

Diagnosis and Treatment of Equine Corneal Disease

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Introduction: The most common reason that horses present for ophthalmic examination is corneal disease. The initial cause is commonly trauma, but immune mediated disease, neoplasia and degenerative changes also frequently occur.

Examination: In order to best possibly identify the causative factors of the corneal disease, it is best to always perform a complete ophthalmic examination. Step back and assess for symmetry, lid and globe position and exudate. Examination of a painful globe can be extremely difficult. Horses have very strong lid muscles and complete examination of the cornea is next to impossible in some cases without a proper nerve block and sedation. In cases of deep ulcerative or malacic corneal disease examination without proper restraint can result in ocular rupture. Placement of an auriculopalpebral nerve block will allow for lid opening without applying pressure to the globe.

A strong light source and adequate magnification are a must to assess the surface, contour and thickness of the cornea. Opacifications and thickening can be due to edema, vascular or cellular infiltrate, lipid or mineral deposit or neoplasia. The lid margins can be assessed to determine if there are defects, masses, or abnormal hairs that may be causing corneal irritation. The palpebral and third eyelid conjunctiva should be critically assessed for masses, inflammation and foreign bodies. Corneal disease can cause a reflex uveitis and conversely, uveitis and glaucoma can cause secondary corneal disease. Assessment of the entire globe is important to determine the difference.

Diagnostics: The palpebral reflex should always be assessed to determine if there is a complete blink reflex that will protect the cornea and spread tears normally. Assess the corneal surface for mucoid debris and for a normal luster. Primary keratoconjunctivitis sicca is uncommon in the horse. However, dry eye secondary to guttural pouch or neurologic disease in the horse does occur. Schirmer values can be measured and qualitative tests of tear film integrity are valuable. In cases of persistent or deep ulceration samples should be obtained for culture and sensitivity. This should be done prior to applying topical anesthesia or diagnostic stains as the preservatives in these drops will inhibit bacterial culture growth. Corneal cytology and gram stain can be even more helpful in the short term than culture, in that results can be returned within 24hrs allowing for alterations in medical management as necessary. The cornea should always be stained with Fluorescein to diagnose corneal erosions or ulcers. Rose Bengal staining will highlight degenerative corneal epithelial cells and will expose tear mucin deficiencies.

Ulcerative Keratitis: Corneal abrasions and ulcers are most commonly caused by trauma. Trauma can be secondary to the equine self destruction gene or irritation secondary to foreign bodies, tumors, eyelid cilia or lid defects. Superficial acute erosions and ulcers are treated with a prophylactic topical broad spectrum antibiotic ointment such as bacitracin/neomycin/polymixin.. Painful eyes can be treated with mydriatic such as atropine to eliminate ciliary spasm. Atropine should be used to effect and care should be taken to protect the eye from sunlight. A non-steroidal anti-inflammatory should be used to augment pain control. A small corneal ulcer will normally heal 1-3 days. If a lesion takes longer than this to heal something more serious than an uncomplicated corneal ulcer should be suspected. Even superficial ulcers it is recommended that they be re-checked frequently due to their tendency to develop more severe problems.

Non-Healing Superficial Erosions: If an superficial ulcer fails to heal within a week, the whole ophthalmic examination should be repeated in search of a potential cause. Often with antibiotic and anti-inflammatory treatments the decrease in inflammation will allow for visualization of things that were obscured initially (abnormal hairs, small foreign bodies, etc). Alternatively, persistent superficial ulceration can be due to a

defect in the healing in which the corneal epithelial cells fail to adhere to the underlying corneal stroma. Dry debridement in these cases is often enough to induce appropriate adherence and normal healing. In the absence of significant edema or cytologic evidence of bacterial or fungal disease, some refractory cases can require a linear grid keratotomy.

Infected bacterial ulcers may result in rapid breakdown of corneal collagen with the risk of corneal perforation. Intensive medical treatment should focus on control of the infection and stopping the enzymatic degradation of the corneal stroma. The bacteria implicated in cases of corneal malacia vary by geographic region and include *Pseudomonas* spp and beta-hemolytic *Streptococcus* spp. Antibiotic selection for deep ulcers can be made empirically or can be based upon cytology and gram stain. Topical fluoroquinolones (ciprofloxacin, ofloxacin, moxifloxacin) or aminoglycosides (tobramycin, gentamicin) and a cephalosporin (cefazolin fortified tears) are recommended. Frequency of administration should be initially be every 1- 2 hours until the malacia is controlled. After the malacia is controlled treatment frequency can decrease to q6-4h. Antibiotic choice may have to be adjusted based on the initial response to therapy or the results of culture and antibiotic sensitivity. The most commonly used anti-proteases/anti-collagenases are autologous serum or 1% EDTA solution. Atropine can be utilized for cycloplegia and mydriasis in cases with reflex uveitis (q12h initially and then to effect). Attempted manual topical medication in these cases is not recommended. If the owners want to save the eye, a sub-palpebral lavage should be placed and an eye-saver should be employed.

Stromal ulcers that penetrate greater than 50% of the corneal depth are best treated with surgical management to provide the cornea with tectonic support as it heals. Cleaning up the cornea and controlling malacia prior to surgery gives the best chance for success. Surgical options include conjunctival pedicle grafts, corneal grafting or transplantation, and the use of amniotic membrane grafts. Many corneal lacerations and perforated corneal ulcers are treated in the same manner. The equine cornea has an amazing capacity to heal even after perforation if treated surgically and with aggressive post-operative medical management.

Fungal keratitis lesions present as white to gray superficial stromal infiltrates with epithelial ulceration or deep stromal keratitis with marked uveitis. Horses with fungal keratitis tend to be exquisitely painful, but this is the norm, not the rule. Some chronic fungal keratitis cases in this area can appear quite comfortable. Superficial plaques can be debrided and treated with topical antifungal agents q4h to q6h. Itraconazole with DMSO, voriconazole, natamycin or miconazole are suitable agents for many of the fungal pathogens seen in the Upper Midwest. Topical treatment can be required for 6 weeks or longer. Fluconazole can be used orally in horses with a loading dose of 14mg/kg PO once, followed by 5mg/kg PO q24h for 10 days. In cases which do not improve with medical therapy surgical treatment is indicated. Surgery involves removing affected cornea and employing an appropriate grafting technique.

Infectious Non-ulcerative Keratitis: Stromal abscesses occur when bacteria or fungi are inoculated in the cornea stroma, followed by re-epithelialization of the inoculation site. The stromal abscess appears as a yellow to white deep corneal opacity. Corneal vascularization will normally be present. Treatment is dependent upon clinician choice and patient comfort level. Most often surgery to remove the affected tissue and to provide tectonic support is the best option, followed by appropriate medical management. Cytology, histopathology and culture will aid in treatment decisions

Immune Mediated Non-Ulcerative Keratitis:

Immune mediated keratitis (IMMK) presents as corneal opacification and vascularization at various levels within the cornea. Unlike infectious corneal disease these lesions cause no pain to only mild discomfort. The treatment depends on the level of the cornea that is affected. Management ranges from topical corticosteroid and cyclosporine to keratectomy to remove areas of opacification. The superficial and stromal conditions tend to be the most amenable to treatment with the endothelial IMMKs often being refractory to treatment.

Eosinophilic keratoconjunctivitis occurs in horses and presents as peripheral corneal vascularization with a significant cellular infiltrate. The presence of superficial granular white plaques is variable. Diagnosis is

made on clinical presentation, lack of response to treatment with topical antibiotics and cytology. Treatment with topical corticosteroid is often effective at treating the disease.

Corneal Trauma: Corneal lacerations should be repaired as soon as possible after the injury to avoid wound retraction, worsening corneal edema and infection. Referral to a veterinary ophthalmologist is recommended in all cases. Sharp lacerations that are limited to the cornea generally have a fair prognosis for vision and comfort. Lacerations that extend into the sclera or lacerations due to blunt trauma present with a poor prognosis. It is important to evaluate the status of the lens in all corneal injuries. Lens capsule laceration with the prolapsed of lens material into the eye results in uveitis that is uncontrollable and the only treatment that will save the eye is lens removal at the time of corneal repair.

Corneal Neoplasia: Various tumors affect the conjunctiva and cornea. Squamous cell carcinoma is the most commonly seen especially in horses with light colored eyes and those that are exposed to high levels of sunlight. Draft breeds (Belgian, Clydesdale and Shire) and Appaloosas have a significantly greater prevalence of ocular and adnexal SCC than other breeds. Cytology or superficial biopsy of corneal lesions is indicated to confirm the diagnosis. Lesions may appear as superficial, diffuse opacification with vascularization or develop into raised and papillomatous mass lesions. Treatment involves debulking the lesion and ancillary therapies which may include chemotherapeutic agents (mitomycin-C or 5-fluorouracil), photodynamic laser therapy, beta or gamma irradiation, cryotherapy, or CO₂ laser ablation.

Resources:

Sub-Palpebral Lavage Kits – Mila

(<http://milainternational.com/ca/products/equine-large-animals/eye-lavage-kit.html>)

Eye Saver (Blind Blinkers) – The Finish Line Oklahoma

(<http://www.tfloki.com>).