in horses.

Corneal sensitivity in foals and adult horses

Corneal sensation is important for corneal healing. The cornea of the adult horse is very sensitive compared to other animals. Corneal touch threshold analysis revealed the corneas of sick or hospitalized foals were significantly less sensitive than those of adult horses or normal foals. The incidence of corneal disease is also much higher in sick neonates than in healthy foals of similar age.

Ulcerative keratitis in the equine neonate often differs from adult horses in clinical signs and disease course. Foals may not show characteristic epiphora, blepharospasm, or conjunctivitis, and the ulcers may be missed without daily fluorescein staining. This decreased sensitivity may partially explain the lack of clinical signs often seen in sick neonates with corneal ulcers.

Corneal Healing in the Horse

The thickness of the equine cornea is 1.0 to 1.5 mm in the center and 0.8 mm at the periphery. The normal equine corneal epithelium is 8 to 10 cell layers thick, but increases to 10 to 15 cell layers thick with hypertrophy of the basal epithelial cells following corneal injury. The epithelial basement membrane is not completely formed six weeks following corneal injury in the horse, in spite of the epithelium completely covering the ulcer site. Healing of large diameter, superficial, noninfected corneal ulcers is generally rapid and linear for 5-7 days, and then slows. Healing of ulcers in the second eye may be slower than in the first and is related to increased tear proteinase activity. Healing time of a 7-mm diameter, midstromal depth, noninfected corneal trephine wound was nearly 12 days in horses (0.6 mm/day).

The Equine Corneal Microenvironment

The environment of the horse is such that the conjunctiva and cornea are constantly exposed to bacteria and fungi. The corneal epithelium of the horse is a formidable barrier to the colonization and invasion of potentially pathogenic bacteria or fungi normally present on the surface of the horse cornea and conjunctiva. A defect in the corneal epithelium allows bacteria or fungi to adhere to the cornea and to initiate infection. *Staphylococcus*, *Streptococcus*, *Pseudomonas*, *Aspergillus*, and *Fusarium* spp. are common causes of corneal ulceration in the horse.

Infection should be considered likely in every corneal ulcer in the horse. Fungal involvement should be suspected if there is a history of corneal injury with vegetative material, or if a corneal ulcer has received prolonged antibiotic and/or corticosteroid therapy with slight or no improvement. Tear film neutrophils and some bacteria and fungi are associated with highly destructive proteinase and collagenase enzymes that can result in rapid corneal stromal thinning, descemetocele formation, and perforation. Excessive proteinase activity is termed "melting", and results in a liquefied, grayish-gelatinous appearance to the stroma near the margin of the ulcer.

Total corneal ulceration ultimately requires the degradation of collagen that forms the framework of the corneal stroma. Horse corneas demonstrate a pronounced fibrovascular healing response. The unique corneal healing properties of the horse in regards to excessive corneal vascularization and fibrosis appear to be strongly species specific.

Many early cases of equine ulcerative keratitis present, initially, as minor corneal epithelial ulcers or infiltrates, with slight pain, blepharospasm, epiphora and photophobia. At first anterior uveitis and corneal vascularization may not be clinically pronounced. Slight droopiness of the eyelashes of the upper eyelid may be an early, yet subtle sign of corneal ulceration. A vicious cycle may be initiated after the first injury to the cornea, with "second injury to the cornea" occurring because of the action of inflammatory cytokines.

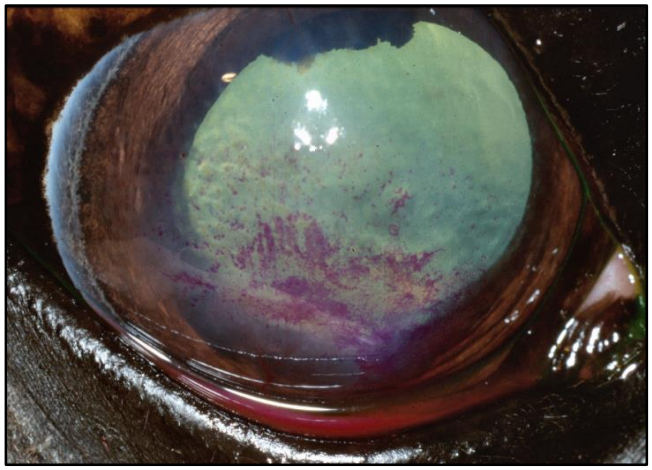
Ulcers, uveitis, blepharitis, conjunctivitis, glaucoma, and dacryocystitis must be considered in the differential for the horse with a painful eye. Corneal edema may surround the ulcer or involve the entire cornea. Signs of anterior uveitis are found with every corneal ulcer in the horse, and include miosis, fibrin, hyphema or hypopyon. Persistent superficial ulcers may become indolent due to hyaline membrane formation on the ulcer bed.

Fluorescein dye retention is diagnostic of a full thickness epithelial defect or corneal ulcer. Faint fluorescein retention may indicate a microerosion or partial epithelial cell layer defect due to infiltration of fluorescein dye between inflamed epithelial cell junctions. All corneal injuries should be fluorescein stained to detect corneal ulcers. Rose bengal retention indicates a defect in the mucin layer of the tear film. RB can be obtained at www.akorn.com

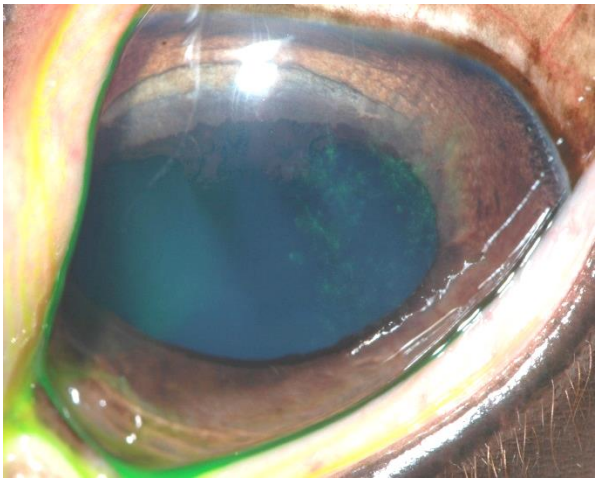
Fungi may induce changes in the tear film mucin layer prior to attachment to the cornea. Early fungal lesions that retain rose bengal are multifocal in appearance and may be mistaken for viral keratitis.

Vigorous corneal scraping at the edge and base of a corneal ulcer is used to detect bacteria and fungal hyphae. Samples can be obtained with the handle end of a sterile scalpel blade and topical anesthesia. Superficial scraping with a cotton swab cannot be expected to yield organisms in a high percentage of cases.

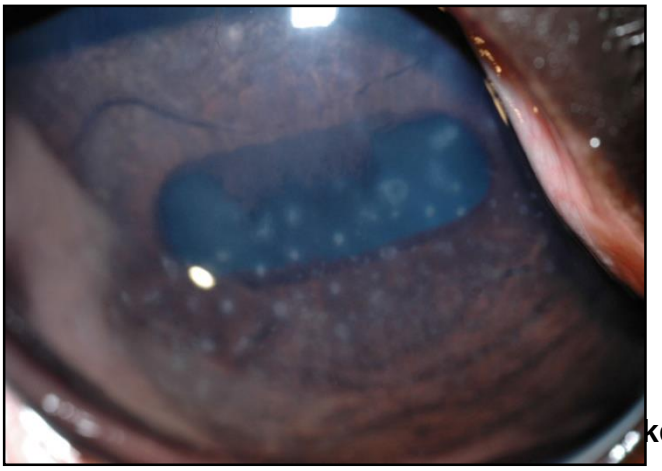
- **Tear film instability with and without microerosions associated with keratomycosis**



Epithelial keratopathy associated with keratomycosis



Subepithelial infiltrates associated with keratomycosis



keratomycosis

Medical therapy: infection, proteases, uveitis

Once a corneal ulcer is diagnosed, the therapy must be carefully considered to ensure comprehensive treatment. Medical therapy almost always comprises the initial major thrust in ulcer control, albeit tempered by judicious use of adjunctive surgical procedures. This intensive pharmacological attack should be modified according to its efficacy. Subpalpebral or nasolacrimal lavage treatment systems are employed to treat a fractious horse or one with a painful eye that needs frequent therapy.

The clarity of the cornea, the depth and size of the ulcer, the degree of corneal vascularization, the amount of tearing, the pupil size, and intensity of the anterior uveitis should be monitored. Serial fluorescein staining of the ulcer is indicated to assess healing. As the cornea heals the stimulus for the uveitis will diminish, and the pupil will dilate with minimal atropine therapy. Self-trauma should be reduced with hard or soft cup hoods.

Antibiotics

Bacterial and fungal growth must be halted and the microbes rendered non-viable. Broad-spectrum topical antibiotics are usually administered with culture and sensitivity tests aiding selection. Topical antibiotic solutions interfere with corneal epithelial healing less than ointments. Gentamicin should be used in ulcers with evidence of stromal melting only.

Topically applied antibiotics, such as chloramphenicol, bacitracin-neomycin-polymyxin B, gentamicin, ciprofloxacin, or tobramycin ophthalmic solutions may be utilized to treat bacterial ulcers. Frequency of medication varies from q2h to q8h.

Cefazolin (55mg/ml), chloramphenicol, bacitracin, and carbenicillin are effective against beta hemolytic Streptococcus. Ciloxan (ciprofloxacin), amikacin (10 mg/ml), and polymyxin B (0.25% IV solution) may be used topically for gentamicin resistant Pseudomonas.

Natamycin, miconazole, itraconazole/ DMSO, fluconazole, amphotericin B, betadine solution, chlorhexidine gluconate, posaconazole, voriconazole, and silver sulfadiazine can be utilized topically. The antifungals can be given q2h to q8h.

Collagenolysis prevention

Severe corneal inflammation secondary to bacterial (especially, Pseudomonas and beta hemolytic Streptococcus) or, much less commonly, fungal infection may result in sudden, rapid corneal liquefaction and perforation. Activation and/or production of proteolytic enzymes by corneal epithelial cells, leucocytes and microbial organisms are responsible for stromal collagenolysis or Amelting@.

Serum is biologically nontoxic and contains an alpha-2 macroglobulin with antiproteinase activity. Autogenous serum administered topically can reduce tear film and corneal protease activity in corneal ulcers in horses. The serum can be administered topically as often as possible, and should be replaced by new serum every 8 days.

Treat Uveitis

Atropine sulfate is a common therapeutic agent for equine eye problems. Topically applied atropine (1%) is effective in stabilizing the blood-aqueous barrier, reducing vascular protein leakage, minimizing pain from ciliary muscle spasm, and reducing the chance of synechia formation by causing pupillary dilatation.

Atropine may be utilized topically q4h to q6h with the frequency of administration reduced as soon as the pupil dilates.

Systemically administered NSAIDs such as phenylbutazone (1 gm BID PO) or flunixin meglumine (1 mg/kg BID, IV, IM or PO) can be used orally or parenterally, and are effective in reducing uveal exudation and relieving ocular discomfort from the anterior uveitis in horses with ulcers. Topical nonsteroidal antiinflammatory drugs (NSAIDs) such as profenol, flurpbiprofen and diclofenamic acid (BID to TID) can also reduce the degree of uveitis.

Horses with corneal ulcers and secondary uveitis should be stall-rested till the condition is healed. Intraocular hemorrhage and increased severity of uveitis are sequelae to overexertion.

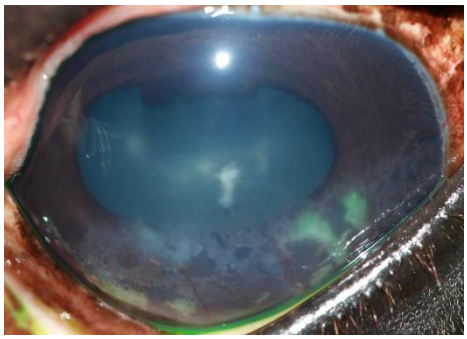
Adjunctive surgical therapy

Bandage soft contact lens (SCL). Bandage SCLs help to maintain apposition of the healing epithelium to the stroma, reduce pain, and protect the new epithelium. Disadvantages include an occasional poor fit in horses thereby resulting in limited retention times. Contact lens retention time may be improved by partial temporary lateral tarsorrhaphy.

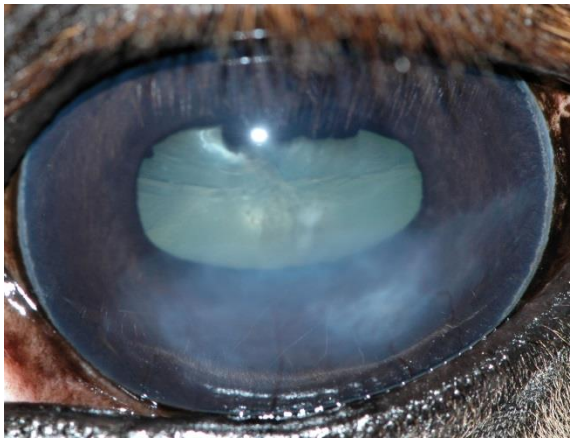

Contact lens and tarsorrhaphy

Debridement, Burring, Keratectomy and Keratotomy

Removing necrotic tissue and microbial debris by keratectomy speeds healing, minimizes scarring, and decreases the stimulus for iridocyclitis. Persistent superficial ulcers may need surgical debridement and keratotomy to remove the hyaline membrane slowing epithelial healing. Debridement to remove abnormal epithelium of refractory superficial erosions can be accomplished with topical anesthesia, a cotton-tipped applicator, or a diamond burr device. Superficial punctate or grid keratotomy of superficial ulcers with a 20-gauge needle can increase the ability of the epithelial cells to migrate and adhere to the ulcer surface.


CBK


Diamond Burr

 Post burring

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