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Eye Conditions - Dec 9th, 03

VISION IN THE HORSE: WHAT DOES THE HORSE "SEE"?

The horse has a total visual field of nearly 360 degrees, meaning a horse can just about see its tail with its head pointed forward. A small frontal binocular field of 65 degrees develops post-natally. The horse's retina is adapted for detection of movement, and the horse utilizes both eyes until an object approaches within 3-4 feet, when it is forced to turn or lower its head to continue to observe with one eye. Cones are present in the horse's retina suggesting that they have the capacity for color vision, in the form of blues and reds.

OCULAR PROBLEMS IN THE FOAL

A newborn foal may exhibit droopy eyelids, low tear secretion, a round pupil, reduced corneal sensitivity, lack of a menace reflex for up to two weeks and prominent lens sutures. Entropion is an inward rolling of the eyelid margin. This causes the eyelid hairs to rub on the cornea. It can be a primary problem in foals, or secondary to dehydration or emaciation as in "downer foals." It may be repaired to prevent corneal ulceration in the neonate by placing sutures at the lid margin to roll out the offending eyelid margin.

Congenital cataracts in foals are common congenital eye defects. Surgery is recommended. Microphthalmos or a small eye is a common ophthalmic congenital defect in the foal. A range of lesions may be present. The microphthalmic eye may be visual or associated with other eye problems that cause blindness.

Iridocyclitis or uveitis in the foal is generally secondary to severe illness and may be in one or both eyes. Proteins, red cells and white cells may be present. Severe unilateral, blinding fibrinous uveitis secondary to plant toxins has been noted in primarily Thoroughbred foals and yearlings in the southern U.S.

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 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 due to their extensive blood supply, and an eyelid "tag" or pedicle flap should never be excised, as exposure keratitis and corneal ulceration can result.

Upper eyelid damage is more significant in horses because the upper lid moves over more of the

equine cornea than does the lower lid. Preservation of the eyelid margin is critical if at all possible in order to preserve eyelid function. The repaired lesion must be protected from "self-trauma" with masks or hard cups.

Neoplasia of the lids

Eyelid melanomas are found in grey horses, with Arabians and Percherons also at increased risk. Melanomas may be single or multiple. Treatment is cimetidine, surgical excision and/or cryotherapy.

Sarcoids are solitary or multiple tumors of the eyelids and periocular region of the horse. Retroviruses and papilloma viruses may be involved in the etiology. It is suspected that flies may be able to transfer sarcoid cells from one horse to traumatic skin lesions in other horses. There are geographic differences in the aggressiveness of the sarcoid in horses. Mules appear to suffer from an aggressive form of sarcoids. Immunotherapy for sarcoids includes using attenuated *Mycobacterium bovis* cell wall extracts such as the immunostimulant Bacillus Calmette-Ga erin (BCG). Shrinking the sarcoid lesion with antipsoriasis skin ointments and/or topical 5-fluorouracil (5-FU) for two weeks may be beneficial before using BCG. Cryotherapy, hyperthermia, carbon dioxide laser excision, intralesional chemotherapy and intralesional radiotherapy can also be effective treatments for sarcoids. Intralesional chemotherapeutics including 5-FU or cisplatin have been used with varying success rates. Homeopathic ointments and caustic chemical lotions are effective in treating some sarcoids.

Squamous cell carcinoma (SCC) is the most common tumor of the eye and lids in horses. The cause 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. Prevalence in horses increases with age with the mean age at diagnosis 11.1 plus or minus (???) 0.4 years in one report. Belgians, Clydesdales and other 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, grey-white and palomino hair colors predispose to ocular SCC, with less prevalence in bay, brown and black hair coats. Cryotherapy, immunotherapy, irradiation, radio-frequency hyperthermia, CO₂ laser ablation or intralesional chemotherapy should follow surgical excision of equine ocular SCC.

DISEASES OF THE CORNEA

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 uveitis 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.

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. 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. 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. Healing of large-diameter, superficial, noninfected corneal ulcers is generally rapid and linear for five to seven 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, non-infected corneal wound is 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. 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. Excessive proteinase activity is termed "melting," and results in a liquefied, grayish-gelatinous appearance to the stroma near the margin of the ulcer. 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. 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. Corneal cultures should be obtained first and then followed by corneal scrapings for cytology. Mixed bacterial and fungal infections can be present.

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.

Antibiotics

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.

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 melting. Serum is biologically nontoxic and contains an alpha-2 macroglobulin with antiproteinase activity. 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 eight 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. 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.

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 anti-inflammatory drugs (NSAIDs) such as profenol, flurbiprofen and diclofenamic acid (BID to TID) can also reduce the degree of uveitis. Horses with corneal ulcers and secondary uveitis should be stall-rested until the condition is healed. Intraocular hemorrhage and increased severity of uveitis are sequelae to overexertion.

Conjunctival flaps

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

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 EYE RUPTURE TOPICAL CORTICOSTEROIDS ARE BAD WHEN THE CORNEA RETAINS FLUORESCEIN STAIN UVEITIS CAUSED BY A CORNEAL ULCER OR STROMAL ABSCESS MAY BE VERY DIFFICULT TO CONTROL

FUNGAL ULCERS IN HORSES

Fungi are normal inhabitants of the equine environment and conjunctival microflora but can become pathogenic following corneal injury. Aspergillus, Fusarium, Cylicostephanospora, Curvularia, Penicillium, Cystodendron, yeasts and molds are known causes of fungal ulceration in horses. Saddlebreds appear to be prone to severe keratomycosis, while Standardbreds are resistant. 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.

CORNEAL STROMAL ABSCESSSES

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. Medical therapy consists of aggressive use of topical and systemic antibiotics, topical atropine and topical and systemic NSAIDs. 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, cytokines and toxins from degenerating leukocytes in the abscess.

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.

Equine Cataract Surgery

Most veterinary ophthalmologists recommend surgical removal of cataracts in foals less than 6 months 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.

Phacoemulsification cataract surgery is the most useful technique for the horse. This extracapsular procedure through a 3.2mm corneal incision utilizes a piezoelectric handpiece with an ultrasonic titanium needle in a silicone sleeve to fragment and emulsify the lens nucleus and cortex following 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 phacoemulsification cataract surgery, and there is a quicker return to normal activity with phacoemulsification than other surgical techniques. The results of cataract surgery in foals by experienced veterinary ophthalmologists are generally very good, but the cataract surgical results in adult horses with cataracts caused by ERU are often poor. The problem is that new blood vessels form on the iris and anterior lens capsule in the eyes with ERU, and they can bleed during the surgeries. The surgeon often cannot stop the hemorrhage and severe hyphema results.

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. It is a group of immune-mediated diseases of multiple origins. Recurrence of anterior uveitis is the hallmark of ERU. The disease is bilateral in approximately 20 percent of cases. While the pathogenesis is clearly immune-mediated, the specific causes of ERU are unknown. Hypersensitivity to infectious agents such as *Leptospira interrogans* is commonly implicated as a possible cause. Leptospiral titers for *L. pomona*, *L. bratislava* and *L. autumnalis* should be requested in the U.S.

Positive titers for serovars of 1:400 or greater are of importance. Serology for *Leptospira pomona* can be used for prognostic evaluation of the likelihood of blindness occurring in one or both eyes. Seropositive Appaloosas (100%) > seronegative Appaloosas (72%) > seropositive non-Appaloosas (51%) > seropositive non-Appaloosas (34%) at having blindness occur in at least one eye within 11 years of the first attack.

A complete ophthalmic examination should be performed to determine if the uveitis is associated with a corneal ulcer. The presence of a corneal ulcer precludes the use of topical corticosteroids, but not topical nonsteroidal drugs. Inflammation of the brain is found in ERU. Irreversible blindness is a common sequelae to ERU and is due to retinal detachment, cataract formation or severe chorioretinitis.

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, as the etiology is not identified in each case. Treatment should be aggressive and prompt in order 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. Some horses require life-long therapy! 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.

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 initially. When the frequent application of topical steroids is not practical, the use of subconjunctival corticosteroids may be used. Systemic corticosteroids may be beneficial in severe, refractory cases of ERU, but pose some risk of inducing laminitis and should be used with caution. The nonsteroidal anti-inflammatory drugs (NSAID) can provide additive anti-inflammatory effects to the corticosteroids, and are effective at reducing the intraocular inflammation when a corneal ulcer is present. Cyclosporine A can be effective topically for ERU. Flunixin meglumine, phenylbutazone 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. Topical atropine minimizes synechiae formation by inducing mydriasis and alleviates some of the pain of ERU by relieving spasm of ciliary body muscles.

Surgical considerations for ERU

Vitrectomy appears more beneficial in European warmbloods with ERU than in Appaloosas with ERU in the U.S. The reasons for this are not known. Cataracts occur in a high percentage of cases post-vitrectomy in both regions. Retinal detachment can also occur postoperatively. Sustained release intravitreal cyclosporine A implants may also be beneficial to treating ERU.

RETINOPATHIES

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. 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 ERG with decreased scotopic b-wave amplitude and a large negative, monotonic a-wave. CSNB appears to be caused by functional abnormality of neurotransmission in the middle retina. There is no therapy for this condition but affected animals should not be bred.

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

EYE DISEASES ASSOCIATED WITH SPECIFIC HORSE BREEDS

APPALOOSA

1. Congenital stationary night blindness (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 (*chocolate coat color 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

STANDARD BREEDS

1. Retinal detachments
2. Congenital Stationary Night Blindness

PASO FINO

1. CSNB
2. Glaucoma

AMERICAN SADDLEBRED

1. Cataracts
2. Aggressive keratomycosis

WARM BLOODS

1. Glaucoma
2. ERU

MINIATURE HORSES

1. Cataracts MULES: Aggressive sarcoids

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