Equine Ophthalmology

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1. Vision in the Horse: What Does the Horse “See”?
The horse has a total visual field of nearly 360°, meaning a horse can just about see its tail with its head pointed forward. A small frontal binocular field of 65° is present. The horse retina is adapted for detection of movement because it contains significant numbers of large-diameter ganglion cells. The nasal extension of the retina, the laterality of the eyes, and the horizontal pupil facilitate tremendous peripheral vision for the horse standing with its head up.

The horse has weak accommodative ability of the lens and therefore has limited near focus capability. The horse uses both eyes until an object approaches within 3–4 ft, when it is forced to turn or lower its head to continue to observe with one eye. Horses need to accommodate <2 D to maintain a focused image on the retina. Mean refractive error of horses is ~1.0 D. This seems to be similar to 20/33 vision on the Snellen chart. Horses have poor acuity in the peripheral retina.

Rods significantly outnumber cones, indicating the high capacity for night vision in horses, but cones are present in the horse retina with the capacity for color vision. Horses see blues and yellows but may have more trouble with longer wavelength colors such as red.

2. The Foal Eye
A newborn foal may exhibit lagophthalmos, low tear secretion, slow pupillary light reflexes until 5 days of age, an oval-shaped pupil, reduced corneal sensitivity, lack of a menace reflex for up to 2 wk, hyaloid artery remnants containing blood for several hours after birth, prominent lens Y sutures, and a round, pale pink to deep red optic disc with smooth margins.

Tapetal color is initially green to yellow-green in foals. Color-dilute foals have a red fundic reflection from a lack of a tapetum and consequential exposure of choroidal vessels.

3. Ocular Problems in the Foal
Dermoids (choristomas) are neoplastic aggregates of skin tissue aberrantly located in the conjunctiva, cornea, or eyelid. Hair follicle development in a dermoid may cause ocular irritation or corneal ulceration. Treatment would be a keratectomy for corneal dermoids and blepharoplasty for eyelid lesions.

Entropion is an inward rolling of the eyelid margin (Fig. 1). This causes the eyelid hairs to rub on the cornea. It can be a primary problem or it can be secondary to dehydration or emaciation in “downer foals.” Entropion should be repaired in foals by placing vertical mattress sutures to evert the offending eyelid margin, preventing corneal ulceration. Hotz-Celsus type entropion surgery should be reserved for older horses.

Lacrimal puncta agenesis or duct atresia may be unilateral or bilateral. Clinical signs are a
chronic mucoid and eventually copious mucopurulent discharge in a young horse (Fig. 2). Presumptive diagnosis of duct or punctal agenesis with dacryocystitis may be made by noting the lack of a distal puncta opening of the nasolacrimal duct at the mucocutaneous junction within the nares. Dacryocystorhinography will identify the specific location of the obstruction. Surgical repair of the congenital defect and medical treatment of the associated dacryocystitis are required.4

Microphthalmos is a common ophthalmic congenital defect in the foal (Fig. 3).4 The entire globe is small. The microphthalmic eye may be visual or is associated with other eye problems that cause blindness. Strabismus is deviation of the globe from its normal orientation and may be noted alone or with other congenital ocular deformities. Anterior Segment Dysgenesis of the Rocky Mountain Horse consists of increased corneal curvature, iris hypoplasia, congenital miosis, uveal cysts, cataracts, and retinal dysplasia.5

Persistent pupillary membranes (PPMs) are embryonic remnants of the iris seen as linear tissue connections between the iris and iris, iris and lens, and/or iris and cornea. PPMs seldom cause any visual impairment, although focal lens or corneal opacities may be present. There is generally no need for surgical transection of PPMs.1,4

Congenital cataracts or lens opacities in foals are common congenital eye defects in the horse (Fig. 4).1,4 Blindness can occur if cataracts are mature. Phacoemulsification cataract surgery has a high success rate in foals with blinding cataracts.6 Congenital lens luxation from zonular weakness is a severe eye problem that also requires surgery for resolution.1,4

Persistent superficial corneal ulcers in the neonatal foal may be associated with decreased corneal sensation. These superficial ulcers require early detection and treatment to prevent ulcer progression.1,4

Iridocyclitis in the foal is generally secondary to septicemia and may be unilateral or bilateral. Fibrin, hyphema, and/or hypopyon may be present. Infectious and toxic etiologies are reported in foals. Severe unilateral, blinding, fibrinous uveitis secondary to plant toxins has been noted primarily in Thoroughbred foals and yearlings in the southern United States.
Congenital glaucoma and congenital retinal detachment are found periodically in foals and represent severe blinding eye problems with no therapy available.1,4

4. Diseases and Surgery of the Eyelids

Traumatic Eyelid Lacerations

Lid trauma needs to be corrected as soon and as accurately as possible to prevent undesirable lid scarring and secondary corneal desiccation and ulceration. Eyelids are highly vascular and have a great capacity to heal and resist infection. They can also swell quite dramatically. Minimal debridement is needed because of their extensive blood supply, and an eyelid “tag” or pedicle flap should never be excised because exposure keratitis and corneal ulceration can result (Fig. 5).

Upper eyelid damage is more significant in horses because the upper lid moves over more of the equine cornea than does the lower lid. Medial canthal lid trauma can involve the nasolacrimal system. It is important to thoroughly examine the globe both externally and through ophthalmoscopy. The nasolacrimal system should also be evaluated for damage when medial canthal injuries are present. Preservation of the eyelid margin is critical if at all possible to preserve eyelid function. The repaired lesion must be protected from “self-trauma” with masks or hard cups.1,4

Neoplasia of the Lids

Eyelid melanomas are found in grey horses; Arabians and Percherons also have an increased risk. Melanomas may be single or multiple. Treatment is surgical excision and cryotherapy.

Sarcoïds are solitary or multiple tumors of the eyelids and pericocular region of the horse. Retroviruses and papilloma viruses may be involved in the etiology. It is suspected that flies may be able to transfer sarcomatous cells from one horse to traumatic skin lesions in other horses. There are geographic differences in the aggressiveness of the sarcomatoid in horses.7

The sarcomatoid lesion induces a fibrovascular inflammatory response that may mask the actual size of the sarcomatoid. Shrinking the sarcomatoid lesion with antipsoriasis skin ointments and/or topical 5-fluorouracil (5-FU) for 2 wk may be beneficial before using Bacillus Calmette-Gaérin (BCG).7 Surgical resection of necrotic tissue is controversial, with some experts suggesting it will exacerbate the sarcomatoid.

Immunotherapy for sarcomas includes autogenous vaccines and immunomodulators of mycobacterial products. Immunomodulation using attenuated Mycobacterium bovis cell wall extracts such as the immunostimulant BCG, however, has produced reasonable remission rates.7 Cryotherapy, hyperthermia, carbon dioxide laser excision, intralereal chemotherapy, and intralereal radiotherapy can also be effective for sarcomatoid. Intralereal chemotherapeutics including 5-FU or cisplatin have been used with varying success rates. Interferon has been used systemically for very large, aggressive equine sarcomas. Homeopathic ointments and caustic chemical lotions are effective in some sarcomas.7

Squamous cell carcinoma (SCC) is the most common tumor of the eye and adnexa in horses (Fig. 6). The etiopathogenesis may be related to the ultraviolet (UV) component of solar radiation, periocular pigmentation, and an increased susceptibility to carcinogenesis. The UV component is the most plausible carcinogenic agent associated with SCC, because it targets the tumor suppressor gene p53, which is altered in equine SCC.8,9

Prevalence of SCC in horses increases with age. Draft horses have a high prevalence of ocular SCC, followed by Appaloosas and Paints, with the least prevalence found in Arabians, Thoroughbreds, and Quarter Horses. White and palomino hair colors predispose to ocular SCC, with less prevalence in bay, brown, and black hair coats.

Fig. 5. (a) Severe lower lid laceration. (b) Healed laceration 3 wk after surgery.
Cryotherapy, immunotherapy, irradiation, radiofrequency hyperthermia, CO2 laser ablation, or intraliteral chemotherapy should follow surgical excision of equine ocular SCC. Additionally, reconstructive eyelid surgery may be required when eyelid margins are lost after tumor excision, and conjunctival grafts are indicated after keratectomy for corneal SCC.

Immunotherapy with BCG cell wall extract has been used successfully for large periocular SCC in horses. Chemotherapy of invasive eyelid SCC with intralesional, slow release cisplatin has been used with and without surgical debulking. Topical 51% 5-FU (q8h) can be effective for epithelial dysplasia and corneal SCC in situ and may be beneficial for extensive periocular SCC.1,4

Tumors may be removed by surgical excision alone if adequate margins can be obtained. However, adjunctive therapy is often recommended to improve the chance for a complete cure, especially with large or invasive tumors. Small, superficial tumors may be treated with radiofrequency hyperthermia or cryosurgery. Malignant cells are killed with local temperatures of 41-50°C, after surgical excision. Cryosurgery with liquid nitrogen or nitrous oxide induces cryonecrosis of malignant cells when temperatures of -20°C to -40°C are achieved using a double freeze-thaw technique.1,4

Excision of corneal limbal SCC followed by CO2 laser ablation has also been advocated. Radiotherapy with beta irradiation (strontium 90) is very beneficial in superficial SCC of the cornea and limbus after superficial keratectomy. Brachytherapy using iridium 192 may be employed after surgical debulking of invasive eyelid tumors. Interstitial radiation therapy has the advantage of providing continuous exposure of the tumor to high levels of radiation over a period of time.

5. Diseases of the Cornea

Corneal Healing in the Horse

The thickness of the equine cornea is 1.0–1.5 mm in the center and 0.8 mm at the periphery.

Fig. 6. Large squamous cell carcinoma at medial canthus.
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.12

Equine Corneal Microenvironment
The environment of the horse is such that the conjunctiva and cornea are constantly exposed to bacteria and fungi.1 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 sp. are common causes of corneal ulceration in the horse.1,13

6. 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.1,4

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. Infection should be considered likely in every corneal ulcer, no matter how small, in the horse.1,4 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 without improvement. Iridocyclitis is present in all types of equine corneal ulcers and must also be treated to preserve vision. Globe rupture, phthisis bulbi, and blindness are possible sequelae to corneal ulceration in horses.

Horse corneas demonstrate a pronounced fibrovascular healing response.1,4 The unique corneal healing properties of the horse in regards to excessive corneal vascularization and fibrosis seem 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 (Fig. 7).

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.14 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 because of hyaline membrane formation on the ulcer bed.

Fluorescein and Rose Bengal Dyes and Other Diagnostics
All corneal injuries should be fluorescein stained to detect corneal ulcers (Fig. 8). Horses with painful eyes need to have their corneas stained with both fluorescein dye and rose bengal dye because fungal

Fig. 7. (a) Upper eyelashes perpendicular to the cornea in a normal eye. (b) Upper eyelashes pointed down in a painful eye.
ulcers in the earliest stage will be negative to the fluorescein but positive for the rose bengal. 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 caused by infiltration of fluorescein dye between inflamed epithelial cell junctions (Fig. 9).1,4,9

Rose bengal retention indicates a defect in the mucin layer of the tear film. Fungi may induce changes in the tear film mucin layer before attachment to the cornea. Early fungal lesions that retain rose bengal are multifocal in appearance and may be mistaken for viral keratitis.1,4

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.1,4

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.

A “crater-like” defect that retains fluorescein dye at its periphery but 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. An iris prolapse will occur if Descemet’s membrane ruptures from a progressive ulcer or corneal laceration. Iris prolapse surgical repair is best for traumatic lacerations, small corneal lesions, and recent rupture (Fig. 10).1,4

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

Cefazolin (55 mg/ml), chloramphenicol, bacitracin, and carbenicillin are effective against beta-hemolytic Streptococcus. 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 (Figs. 11 and 12). Activation and/or production of proteolytic enzymes by corneal epithelial cells, leucocytes, and microbial organisms are responsible for stromal collagenolysis or “melting.”

Serum is biologically nontoxic and contains an alpha-2 macroglobulin with antiproteinase activity. Growth factors are present in serum, but not found in plasma. 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 5 days. Acetylcysteine (5–10%) and/or 0.17% sodium ethylenediamine tetra-acetic acid (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.

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 used 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 and reduce and abolish intestinal sounds in the small intestine and large colon of horses.
horses. Some horses seem more sensitive than others to these atropine effects and may “respond” by displaying signs of colic. Cecal impaction may occur secondary to topical atropine administration.\textsuperscript{1,4}

Systemically administered nonsteroidal anti-inflammatory drugs (NSAIDs) such as phenylbutazone (1 g, q12h, PO) or flunixin meglumine (1 mg/kg, q12h, 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 NSAIDs such as diclofenamic acid (q12h to q8h) can also reduce the degree of uveitis.

Adjuvant Surgical Therapy

**Bandage Soft Contact Lens**

Bandage soft contact lens (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.\textsuperscript{1,4}

**Debridement, Keratectomy, and Keratotomy**

Removing necrotic tissue and microbial debris by keratectomy speeds healing, minimizes scarring, and decreases the stimulus for iridocyclitis. 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 in superficial ulcers.\textsuperscript{1,4}

**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 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 360° hood, island, pedicle, or bridge flap should be maintained for 4–12 wk. Reoccurrence of the inflammation may occur after premature flap removal.\textsuperscript{1,4}

**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. They contain antiproteinases that inhibit the proteinases found in the tears of horse eyes with corneal ulcers.\textsuperscript{15}

**Third-Eyelid 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.

**Temporary Tarsoorhaphy**

Horizontal mattress sutures enter the eyelid 2–3 mm 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 after 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 severe pain, enucleation is the humane alternative.

**Inappropriate Therapy and Ulcers**

Topical corticosteroids may encourage growth of bacterial and fungal opportunists by interfering with non-specific 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.

**Please Remember the Following**

- Corneal ulcers are frequently not clearly visible even with proper examination lighting
- All red or painful eyes must be stained with fluorescein and rose bengal dyes
- A slowly progressive, indolent course often belies the seriousness of the ulcer
- Corneal ulcers in horses may rapidly progress to descemetoceles
- Topical corticosteroids are contraindicated when the cornea retains fluorescein stain
- Anterior uveitis caused by a corneal ulcer or stromal abscess may be very difficult to control
- Local anesthetics should not be used in treatment of corneal ulcers as they retard epithelial healing

7. **Fungal Ulcers in Horses**

Fungi are normal inhabitants of the equine environment and conjunctival microflora but can become pathogenic after corneal injury. *Aspergillus, Fusarium, Cylindrocarpon, Curvularia, Penicillium, Cystodendron*, yeasts, and molds are known causes of fungal ulceration in horses.\textsuperscript{1,4}
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, however, also have the ability to invade the corneal epithelium after disruption of the tear film.

Stromal destruction results from the release of proteinases and other enzymes from the fungi, tear film leukocytes, and keratocytes (Fig. 12). Fungi may produce antiangiogenic compounds that inhibit vascularization. Fungi seem 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 seem 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 after 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, 1% miconazole, 1% itraconazole/30% dimethyl sulphoxide, (DMSO) 0.2% fluconazole, 0.15% amphotericin B, 2% Betadine solution, and silver sulfadiazine can be used topically.

Uveitis may be worse the day after initiation of antifungal therapy caused by fungal death. Lufenuron (5 mg/kg, q6h, PO) is a chitin synthase inhibitor used orally to attack the fungal cell wall in horses with keratomycosis. Systemically administered itraconazole (3 mg/kg, q12h, PO) or fluconazole (1 mg/kg, q6h, PO) may be useful in recalcitrant cases.

Conjunctival flap and penetrating keratoplasty are used in treating aggressive keratomycosis in horses.

Viral Keratitis is seen as a superficial punctate keratitis but is uncommon. Slight fluorescein and/or rose bengal dye retention may be seen superficial to stromal neovascular capillary tufts. Clinical signs can intermittently wax and wane. Equine herpesvirus-2 has been identified. There is a variable response to topical antivirals, but topical NSAIDs can be helpful. This can be seen as a herd problem.

8. Corneal Stromal Abscesses

Focal trauma to the cornea can inject microbes and debris into the corneal stroma through small epithelial ulcerative micropunctures. 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 (Fig. 13).

Conical 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 Descemet’s membrane.

Medical therapy consists of aggressive use of topical and systemic antibiotics, topical atropine, and topical and systemic NSAIDs. Both superficial and deep stromal abscesses do not heal until they be-
come vascularized. The patterns of corneal vascularization are often unique suggesting that vasoactive factors are being released from the abscess that influences the vascular response.

Superficial stromal abscesses may initially respond positively to medical therapy. If reduced inflammation of the cornea and uvea are not found after 2–3 days of medical treatment, surgical removal of the abscess should be considered. Daily removal of superficial epithelium may speed drug penetration to the abscess site.

Penetrating Keratoplasty for Deep Corneal Stromal Abscesses

Deep lamellar and penetrating keratoplasties (PK) are used 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, cytokines, and toxins from degenerating leukocytes in the abscess.

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 used. Few equine PK grafts remain clear after their vascularization.

9. Cataracts in the Horse

Cataracts are opacities of the lens and are the most frequent congenital ocular defect in foals. Horses manifest varying degrees of blindness as cataracts mature. Very small incipient lens opacities are common and not associated with blindness. As cataracts mature and become more opaque, the degree of blindness increases. The tapetal reflection is seen with incipient and immature cataracts but is not seen in mature cataracts.

Examination of the fundus may be difficult because of the cataract. The rate of cataract progression and development of blindness cannot be predicted in most instances. Heritable, traumatic, nutritional, and post-inflammatory etiologies have been proposed for equine cataracts. Cataracts secondary to equine recurrent uveitis (ERU) or trauma are frequently seen in adults. True senile cataracts that interfere with vision are found in horses older than 20 yr. Increased cloudiness of the lens normally occurs with age and is called nuclear sclerosis. It is common in older horses, but vision is clinically normal, because nuclear sclerosis does not cause vision loss.

Equine Cataract Surgery

Most veterinary ophthalmologists recommend surgical removal of cataracts in foals less than 6 mo of age if the foal is healthy, no uveitis, or other ocular problems are present, and the foal’s personality will tolerate aggressive topical medical therapy. Horses considered for lens extraction should be in good physical condition. General anesthesia with its attendant risks is required for cataract surgery.

Slow or absent pupillary light reflexes (PLRs) may indicate active iridocyclitis with or without posterior synechiae, retinal disease, optic nerve disease, or iris sphincter muscle atrophy. Afferent pupillary defects in a cataractous eye cannot be attributed to the cataract alone, as well as the fact that normal PLRs do not exclude some degree of retinal or optic nerve disease.

B-scan ultrasound and electroretinography are beneficial in assessing the anatomical and functional status of the retina if a cataract is present.

Phacoemulsification cataract surgery is the most useful technique for the horse. This extracapsular procedure through a 3.2-mm corneal incision uses a piezoelectric handpiece with an ultrasonic titanium needle in a silicone sleeve to fragment and emulsify the lens nucleus and cortex after removal of the anterior capsule. The emulsified lens is then aspirated from the eye while intraocular pressure is maintained. The thin posterior capsule is left intact. There is little inflammation postoperatively in most horses following successful phacoemulsification cataract surgery and a quicker return to normal activity with phacoemulsification.

Postoperative Cataract Surgery Therapy and Results

Topically applied antibiotics, such as chloramphenicol, gentamicin, ciprofloxacin, or tobramycin ophthalmic solutions may be used pre- and postoperatively. Topically applied 1% atropine is effective in stabilizing the blood-aqueous barrier, minimizing pain from ciliary muscle spasm, and causes pupillary dilatation. Topically applied corticosteroids are essential to suppress postoperative inflammation. Systemically administered NSAIDs can be used orally or parenterally, and are effective in reducing anterior uveitis in horses with cataracts. Topically administered NSAIDs such as diclofenac, flurbiprofen, and suprofen must also be used to suppress signs of anterior uveitis.

The results of cataract surgery in foals by experienced veterinary ophthalmologists are generally very good, with success near 80%. The cataract surgical results in adult horses with cataracts caused by ERU are often poor.

Aphakic Vision in Horses

Most reliable reports of vision in successful cataract surgery in horses indicate vision is functionally normal postoperatively. From an optical standpoint, the aphakic eye should be quite far-sighted or hyperopic postoperatively. Images close to the eye would be blurry and appear magnified.
IN DEPTH: OPHTHALMOLOGY

10. Diseases of the Uveal Tract

Equine Recurrent Uveitis (Periodic Ophthalmia, Moon Blindness, Iridocyclitis)

Equine recurrent uveitis (ERU) is a common cause of blindness in horses.\(^1,4,20,21\) It is an autoimmune disease with multiple causes. Recurrence of anterior uveitis is the hallmark of ERU. The disease is bilateral in approximately 20%. Hypersensitivity to infectious agents such as Leptospira interrogans servars is commonly implicated as a possible cause. ERU may represent a case of molecular mimicry where ocular antigens found in the horse eye are identical to antigens found in Leptospiral and other microbial organisms. The presence of living Leptospira organisms is not necessary for disease production but is found in many cases.\(^1,4\) Autoimmune activity against retinal proteins and antigens is also an etiologic component of this disease.

Toxoplasmosis, brucellosis, salmonellosis, streptococcus, Escherichia coli, Rhodococcus equi, borreliosis, intestinal strongyles, onchocerciasis, parasites such as Halicephalobus deletrix, and viral infections (e.g., equine influenza virus, herpes virus 1 and 4, arteritis virus, and infectious anemia virus) have also been implicated as causes of ERU with no consistency in isolation of these organisms from affected horses.\(^1,4\)

Serology for Leptospira pomona can be used for prognostic evaluation of the likelihood of blindness occurring in one or both eyes. Seropositive (>1:400) Appaloosas (100%) > seronegative Appaloosas (72%) > seropositive non-Appaloosas (51%) > seronegative non-Appaloosas (34%) at having blindness occur in at least one eye within 11 yr of the first attack.\(^21\)

Horses with ERU display increased lacrimation and blepharospasm. Subtle corneal edema, conjunctival hyperemia, and ciliary injection will be present initially and can become prominent as the condition progresses. Aqueous flare, hyphema, intraocular fibrin, and hypopyon may be observed. Miosis is usually a prominent sign and can result in a misshapen pupil and posterior synechiae. Intraocular pressure (IOP) is generally low, but ERU may be associated with intermittent and acute elevations in IOP. Cataract formation may occur if the inflammation does not subside quickly. Chorioiditis may result in focal or diffuse retinitis and exudative retinal detachments. The vitreous may develop haziness because of leakage of proteins and cells from retinal vessels.\(^1,4\)

In acute stages of ERU, lymphocytic infiltration with some neutrophils can be found in the uveal tract, resulting in edema and plasmoid vitreous. Lymphocytes and plasma cells can surround the blood vessels of the iris, ciliary body, choroid, and retina. In chronic cases, corneal vascularization, permanent corneal edema, synechiation, cataract formation, and iris depigmentation or hyperpigmentation can result. Retinal degeneration indicated by focal to generalized peripapillary regions of depigmentation in the nontapetum can result. The optic nerve head will appear pale if atrophic. Secondary glaucoma and phthisis bulbi occur. Irreversible blindness is a common sequelae to ERU.\(^1,4\)

ERU Therapy

The major goals of treatment of ERU are to preserve vision, decrease pain, and prevent or minimize the recurrence of attacks of uveitis. Specific prevention and therapy is often difficult, because the etiology is not identified in each case. Treatment should be aggressive and prompt to maintain the transparency of the ocular structures. Medications should be slowly reduced in frequency once clinical signs abate. Therapy can last for weeks or months and should not be stopped abruptly or recurrence may occur. Overall, the prognosis for ERU is usually poor for a cure to preserve vision, but the disease can be controlled. The Appaloosa breed seems to suffer from the most severe cases.\(^1,4\)

Some horses require life-long therapy!

It is imperative to immediately differentiate a painful eye in a horse as a result of ulcerative keratitis or stromal abscission from the pain associated with ERU by employing a fluorescein dye test. While corticosteroids are the treatment of choice for ERU, they can lead to the rapid demise of an eye with a corneal ulcer or abscess.

Anti-inflammatory medications, specifically corticosteroids and nonsteroidal drugs, are used to control the generally intense intraocular inflammation that can lead to blindness. Medication can be administered topically as solutions or ointments, subconjunctivally, orally, intramuscularly, and/or intravenously. Prednisolone acetate or dexamethasone should be applied topically initially. Systemic corticosteroids may be beneficial in severe, refractory cases of ERU, but pose some risk of inducing laminitis.

The NSAIDs can provide additive anti-inflammatory effects to the corticosteroids and are effective at reducing the intraocular inflammation when a corneal ulcer is present. Flunixin meglumine, pethynbutazone, or aspirin are frequently used systemically to control intraocular inflammation. Some horses become refractory to the beneficial effects of these medications, and it may be necessary to switch to one of the other NSAID to ameliorate the clinical signs of ERU. Cyclosporine A, an immunosuppressive drug, can be effective topically for ERU.

Topical atropine minimizes synechiae formation by inducing mydriasis and relieving spasm of ciliary body muscles. It also reduces capillary leakage. Although topically administered atropine can last several days in the normal equine eye, its effect may be only a few hours in duration in the inflamed ERU eye. The ease with which mydriasis can be achieved with intermittent use of atropine is an important indication as to the stimulus intensity of the ERU. Gut motility can be markedly reduced by...
atropine in some horses. Should gut motility decrease during treatment with topically administered atropine, one can either discontinue the drug or change to the shorter acting tropicamide.

The use of systemically and topically administered antibiotics is often recommended for ERU. Antibiotics should be broad spectrum and appropriate for the geographic location of the patient. Topical antibiotics are indicated in cases of uveitis caused by penetrating ocular trauma or ulcerative keratitis. Antibiotic treatment for horses with positive titers for Leptospira remains speculative but streptomycin (11 mg/kg, q12h, IM) may be a good choice for horses at acute and chronic stages of the disease. Penicillin G sodium (10,000 U/kg, q6h, IV or IM) and tetracycline (6.6–11 mg/kg, q12h, IV) at high dosages may be beneficial during acute leptospiral infections.

Tissue plasminogen activator (TPA) has been used to accelerate fibrinolysis and clear hypopyon in the anterior chamber of horses with severe iridocyclitis. An intracameral injection of 50–150 μg/eye can be made at the limbus with a 27-gauge needle under general anesthesia. TPA should be avoided if recent hemorrhage (<48 h) is present.

Alternative Therapy for ERU

Homeopathic remedies (e.g., poultices of chamomile and oral methylsulfonylmethane) for ERU have been used. Acupuncture has been used to treat ERU.8,9

Surgical Considerations for ERU

In addition to medical treatment, pars plana vitrectomy in horses with ERU has been used successfully to remove fibrin, inflammatory cells, and debris trapped in the vitreous to improve vision and delay the progression of the clinical signs. Vitrectomy seems more beneficial in European Warmbloods with ERU than in Appaloosas with ERU in the United States. The reasons for this are not known. Cataract formation and acceleration occur in up to 50% of eyes post-vitrectomy in both regions. Retinal detachment can also occur postoperatively after vitrectomy.22

Sustained release intravitreal cyclosporine A implants may also be beneficial to treating ERU. Cyclosporine A inhibits T-lymphocytes to suppress the immune response of the leukocytes present in the horse uveal tract. This is a physiologically sound approach to ERU therapy. Implants may last 5 yr. Cataract acceleration is also a problem with this technique.

11. Equine Glaucoma

The glaucomas are a group of diseases resulting from alterations of aqueous humor dynamics that cause an intraocular pressure (IOP) increase above that which is compatible with normal function of the retinal ganglion cells and optic nerve.1,4

Horses with previous or concurrent uveitis, aged horses, and Appaloosas are at increased risk for the development of glaucoma.1,4 Iris and ciliary body neoplasms can cause secondary glaucoma. Congenital glaucoma is associated with developmental anomalies of the iridocorneal angle in foals.

The infrequency of diagnosis in the horse may be caused, in part, by the limited availability of tonometers in equine practice, but also to the fact that large fluctuations in IOP, even in chronic cases, may make documentation of elevated IOP difficult (Fig. 14). Dilated pupils, corneal band opacities, decreased vision, lens luxations, mild iridocyclitis, and optic nerve atrophy/cupping may also be found in eyes of horses with glaucoma.

The topically administered carbonic anhydrase inhibitor dorzolamide (q12h), and the beta-blocker timolol maleate (0.5% q12h) have been used to lower IOP in horses with varying degrees of success. The newer prostaglandin derivatives cause low grade uveitis and may exacerbate the IOP in horses with glaucoma. Topical atropine therapy was once thought to reduce the incidence of glaucoma in horses with uveitis, but should be used cautiously in horses with glaucoma as it may cause IOP spikes.

Laser destruction of the ciliary body (cyclophotoagulation) works the best at controlling IOP and preserving vision in horses.

Contraindications/Possible Interactions

Conventional glaucoma treatment with miotics may provide varying amounts of IOP reduction in horses. Miotics and prostaglandins can potentiate the clinical signs of uveitis and should be used cautiously in horses with anterior uveitis. The horse eye seems to tolerate elevations in IOP for many months to years that would blind a dog; however, blindness is the end result. Buphthalmia can be associated with exposure keratitis.
12. Retinopathies

Chorioretinitis

Chorioretinitis is inflammation of the choroid and retina. Inactive lesions are more often reported than active lesions. The tapetal region is rarely affected. It is manifested in equine eyes as focal “bullet-hole” retinal lesions, diffuse chorioretinal lesions, horizontal band lesions of the nontapetal retina, and chorioretinal degeneration near the optic nerve head. Active chorioretinitis appears as focal white spots with indistinct edges and as large diffuse gelatinous grey regions of retinal edema. Inactive chorioretinitis can appear as circinate depigmented regions with hyperpigmented centers, or large areas of depigmentation that appear in some cases as the wings of a butterfly. Chorioretinitis may be found with or without the signs of anterior uveitis found with ERU.

Lesions can be caused by infectious agents (e.g., leptospirosis, EHV-1, *Onchocerca cervicalis*), immune-mediated uveitis of unknown origin, trauma, or vascular disease. Systemic NSAID medication is administered for chorioretinitis. Fluimixin meglumine, phenylbutazone, or aspirin are indicated. Topical medication does not reach the retina and is only indicated if signs of anterior uveitis are also present.

Congenital Stationary Night Blindness

Congenital stationary night blindness (CSNB) is found mainly in the Appaloosa and is inherited as a sex-linked recessive trait. Cases are also noted in Thoroughbreds, Paso Finos, and Standardbreds. CSNB seems to be caused by a functional abnormality of neurotransmission in the middle retina. Clinical signs include visual impairment in dim light with generally normal vision in daylight and behavioral uneasiness and unpredictability occurring at night. CSNB does not generally progress, hence its name, but cases of progression to vision difficulties in the daytime are noted. Ophthalmoscopic examination is normal.

Diagnosis is by clinical signs, breed, and electroretinogram (ERG) with decreased scotopic b-wave amplitude and a large negative, monotonic a-wave. There is no therapy for this condition but affected animals should not be bred.

Retinal Detachments

Retinal detachment is a separation of the nine layers of the sensory retina from the retinal pigment epithelium (RPE). It is associated with slowly progressive or acute blindness in horses. It can be congenital in newborn foals or acquired later in life in adults. Retinal detachments can occur bilaterally or unilaterally, and be partial or complete. Retinal detachments are a complication of ERU and are also associated with microphthalmos, head trauma, perforating globe wounds, cataract surgery, and may be secondary to intraocular tumors.

Complete retinal detachments are seen clinically as free-floating, undulating, opaque veins in the vitreous overlying the optic disc. The tapetum is hyperreflective. If the media of the eye are so opaque (e.g., corneal edema, cataract) that the fundus cannot be visualized, b-scan ultrasound can be used to diagnose the classic “seagull sign” of retinal detachment.

Laser surgery and pneumatic retinopexy to reattach the retina are well described for the dog, but have not yet been reported for the horse.

14. Sudden Blindness

Acute blindness may be associated with head or ocular trauma, ERU, glaucoma, cataracts, intraocular hemorrhage, exudative optic neuritis, retinal detachment, or CNS disease. Acutely blind horses are extremely agitated, anxious, and dangerous. Horses recovering from anesthesia after enucleation of sighted eyes for SCC can be very confused and agitated in the postoperative period. Extreme care should be used and the animals approached cautiously on the blind surgical side until the horse adapts to its condition.

Horses can adapt amazingly well to blindness, whether unilateral or bilateral, if allowed to adjust to their new condition. Several internet websites are devoted to the care of blind horses and other blind animals.

15. Eye Diseases Associated with Specific Horse Breeds

Appaloosa

1. CSNB
2. Congenital cataracts
3. Glaucoma
4. ERU
5. Optic disc colobomas

Arabian

1. Congenital cataracts

Belgian Draft Horses

1. Aniridia and secondary cataracts
2. Cataracts

Morgan

1. Cataracts—nuclear, bilateral, symmetrical, and non-progressive

Quarter Horse

1. Congenital cataracts
2. Entropion

Rocky Mountain Horse

The chocolate coat color is most often affected. Collectively the cornea, iris, and ciliary body lesions are termed anterior segment dysgenesis.
1. Congenital miosis and corpora nigra and iris hypoplasia
2. Macrocornea
3. Ciliary cysts
4. Cataract, lens luxation
5. Retinal dysplasia, retinal detachment

Thoroughbred

1. Congenital cataracts
2. Microphthalmia associated with multiple ocular defects
3. Retinal dysplasia associated with retinal detachments in some cases
4. Entropion
5. Progressive retinal atrophy

Color Dilute Breeds

1. Iridal hypoplasia—photophobia

Standardbreds

1. Retinal detachments
2. CSNB

Paso Fino

1. CSNB
2. Glaucoma

American Saddlebred

1. Cataracts
2. Aggressive keratomycosis

Warmbloods

1. Glaucoma
2. ERU

Miniature Horses

1. Cataracts

Mules

1. Aggressive sarcoïds

References