EQUINE OPHTHALMOLOGY

Dennis E. Brooks, DVM, PhD
Diplomate, American College of Veterinary Ophthalmologists
Professor of Ophthalmology
Ophthalmology Service Chief
University of Florida
352-392-2229
brooksd@ufl.edu

Equine Keratopathies

EQUINE CORNEAL ULCERATION

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.

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.

Proteinases in the tear film
Tear film proteinases normally provide a surveillance and repair function to detect and remove damaged cells or collagen caused by regular wear and tear of the cornea. These enzymes exist in a balance with inhibitory factors to prevent excessive degradation of normal tissue.
Two major families of proteinases that may affect the cornea include the matrix metalloproteinases (MMP) and the serine proteinases. MMPs predominate in the horse.

Bacterial and fungal pathogens induce corneal epithelial cells, corneal stromal fibroblasts, and leukocytes (PMN) in the tear film to upregulate cytokines (IL-1, IL-6 and IL-8) that induce MMP production and elicit inflammatory and degradative processes.

Proteinases that may contribute to corneal ulceration in the early stages of infection could be of bacterial or corneal cell origin. In the later stages as PMNs accumulate, PMN-derived proteinases predominate as the main factor in corneal tissue destruction.

In pathologic processes such as ulcerative keratitis, excessive levels of these proteinases can lead to rapid degeneration of collagen and other components of the stroma, potentially inducing keratomalacia or corneal "melting".

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.

Horses with painful eyes need to have their corneas stained with both fluorescein dye and rose bengal dye as fungal ulcers in the earliest stage will be negative to the fluorescein but positive for the rose bengal.

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.

Microbiologic culture and sensitivity for bacteria and fungi are recommended for horses with rapidly progressive, and deep corneal ulcers. Corneal cultures should be obtained first and then followed by corneal scrapings for cytology. Mixed bacterial and fungal infections can be present.

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.

A "crater-like" defect that retains fluorescein dye at its periphery and is clear in the center is a descemetocele, and indicates the globe is at high risk of rupture. Descemet's membrane does not retain fluorescein dye, whereas deep ulcers that continue to have stroma anterior to Descemet=s membrane will retain fluorescein.

Deep penetration of the stroma to Descemet's membrane with perforation of the cornea is a possible sequelae to all corneal ulcers in horses.

Medical therapy

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 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), 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.

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 five days.

Five to 10 per cent acetylcysteine, and/or 0.05% sodium EDTA can be instilled hourly, in addition to the other indicated drugs, for antimelting effect until stromal liquefaction ceases.

It may be necessary to use serum, EDTA, and acetylcysteine simultaneously in severe cases.

Subconjunctival tetanus antitoxin contains macroglobulins with anticollagenase effects and can also slow corneal melting.

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.

Topical atropine has been shown to prolong intestinal transit time, reduce and abolish intestinal sounds, and diminish the normal myoelectric patterns in the small intestine and large colon of horses. Some horses appear more sensitive than others to these atropine effects, and may "respond" by displaying signs of colic and/or prolonged intestinal transit time.

Cecal impaction may occur secondary to topical atropine administration.

Horses receiving topically administered atropine should be monitored for signs of colic.

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.

Debridement, 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 and a cotton-tipped applicator.

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.

Conjunctival Flaps
Conjunctival grafts or flaps are used frequently in equine ophthalmology for the clinical management of deep, melting, and large corneal ulcers, descemetoceles, and for perforated corneal ulcers with and without iris prolapse.

To augment lost corneal thickness and strength, deep corneal ulcers threatening perforation may require conjunctival flap placement. Conjunctival flaps are associated with some scarring of the ulcer site. Coverage with a 360E, hood, island, pedicle, or bridge flap should be maintained for 4 to 12 weeks. Reoccurrence of the inflammation may occur following flap removal.

A conjunctival pedicle flap is made by incising conjunctiva (excluding Tenon's capsule) 1-2 mm posterior to and parallel to the limbus with Steven's tenotomy scissors. The flap is undermined posteriorly toward the fornix as needed. A perpendicular incision is made at the distal end of the flap, and an incision parallel to the first incision and limbus is made several millimeters posterior to the first incision. The flap is rotated over the defect and sutured in place with absorbable 5-0 to 7-0 suture.

Amniotic Membrane Flaps
Amniotic membrane transplantation may provide decreased fibrosis, reduced vascularization of corneal ulcers, and faster reepithelialization in horses with superficial and/or deep corneal ulcers. They may be used alone or with conjunctival flaps.

Third-Eyelid (TE) Flaps
Nictitating membrane flaps are used for superficial corneal diseases including corneal erosions, neuroparalytic and neurotropic keratitis, temporary exposure keratitis, superficial corneal ulcers, superficial stromal abscesses, and to reinforce a bulbar conjunctival graft.

Formation of a third-eyelid flap with attachment to the upper eyelid is performed by placing 2-4 horizontal mattress sutures. Initially pass the cutting needle through the upper eyelid through the fornix at the desired location. Direct the needle (3-0 suture) through the anterior face of the TE approximately 3 mm from the leading edge, and then again in the skin through the fornix adjacent to the first bite. These sutures should not be full-thickness in the TE. One to three additional sutures are placed and then tied.

Temporary tarsorrhaphy
Horizontal mattress sutures enter the eyelid two to three millimeters from the eyelid margin with the cutting needle emerging from at the central aspect (Meibomian gland line) of the eyelid margin, and then reentering the apposing lid margin to exit in the skin. 4-0 silk or nylon is commonly used for this procedure.

Enucleation
Panophthalmitis following perforation of an infected corneal stromal ulcer has a poor prognosis. Phthisis bulbi is likely to result after a chronically painful course. Affected horses can be febrile and manifest signs of septicemia. To spare the unfortunate animal this discomfort, enucleation is the humane alternative. Histopathologic examination of the globe is recommended.

Inappropriate therapy and ulcers
Topical corticosteroids may encourage growth of bacterial and fungal opportunists by interfering with nonspecific inflammatory reactions and cellular immunity.

Corticosteroid therapy by all routes is contraindicated in the management of corneal infections. Even topical corticosteroid instillation, to reduce the size of a corneal scar, may be disastrous if organisms remain indolent in the corneal stroma.

FUNGAL ULCERS IN HORSES

Fungi are normal inhabitants of the equine environment and conjunctival microflora, but can become pathogenic following corneal injury.

Aspergillus, Fusarium, Cylindrocarpon, Curvularia, Penicillium, Cystodendron, yeasts, and molds are known causes of fungal ulceration in horses.

Ulcerative keratomycosis is a serious, sight-threatening disease in the horse. Blindness can occur.

The most often proposed pathogenesis of ulcerative fungal keratitis in horses begins with slight to severe corneal trauma resulting in an epithelial defect, colonization of the defect by fungi normally present on the cornea, and subsequent stromal invasion. Seeding of fungi from a foreign body of plant origin is also possible. Some fungi may have the ability to invade the corneal epithelium following disruption of the tear film. Stromal destruction results from the release of proteinases and other enzymes from the fungi, tear film leukocytes and keratocytes. Fungi may produce antiangiogenic compounds that inhibit vascularization.

Fungi appear to have an affinity for Descemet's membrane with hyphae frequently found deep in the equine cornea. Deeper corneal invasion can lead to sterile or infectious endophthalmitis.

Saddlebreds appear to be prone to severe keratomycosis, while standardbreds are resistant.

Diagnostic tests should include fluorescein and rose bengal staining, corneal cytology, corneal culture with attempted growth on both fungal and aerobic plates, and biopsy if surgery is performed.

Prompt diagnosis and aggressive medical therapy with topically administered antifungals, antibiotics and atropine, and systemically administered NSAIDs will positively influence visual outcome, and may negate the need for surgical treatment.

Treatment must be directed against the fungi as well as against the iridocyclitis that occurs following fungal replication and fungal death.

Therapy is quite prolonged and scarring of the cornea may be prominent.

The fungi are overall more susceptible to antifungal drugs in this order: natamycin = miconazole > itraconazole > ketoconazole > fluconazole.

Natamycin, miconazole, itraconazole/ DMSO, fluconazole, amphotericin B, betadine solution, chlorhexidine gluconate, posaconazole, voriconazole, and silver sulfadiazine can be utilized topically.

Uveitis may be worse the day following initiation of antifungal therapy due to fungal death.

Systemically administered itraconazole or fluconazole may be useful in recalcitrant cases.

ULCER "COCKTAILS"

Equal parts of equine serum, tobramycin, natamycin and cefazolin when combined are very effective against Staphylococcus, Streptococcus and fungi.

VIRAL KERATITIS is seen as a superficial punctate keratitis but is uncommon. Equine herpes¬virus-2 has been identified. Variable response to topical antivirals. Can be seen as a herd problem!

CORNEAL STROMAL ABSCESSES

Focal trauma to the cornea can inject microbes and debris into the corneal stroma through small epithelial ulcerative micropunctures. Some stromal abscesses may be secondary to systemic disease.

A corneal abscess may develop after epithelial cells adjacent to the epithelial micropuncture divide and migrate over the small traumatic ulcer to encapsulate infectious agents or foreign bodies in the stroma. Epithelial cells are more likely to cover a fungal than a bacterial infection.

Reepithelialization forms a barrier that protects the bacteria or fungi from topically administered antimicrobial medications. Reepithelialization of stromal abscesses interferes with both routine diagnostics and treatment.

Corneal stromal abscesses can be a vision threatening sequelae to apparently minor corneal ulceration in the horse. A painful, blinding chronic iridocyclitis may result.

Most stromal abscesses involving Descemet's membrane are fungal infections. The fungi seem "attracted" to the type IV collagen of Descemet's membrane.

Both superficial and deep stromal abscesses do not heal until they become vascularized. The patterns of corneal vascularization are often unique suggesting that vasoactive factors are being released from the abscess that influences the vascular response.

Medical therapy consists of aggressive use of topical and systemic antibiotics, topical atropine, and topical and systemic NSAIDs.

Superficial stromal abscesses may initially respond positively to medical therapy. If reduced inflammation of the cornea and uvea are not found after two to three days of medical treatment, surgical removal of the abscess should be considered.

Deep lamellar and penetrating keratoplasties (PK) are utilized in abscesses near Descemet's membrane, and eyes with rupture of the abscess into the anterior chamber. PK eliminates sequestered microbial antigens, and removes necrotic debris, cyotokines and toxins from degenerating leukocytes in the abscess.

Penetrating Keratoplasty (PK) for Deep Corneal Stromal Abscesses
Corneal transplantation is performed to restore vision, to control medically refractory corneal disease, and to re-establish the structural integrity of the eye.

Penetrating keratoplasty is considered high-risk for rejection in infected, vascularized corneal tissue. Nearly all PKs in horses are in high-risk corneas.

Fresh corneal grafts are preferred in horse PK, but frozen tissue can be utilized.

Vascularization of the grafts, indicating rejection, begins at 5-10 days postoperatively.

Few equine PK grafts remain clear following their vascularization. They form a therapeutic and tectonic function.

OTHER CORNEAL PROBLEMS

Squamous cell carcinoma and other corneal tumors
Preneoplastic epithelia dysplasia, intraepithelial carcinoma in situ, and the invasive squamous cell carcinoma (SCC) are common to the limbus and cornea of horses. Epithelial dysplasia can be treated with topical 5-fluorouracil. Keratectomy and adjunctive therapies are needed for carcinoma in situ and SCC. Rapidly progressive and invasive SCC may necessitate enucleation.
Limbal melanomas and hemangiosarcomas have also been reported.

Corneal foreign bodies
Penetrating and perforating corneal foreign bodies cause varying degrees of keratitis and uveitis and are common in horses. Superficial foreign bodies can be removed under topical anesthesia and the subsequent ulcer treated medically Deep corneal and penetrating foreign bodies may cause severe uveitis/endophthalmitis and require more aggressive care.

Endothelial detachment following blunt trauma
Profound and persistent corneal edema may be present following blunt trauma to the globe of the horse. Detachment of the endothelium is a proposed mechanism of this syndrome. The prognosis for a return to normal is poor. Hypertonic solutions (5% sodium chloride) may be beneficial in the early stages. Thermatokeratoplasty may be necessary to reduce the edema in severe cases. Endothelial cell reattachment and cellular hypertrophy can occur to resolve the condition in some horses.

Nonulcerative keratouveitis (NKU)
Nonulcerative keratouveitis (NKU) is characterized by a nonulcerated, fleshy, paralimbal corneal stromal infiltrate combined with a pronounced anterior uveitis. It is probably immune-mediated.

Topical corticosteroids (1% prednisolone acetate or 0.1% dexamethasone 4 to 6 times daily), cyclosporine A (BID to TID), and mydriatics/cycloplegics (1% atropine SID to QID), with systemic nonsteroidal antiinflammatory drugs are indicated.

Persistent, painful uveitis is severe with NKU and often results in enucleation due to intractable pain.

Nonulcerative interstitial keratitis (NIK)
Several forms of NIK are found in Europe. Some are associated with a "history of ocular trauma". The etiology is presumed to be altered corneal immune privilege from abnormal exposure or expression of corneal antigens inducing autoimmune dysregulation. Nonulcerative superficial and nonulcerative recurrent forms of stromal keratitis are two types of NIK noted in European warmbloods. Stromal pigmentation may occur in some eyes. An endotheliitis with slight corneal edema is another form of NIK. These eyes may partially respond to topically administered corticosteroids, NSAIDs, tacrolimus or cyclosporine A, and may require parenteral antibiotics, corticosteroids, or NSAIDs. Endotheliitis may be found with lens subluxations.

Eosinophilic keratoconjunctivitis
Eosinophilic keratoconjunctivitis has an unknown etiology, but may be an immune-mediated disease.

All ages and breeds of horses can be affected with many cases reported in the spring. Clinical signs include

corneal granulation tissue, blepharospasm, chemosis, conjunctival hyperemia, mucoid discharge, and corneal ulcers covered by raised, white, necrotic plaques. Eosinophilic keratoconjunctivitis resembles a corneal tumor in appearance.

KCS may develop in affected horses due to lacrimal gland inflammation. The lacrimal gland should be palpated to detect swelling.

Corneal cytology typically contains numerous eosinophils and a few mast cells to rule out similar appearing infectious and neoplastic causes.

Superficial lamellar keratectomy to remove plaques speeds corneal healing.

Topical corticosteroids (1% prednisolone acetate or 0.1% dexamethasone) 4 to 6 times a day in early stages (in spite corneal ulcerations), antibiotics (e.g., bacitracin-neomycin-polymyxin or chloramphenicol), 1% atropine, and 0.03% phospholine iodide (BID) in combination with systemic nonsteroidal antiinflammatory drugs are indicated. Topical cromolyn sodium (4.0% TID) or lodoxamide (0.1% TID), mast cell stabilizers, can also aid healing. Systemic corticosteroids may be necessary.

Horses with EK should be dewormed twice with ivermectin 10 days apart.

These lesions are typically slow to heal. Scarring of the cornea occurs.

Herpes keratitis
Multiple, superficial, white, punctate or linear opacities of the cornea, with or without fluorescein dye retention, are found associated with equine herpes virus 2.

The focal punctate corneal opacities may be found at the end of superficial corneal vessels, and may retain rose bengal stain.

Varying amounts of ocular pain, conjunctivitis, and iridocyclitis are present.

Multiple foals in a herd may be affected.

Topically administered idoxuridine and trifluorothymidine (TID) have been used with topical NSAIDs for treatment of equine herpes ulcers, but recurrence is common.

Burdock pappus bristle keratopathy
Burdock pappus (Arctium spp) bristles are common conjunctival foreign bodies in the northeastern United States that can lead to chronic nonhealing corneal ulcers.

Unilateral ocular signs include blepharospasm, serous or mucopurulent ocular discharge, and a positive corneal fluorescein dye uptake.

Chronic epithelial erosion or ulceration can develop.

Differential diagnoses include lid abnormalities such as ectopic cilia, distichiasis, trichiasis, and entropion; neuroparalytic and neurotrophic keratitis; keratoconjunctivitis sicca; infection, and corneal foreign bodies.

Conjunctivalectomy of the bristle foreign body and surrounding tissue is required under sedation and auriculopalpebral nerve block. After conjunctivalectomy, topical antibiotics and atropine, and systemic phenylbutazone are used.

Healing of the corneal defect occurs within three to 14 days after removal of the bristle.

Calcific band keratopathy
Calcific band keratopathy (CBK) is a complication of chronic uveitis and consists of deposition of dystrophic calcium in the superficial corneal epithelium and stroma.

Dense, white bands of calcium are noted in the interpalpebral region of the central cornea.

Scattered areas of fluorescein retention are present as the calcium disrupts the epithelium to result in painful superficial ulcers. Deep ulcers can develop.

A gritty sensation is found durinq scraping for corneal cytology.

It appears to develop in the eyes of horses most aggressively treated with topical corticosteroids for ERU. CBK

is rare in ERU horses that have not been treated medically!

Treatment is topically administered calcium chelaters (dipotassium ethylene diamine tetraacetate 13.8%, Sequester-Sol(r)) to decrease tear calcium levels and aid healing.

Topical antibiotics, atropine, and systemic non-steroidal anti-inflammatory drugs are also beneficial for the ulcers.

Superficial keratectomy may be necessary to remove the painful calcium deposits.

Healing of keratectomy sites can occur with severe scarring.

Recurrence of calcium band keratopathy is possible with continued episodes of uveitis. The prognosis for vision is guarded because of subsequent corneal scarring and further uveitis episodes.

Reference

- Gelatt KN: Veterinary Ophthalmology 4th Ed, Lippincott, Williams and Wilkins, Philadelphia, 2007.
- Brooks DE: Ophthalmology for the Equine Practitioner. Teton NewMedia, Jackson, WY, 2008.
- Gilger BC (ed): Equine Ophthalmology, Ed 2. Elsevier, Maryland Heights, MO, 2010.
- Clinical Techniques in Equine Practice: Equine Ophthalmology, 4(1); 2005.