Ophthalmologic Disorders in Aged Horses

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KEYWORDS

• Geriatric • Horse • Eye • Uveitis • Blind

KEY POINTS

- Ocular abnormalities are common in aged horses.
- Superficial nonhealing corneal ulcers seem more prevalent in older horses, perhaps as a result of decreased corneal sensitivity.
- Significant ocular disorder as a result of recurrent uveitis can manifest more clearly as horses age. Important consequences of recurrent uveitis are cataracts and glaucoma.
- Several retinal and vitreal abnormalities are commonly seen in old horses, with variable effects on vision.

INTRODUCTION

Ophthalmologic disease seems to be common in geriatric animals. Analysis of the records of a large number of geriatric (≥20 years old) horses admitted to an American veterinary hospital indicated that 11% had ocular disease.[1] However, this prevalence increased when the general equine geriatric population was considered. Studies in the United Kingdom have shown that 94% of horses 15 years of age or older had at least 1 ocular abnormality detected by a veterinarian.[2–4] This number increased to 100% when only horses 30 years of age or older were examined.[5] Only approximately 3.5% of owners reported any ocular problems in these horses, and 10% noted ocular discharge. Survey studies in Australia have also shown a high prevalence of ocular disease; 22.3% of horses 15 years of age and older in Queensland were reported to have ocular discharge, making this the fourth most common clinical sign mentioned by owners.[6] A clear positive correlation was noted between the presence of ocular discharge and increasing age. Again, although ocular discharge seemed to be common, only 3.3% of owners reported ocular problems in their horses and only 2.6% perceived eyesight as an important health issue. It seems that a large number of aged horses may have undetected ocular disease that could be a source of chronic

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low-grade discomfort. In addition, some of these ophthalmic abnormalities may have a significant effect on the horses' vision, with important human safety and animal welfare implications. This article reviews the most common ocular abnormalities in geriatric horses.

CORNEAL DISEASE

Corneal disease is a common problem in equine practice.[7] In a study assessing the prevalence of disease in a geriatric (≥15 years old) population, corneal lesions were detected in 2.6% of the horses examined.[3] These abnormalities included corneal edema, opacities, and scarring.

As part of the aging process, changes occur that affect the ocular surface. In humans, the lacrimal gland has been shown to decrease its secretion with age.[8] Tear deficiency and evaporative dry eye syndromes are rare in horses,[9] but these may become more common with increasing age.[10] Although studies have shown no difference in the amount of tear production between young and old horses,[11] orbital fat loss may result in enophthalmos and inadequate spreading and stability of the tear film.[10] In addition, the composition of the tear film also varies. The levels of lactoferrin and lysozyme, two potent antimicrobial agents, have been shown to decrease with age.[8,12] Other factors can also influence the immune response on the ocular surface in older animals, such as a reduced phagocytic activity of polymorphonuclear leukocytes and impaired T-cell function.[12] This combination of factors can increase the susceptibility to microbial disease, especially keratomycosis, and may make these conditions more difficult to treat as horses get older. For these reasons, it may be advisable to select bactericidal rather than bacteriostatic antimicrobials when treating bacterial keratitis. In addition, topical corticosteroids should be used with caution where these are warranted for ocular conditions.[12]

Superficial ulcerative keratitis is one of the most commonly observed ophthalmic problems in horses. In most cases the ulceration heals without complications in 24 to 72 hours[13]; however, on some occasions these ulcers show a prolonged healing time or fail to epithelize. These superficial, nonhealing corneal ulcers are characterized by the presence of chronic (>1 week) ulceration with redundant, loose epithelial borders and no evidence of stromal involvement, infectious agents, or inflammatory cellular infiltrate[7,13–15] (**Fig. 1**). Superficial nonhealing corneal ulcers can

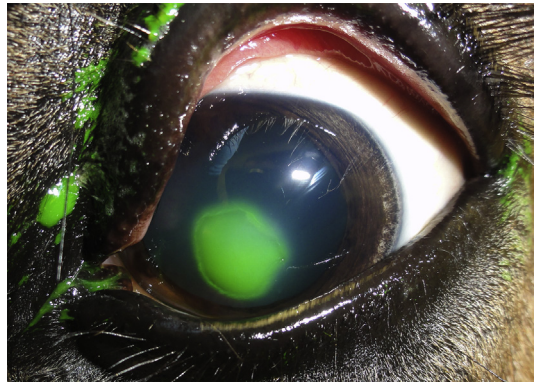

Fig. 1. Superficial, nonhealing, corneal ulcer in a 15-year-old mare. Note the poorly defined ulcer margins and the underrunning of fluorescein beyond the edge of the ulcer, indicating poorly adhered epithelium.

affect horses of any age but they seem to be more common in middle-aged to aged patients.[7,13,14] The reason for this increased prevalence as horses get older is probably multifactorial. Keratocyte density seems to be higher in younger individuals than in adults[16] and there is also thickening of the epithelial basement membrane with age,[8] which may contribute to the delayed healing. Perhaps more importantly there is a decrease in the nerve density at the level of the sub-basal plexus, below the epithelium.[8] The cornea is the most densely innervated tissue of the body, and these nerve fibers exert trophic influences on the corneal epithelium, stimulating the growth, proliferation, and differentiation of the epithelial cells and the production of type VII collagen.[11] Reduction in corneal sensitivity can result in epithelial defects and ulceration. Studies assessing the corneal touch threshold with a Cochet-Bonnet esthesiometer showed a significant decrease in corneal sensitivity between young (<10 years) and old (>15 years) horses, and this decrease was more marked if the older horses were showing clinical signs of pituitary pars intermedia dysfunction.[11]

Superficial, nonhealing corneal ulcers can represent a clinical challenge and numerous surgical treatment options are commonly required, such as debridement of the elevated epithelial edges manually or with a diamond burr, punctate or grid keratotomy, and/or superficial keratectomy with or without conjunctival flaps.[7,13,14] Anecdotally, some cases seem to respond better to treatment when serum from a young horse is applied topically on the ocular surface.[10]

With aging there is also a decrease in the number and density of corneal endothelial cells.[8,16] These cells are essential for maintaining the dehydrated status of the cornea. Animals are born with a fixed number of corneal endothelial cells, and this number decreases gradually with age. Because these cells do not divide, cell loss induced by age or disease cannot be reversed. This cell loss leads to corneal edema and loss of corneal transparency. In addition, this accumulation of fluid can induce the separation of the corneal epithelium from the underlying stroma in the form of small blisters known as bullae[8] that may also affect ulcer healing. Primary corneal endothelial dystrophy has been reported as a cause of age-related corneal edema in horses, frequently presenting clinically as a central vertical band[17] (**Fig. 2**). This condition should be differentiated from other potential causes of corneal edema, such as glaucoma, uveitis, or traumatic injury.[10]

Fig. 2. Central, vertical band of corneal edema in an otherwise normal eye of a 19-year-old warmblood gelding. Serial measurements of the intraocular pressure have always remained within normal limits.

UVEITIS

Equine recurrent uveitis (ERU) is a spontaneous disease characterized by repeated episodes of intraocular inflammation.[18] Although a uveitic episode can occur at any age, a clear correlation ($r = 0.983$) has been found between age and the occurrence of ERU.[19] One study in the United Kingdom found uveal abnormalities in 23.4% of horses 15 years of age and older,[3] although another study in Germany detected signs of ERU in almost a third of horses from the same age group. In the United States, the mean age at presentation for ERU was 11.6 and 13.3 years depending on whether the horses were seropositive or seronegative to *Leptospira* spp respectively.[20] Signs of acute anterior uveitis include ocular pain, blepharospasm, lacrimation, chemosis, corneal changes (including edema, vascularization, and keratic precipitates), aqueous flare, hypopyon, hyphema, marked miosis, and iris color changes. Posterior uveitis can be more difficult to diagnose and is characterized by vitritis with liquefaction of the vitreous, vitreal floaters, and retinal changes. Because of the recurrent nature of the disease, changes associated with previous episodes are sometimes noted in an otherwise quiescent eye; these include corneal scarring, iris depigmentation, synechiae, granula iridica degeneration, cataracts, glaucoma, phthisis bulbi, and fundic changes[10,21] (**Fig. 3**).

In geriatric horses with ERU, because of the potential for a lifetime of accumulated episodes of intraocular inflammation, significant ocular disorder is commonly observed. Secondary complications such as cataracts and glaucoma are frequent (see elsewhere in the text) and vision can be significantly affected. Frequently in these cases treatment is directed to avoid further deterioration and control the painful episodes. Suprachoroid slow-release cyclosporine A implants have been successfully used in patients with ERU to provide long-term control of the condition.[22] Enucleation may be indicated for blind eyes in which the painful episodes cannot be avoided.

GLAUCOMA

Although the estimated prevalence of glaucoma in the general equine population is considered low, geriatric horses are at increased risk, with a recent study documenting that 65% of glaucoma cases presented in horses older than 15 years.[23–29] There

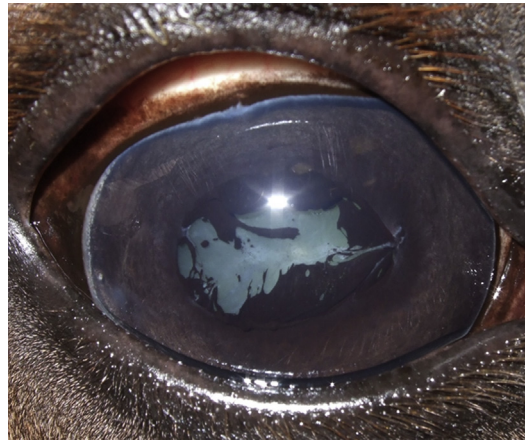

Fig. 3. An 18-year-old Welsh section D gelding showing signs of chronic intraocular inflammation in his right eye. Note the abnormal superior limbal margin, ruptured granula iridica, abnormal pupillary margin with numerous synechiae and dense cataract.

are 2 routes by which the aqueous humor exits the eye: the conventional and the unconventional pathways.[25–29] The conventional pathway refers to aqueous outflow via the iridocorneal angle and trabecular meshwork. With age, the trabecular meshwork changes histologically: the trabecular endothelial cellularity is reduced and the outflow spaces are decreased, which may account for an increase in intraocular pressure observed in older horses.[16] The unconventional pathway makes reference to the drainage of fluid via the uveoscleral route. An age-related decrease in intracellular pores in the scleral venous sinus has been shown, which may also result in a decrease in aqueous flow facility.[16] However, age alone may not explain the increased risk of glaucoma in geriatric horses. Most cases of equine glaucoma are secondary to ERU, in which the deposition of inflammatory cells and debris at the drainage angle and the presence of adhesions and inflammatory fibrovascular membranes affect and limit the outflow of aqueous humor.[24]

Horses seem to tolerate higher intraocular pressures for longer compared with dogs or humans.[24,26–28] In addition, clinical signs of glaucoma in horses tend to be subtle, with little evidence of ocular discomfort, making diagnosis difficult. Signs commonly associated with glaucoma in horses include hydrophthalmos, corneal edema, corneal striae (Haab striae), a mildly dilated pupil, lens luxation, and optic nerve cupping and degeneration (**Fig. 4**). Signs of chronic intraocular inflammation are frequently observed in cases secondary to ERU. The intraocular pressure shows diurnal variation in horses, so, if glaucoma is suspected, repeat measurement may be required.[24–29] Pressures more than 30 mm Hg indicate glaucoma. It is important that, when repeat measurements are taken, these are done in identical circumstances; factors such as head positioning, placement of an auriculopalpebral nerve block, or sedation can greatly affect the measurements.[26,28,30,31]

Medical therapy for glaucoma involves the administration of drugs that reduce aqueous humor production. β-Blockers, such as 0.5% timolol maleate, and carbonic anhydrase inhibitors, such as 2% dorzolamide or 1% brinzolamide, are commonly administered alone or in combination.[24–29] In a large number of cases medical therapy alone is not enough to control the disease; in such cases surgery is indicated. Selective destruction of the ciliary body with laser (cyclophotoablation) transsclerally is intended to reduce aqueous humor production, whereas placement of gonioimplant shunts increase aqueous outflow.[32–37] In addition to specific glaucoma treatment,

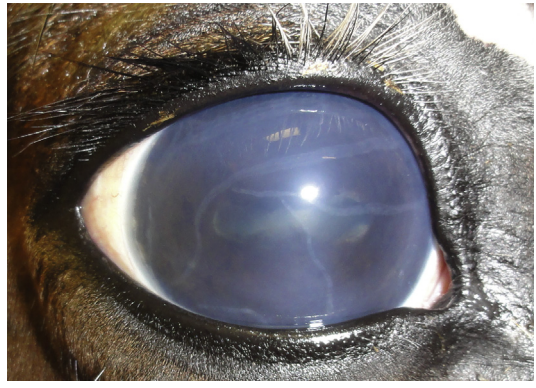

Fig. 4. Glaucomatous eye in a 19-year-old mare. There is diffuse corneal edema and numerous striae (Haab striae) caused by thinning of the Descemet membrane. The intraocular pressure at the time of diagnosis was 47 mm Hg. Note the abnormal pectinate ligament temporally.

therapy for the potential underlying ERU is essential. Chronically painful and blind glaucomatous eyes should undergo chemical ablation of the ciliary body by intravitreal injection of gentamicin or should be enucleated.[26]

THE LENS

The lens continues to grow throughout life by the sequential layering of new fibers around its nucleus.[38,39] Nuclear sclerosis, defined as the altered refractivity of the central lens observed in older animals caused by the compression of the central nucleus by enveloping fibers as part of the normal lens growth, has not been definitively described in horses.[12,38,39] However, cataracts, defined as any opacity or alteration in the optical homogeneity of the lens, are common in horses.[38] Cataracts were common findings in studies with geriatric horses and were present in up to 58.5% of horses more than 15 years of age.[3,40] When only those horses older than 30 years were considered, the prevalence of cataracts increased to 97%.[5]

The lens is rich in proteins called crystallines, which ensure its transparency.[38] Lens epithelial cells lose their organelles as they differentiate into fibers and with them their biosynthetic capacity. Therefore, lens fibers have a limited capacity to restore crystallines that may become damaged during the aging process.[41] Under normal conditions, the lens experiences years of exposure to factors such as ultraviolet light and chemical insult that are well known to destabilize proteins.[41] In addition, there is a loss of the redox balance of the major antioxidant system of the lens fibers. Once these crystallines are denatured, they condense into aggregates that induce light scatter and cause the white appearance of the lens in horses with cataracts.[41]

Typically, early senile cataracts appear as microvesiculation in the posterior suture lines. In older animals this progresses to more dense condensation around the posterior suture, together with perinuclear and cortical (anterior and posterior) cataracts.[12,38] Complete and occasionally hypermature cataracts can occur in some cases, with animals experiencing more significant visual compromise[12] (**Fig. 5**). Senile cataracts are usually bilateral but not necessarily symmetric.[38]

Although the use of antiinflammatories and antioxidants has been examined, the only treatment of cataracts is surgical removal of the lens.[38] Phacoemulsification is the technique of choice for lens removal in horses, with or without implantation of

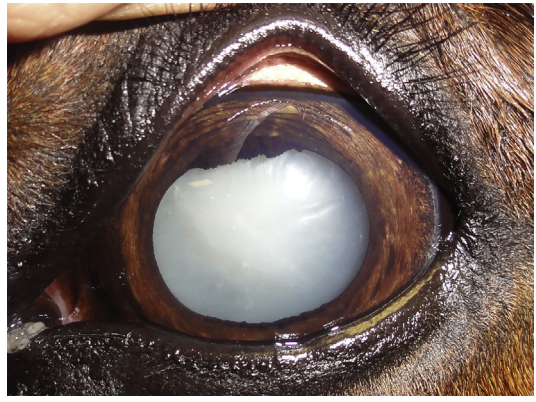

Fig. 5. Complete cataract in the left eye of an 18-year-old Irish draught mare. The lens was removed by phacoemulsification. Fourteen months after surgery the eye remains comfortable and visual. A faint perinuclear cataract was also present in the right eye in this mare.

an acrylic intraocular lens. However, surgery is typically reserved for those horses with a significant visual impairment.[42] Although the success rate for vision in the immediate postoperative period seems to be good, a recent study suggested that only 26.3% of horses remained visual 2 years after surgery.[43] However, this study considered horses lost to follow-up as nonvisual and therefore the long-term success rate may be higher. Another recent study found age not to be significantly associated with poorer outcomes following phacoemulsification, although horses older than 15 years had a lower visual outcome.[44] Intraocular inflammation is a potent cause of cataracts and therefore cataracts are a common consequence of ERU.[38] Horses with preoperative uveitis are significantly less likely to remain visual following phacoemulsification.[44]

THE VITREOUS

The vitreous occupies a great part of the ocular volume and has several important functions, including metabolic support and oxygenation, removal of the metabolic waste, and light transmission to the retina.[12,40] The vitreous is a clear, gel-like substance with a network of collagen fibrils that extend throughout the gel.[41] With aging, likely because of alteration or degradation of this collagen fiber network, there is a tendency for the gel to collapse; the vitreous attachments to the retina weaken and the space is filled with liquefied vitreous (termed syneresis).[16,41] In horses, true age-related liquefaction only happens in extreme old age; however, progressive dilution of the gel throughout life may give the impression of liquefaction from a fairly early age.[12] Two different studies in the United Kingdom found vitreous degeneration to be the most common ocular abnormality in geriatric horses, with a prevalence of 46% and 66% respectively.[3,40] Vitreous degeneration shows as clouding or discoloration of the vitreous gel with membranous or cellular debris that appears suspended within the vitreous body, sometimes termed floaters or muscae volantes.[12,45] In addition, inflammatory debris, often as a result of ERU, can contribute to vitreous opacity.[40]

Two other conditions of the vitreous seem to be more common in older horses. Asteroid hyalosis is a rare finding and manifests as white or refractive lipoid deposits within the vitreous gel structure, approximately 1 to 2 mm in size. These asteroid bodies remain suspended in the vitreous body but on occasions they move when the globe moves.[10,12,45] Synchysis scintillans or cholesterosis bulbi is also a rare condition, showing as multiple, highly refractile, golden particles formed by cholesterol crystals that flutter upward and then float back down with eye motion.[10,12,45]

THE RETINA

Several changes have been observed in the retinas of humans with aging. There is thickening of the internal limiting membrane and a decrease in the neural elements of the retina. The retinal pigment epithelium (RPE) may migrate into the sensory retina. The RPE may atrophy in areas surrounded by RPE hypertrophy and hyperplasia, thought to be as a result of insufficiency of the choroidal vasculature. There is also a gradual, age-related loss of rods.[16] Two significant changes have been observed in equine retinas of older horses compared with young horses. Large vacuoles, caused by bullous elevation of the epithelial cell layer, were detected at the level of the pars ceca retinae. The second change, observed in almost half of the retinas studied, was degeneration of the pars optica retinae with complete loss of the normal structure, affecting the first 0.5 mm of the retina from the border of the ora serrata.[46]

Senile retinopathy is the most common retinal condition found in older horses. Studies have shown a prevalence of between 33% and 42% in the general geriatric

population; however, this prevalence increased to 73% when only horses older than 30 years were considered, suggesting a progressive nature.[3,5,40] In addition, the median age of horses affected bilaterally is significantly higher than the age of animals with unilateral lesions.[3] Senile retinopathy affects the retinal pigmented epithelium and photoreceptor layer and appears as irregular linear hyperpigmentation surrounded by hypopigmented areas, affecting typically the nontapetal fundus, although the tapetal fundus can also be involved[10,45,47,48] (**Fig. 6**). The pathogenesis of senile retinopathy is not fully understood but possible causes include oxidative damage or choroidal vasculature disease.[40] There is some debate as to whether senile retinopathy causes visual deficits or not. Some investigators think that this alteration is of no clinical significance, whereas others report problems with vision in the affected animals, particularly in poor lighting conditions.[12,47]

Inactive chorioretinitis lesions typically present as focal depigmented areas (so-called bullet-hole lesions) or as more extensive areas in the peripapillar region (butterfly lesions).[10,45,47,49] These lesions can be frequently found in young animals, but because they represent chorioretinal scars from previously active lesions is it likely that they present more commonly as horses get older. On some occasions they can cause visual deficits, but most often they represent an incidental finding.

Nonneoplastic masses of the optic nerve head also seems to be more common in older horses. Proliferative optic neuropathy appears as a white or pink lobulated mass at the edge of the optic nerve (**Fig. 7**). It is normally an incidental finding, although it may affect vision if it is big enough to obstruct the optic nerve head or central retina or when it results in continued movement (causing shying behavior).[10,45,47,48]

Exudative optic neuritis presents as sudden-onset bilateral blindness in horses more than 15 years of age.[10,47] On fundoscopic examination a lot of material can be seen extending from the surface of the optic nerve head towards the vitreous, with or without focal hemorrhages. If the optic disc is visible, it will appear swollen and edematous.

NEOPLASIA

Squamous cell carcinoma is the most common tumor of the eye and ocular adnexa in horses and there is an increased prevalence with age.[50] Risk factors include increased exposure to ultraviolet light and periocular hypopigmentation, although

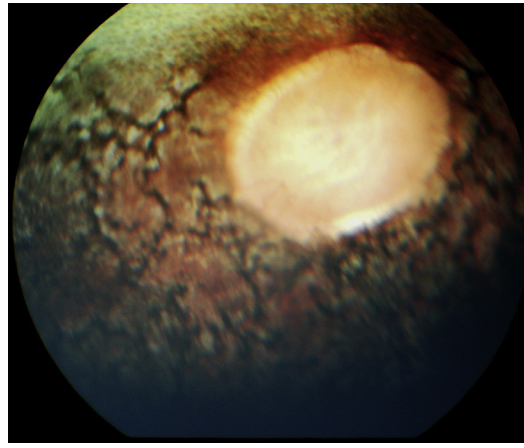

Fig. 6. Senile retinopathy.

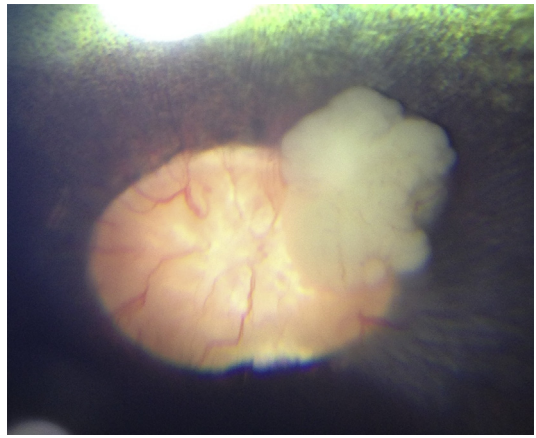

Fig. 7. Proliferative optic neuropathy in the right eye of a 15-year-old mare. This mare presented for examination for an unrelated condition. The owner reported no visual deficits and this was considered an incidental finding.

draught horse breeds with darkly pigmented periocular structures also show a higher incidence of the condition. In addition, geldings are 5 times more likely to develop ocular squamous cell carcinoma than stallions and twice more likely than mares.[50,51] The 2 most common locations are the third eyelid and the limbal conjunctiva[12] (**Fig. 8**) but any epithelial structure can be affected. Treatment of these tumors is challenging and frequently involves surgical excision followed by adjunctive therapy, including radiation, chemotherapy, and/or cryotherapy.[52] Recurrence following treatment is common.[10]

Melanoma is a common tumor in mature grey-coated or white-coated horses,[12,53,54] although it can affect horses with any coat color, and frequently affects the eyelids or other periocular structures. In contrast, most cases of intraocular melanoma tend to occur in younger horses between 5 and 10 years of age.[55] Early surgical removal of solitary early-stage melanomas affecting the eyelid is the recommended treatment[53,54] (**Fig. 9**). Other treatment options, with variable success rates reported, include intratumoral chemotherapy, immunotherapy, and cimetidine administration[53,54,56]

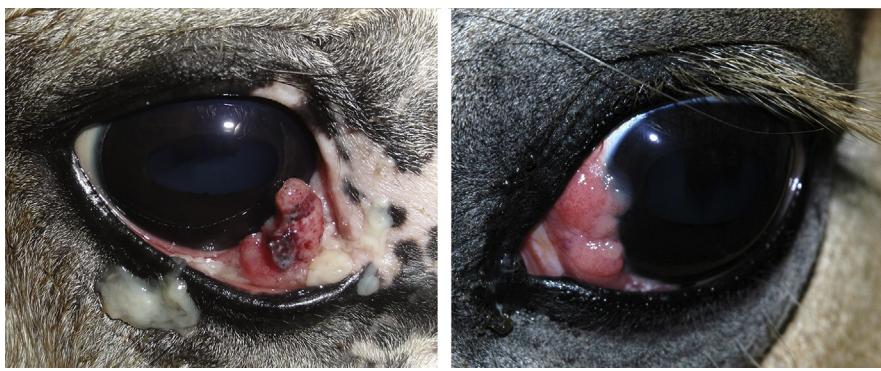

Fig. 8. Squamous cell carcinomas affecting the third eyelid of a 15-year-old Appaloosa gelding (*left*) and the nasal corneolimbal margin of a 17-year-old Haflinger mare (*right*).

Fig. 9. Melanoma affecting the lower eyelid of a 16-year-old mare before (*left*) and after (*right*) surgical resection.

DISTURBANCES OF VISION

Objectively assessing vision in horses remains a challenge. Horses with significant ocular disease frequently show no behavioral changes that indicate visual compromise,[57] especially if the vision loss has developed over a long period of time.[2,57] This finding is particularly important in geriatric animals; these horses are usually managed in such a way, being kept in familiar surroundings and with a regular routine, that owners may miss early cues to visual impairment. The prevalence of owner-reported visual deficits in geriatric horses varies between 3.9% and 8% according to several studies,[2,4,40] but this number increased to 19% if only horses older than 30 years were considered.[5] Of these horses with owner-reported visual problems, veterinary examination showed that approximately half had a reduced pupillary light reflex and half had an absent or reduced menace response. Note that 1 of these studies showed that, despite the owners' concerns about the visual capacity of their horses, 50% were still used for ridden exercise, raising some important safety considerations.[2] In addition to the concerns for human safety, there are considerable animal welfare implications. Horses are naturally grazing animals that are hunted by predators and rely on vision as their primary sense. Blind horses, or those with significantly reduced vision, can show a high level of fear and anxiety; they can become unpredictable and require cautious handling by experienced caretakers.[58]

SUMMARY

Ocular abnormalities are a common finding in aged horses. Although these seldom cause overt visual deficits detected by their owners, they can be a source of chronic or acute discomfort so early detection, and treatment when available, is essential. Some of these abnormalities are specific to old horses, whereas others are a result of ongoing disorder or inflammation that started earlier in life but that becomes more evident when the damage sustained to the eye is advanced. If vision is significantly affected, consideration of human safety and animal welfare is paramount.

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