INTRODUCTION

Eosinophilic keratitis (EK) is a rare condition in horses and the cause is unknown. Although links with parasitism or use of anthelmintics have been suggested, no correlation with EK has been found. Some studies have suggested that younger horses may be predisposed (Yamagata et al 1996). Similarities have been noted to vernal keratoconjunctivitis in human beings (Yamagata et al 1996) and eosinophilic keratitis in cats (Ramsey et al 1994).

CASE REPORT

A 29-year-old donkey was presented to a colleague with a two-day history of serous ocular discharge in the right eye. A raised area was observed at the dorsal limbus. No improvement was noted on topical antibiotic therapy.

At re-examination one week later, the eye was open and appeared comfortable. An elliptical, raised, cream-coloured plaque was present at the dorsal limbus (2cm by 1cm). Adjacent conjunctiva was slightly hyperaemic. There was dorsal cornea oedema and superficial and deep vascularisation around the periphery of the raised area and lateral to it. It was not possible to transilluminate the plaque or visualise the area behind it.

Figure 1: Right eye at presentation
Under sedation, swabs were taken from the ventral conjunctival sac and the periphery of the lesion in the right eye. No corneal ulceration was evident with fluorescein staining. A blood sample was taken for a complete blood count. A fine needle aspirate was taken from the raised area.

The complete blood cell count was unremarkable. Swabs collected yielded microbes considered part of the normal equine flora. The FNA contained multiple eosinophils with occasional mast cells, macrophages and red blood cells. A diagnosis of EK was considered most likely. Medication was altered to dexamethasone/ polymixin B/ neomycin (Maxitrol®; Alcon®) (2 drops, twice daily, right eye).

At re-examination ten days later the raised, cream-coloured plaque had disappeared. The sclera and cornea in this area now showed marked pigmentation. Corneal oedema was much reduced but some vascularisation remained. Medication was continued for a further four weeks. At this stage corneal vascularisation was minimal, although there was still significant pigmentation. Medication was subsequently stopped; there has been no deterioration in the eye to date.

Figure 2: Right eye 10 days after starting treatment

**DISCUSSION**

Although EK has been infrequently described in the horse, there are no known reported cases in donkeys. The raised limbal plaque seen at presentation was associated with corneal oedema and vascularisation; these observations have been made in other cases of EK (Ramsey et al 1994). Plaques are described as sub-epithelial (Brooks 2004) and may be single or multiple. Although corneal scrapes are frequently used to obtain samples for cytology, FNA was a successful method of sampling the single, large plaque in this case.
In the light of clinical signs, bacteriology and FNA findings a presumptive diagnosis of EK was made. It is important to rule out bacterial, fungal, allergic, parasitic and neoplastic causes of keratitis as far as possible before commencing therapy. EK is usually treated with topical corticosteroids, in particular dexamethasone (Yamagata et al 1996, Brünott et al 2005). A mean duration of treatment of 64 days has been reported (Yamagata et al 1996); this case was treated for 42 days.

Superficial keratectomy to surgically remove plaques has been used successfully with cases of EK and can shorten the treatment period (Barnett et al 2004). However EK in this donkey responded to topical treatment alone and there has been no recurrence to date.

References

Mutations In HSF4 Are Associated With Dominant And Recessive Forms Of Hereditary Cataract In Different Breeds of Dog.

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Previously we have reported a single nucleotide insertion in HSF4 that is responsible for hereditary cataract (HC) in the Staffordshire Bull Terrier (SBT). HC in this breed is bilateral, symmetrical in the two eyes, and progressive with resultant blindness. It is not congenital but appears at a few weeks to months in age, progressing to total cataract by 2 to 3 years of age. We also reported preliminary evidence to suggest an identical mutation is also responsible for early onset hereditary cataract (EHC) in the Boston Terrier (BST), a condition which is clinically very similar to HC in the SBT. In both breeds HC is inherited as a simple autosomal recessive trait. To further investigate the association between HSF4 and both the early- and late-onset forms of HC observed in the BST we sequenced the entire coding region of HSF4 in BSTs affected with both EHC and late onset HC (LHC). Our results prove the HSF4 insertion is indeed the cause of EHC in the BST and also that the mutation is not associated with LHC, providing the first molecular evidence that the two forms of cataract observed in the BST are genetically different. We will also present evidence that the same mutation is responsible for HC in French Bulldogs, a cataract which is again very similar clinically.

We have also tested samples from Golden retrievers and American Cocker spaniels affected with another HC, clinically quite dissimilar from that which occurs in the SBT and BST. Neither of these breeds carried the HSF4 insertion, proving that the cataract in these two breeds is different both clinically and genetically to that seen in the SBT and BST.

In contrast to the HSF4 insertion, which causes a recessive form of HC in the above breeds, we have also identified a single nucleotide deletion in the same gene that is associated with a dominant form of HC in the Australian Shepherd (AS). We have collected DNA from a large number of ASs all of which have been examined by a veterinary ophthalmologist and around 40 of which have been diagnosed with bilateral cataracts. We have genotyped all dogs for the HSF4 deletion and present evidence that the HSF4 deletion is significantly associated with HC in the AS. The majority of affected dogs carry only a single copy of the deletion, indicating HC is inherited as a dominant, rather than a recessive, condition in this breed. We will also present preliminary evidence that dogs that are homozygous for the HSF4 deletion may have a different, more progressive form of cataract than heterozygous dogs.
Effect of mydriasis on intraocular pressure in dogs: preliminary findings.

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Introduction

The anatomy of the canine iridocorneal angle might suggest that mydriasis would elevate intraocular pressure by a blockage of the conventional drainage pathway. This can indeed be the case in man and has more recently been documented in the cat. A number of studies in the dog, where ocular hypertension after mydriasis for diagnostic fundoscopy could be a significant problem, have not however showed a similar increase in intraocular pressure to that seen in the cat or in man. These canine studies measured intraocular pressure before pupil dilation was initiated and at maximal dilation, with the researchers not finding a difference between these two values. We sought in this study to document changes in intraocular pressure during mydriasis using the Tonovet rebound tonometer, the least invasive of the contact tonometers, to measure intraocular pressure at several time-points throughout the period of active pupil dilation and beyond.

Materials and methods

Four adult greyhounds were used as subjects in the initial phase of this study. Each was given 0.5% tropicamide for unilateral pupillary dilation prior to routine fundoscopy by veterinary students. After 24 hours the process was repeated on the second eye. In each case the non-treated eye was used as a control. Intraocular pressure was measured in both eyes using the Tonovet rebound tonometer every five minutes for 65 minutes after application of the topical mydriatic. In the second phase of the study 10 clinical cases receiving tropicamide for mydriasis prior to diagnostic fundoscopy were subject to tonometry using the same technique before mydriasis and at 40 minutes after mydriatic application, this having been shown to be the time-period at which the highest mean intraocular pressure was documented in the phase I study.

Initial intraocular pressure and highest intraocular pressure in phase I were compared with a paired t-test as were initial intraocular pressures and intraocular pressures at 40 minutes after application of the mydriatic in the phase II study.

Results

The intraocular pressures study throughout the study for the treated and control eyes of dogs in the phase I study are shown graphically in figure 1. Initial and peak intraocular pressures were different with a significance of p<0.001. The mean difference between initial intraocular pressure and peak intraocular pressure was 11.4±3.5mmHg. Similarly differences between initial intraocular pressures and intraocular pressures at 40 minutes in the ten dogs in the phase II study, as shown graphically in figure 2, were significant at p<0.00001. The mean increase in intraocular pressure between these two times was 6.6±1.5mmHg.

Discussion

These results have importance in that, although the rise in intraocular pressure was temporary, they suggest that mydriasis of a potentially glaucomatous eye might initiate a pathological rise in intraocular pressure of longer duration. The mechanism by which the rise in intraocular pressure occurs appears somewhat perplexing as is the difference between this work and previous canine
The rise in intraocular pressure reaches its maximum well after maximal pupil dilation; indeed this may well be why these increases in intraocular pressure after application of mydriatics have not been reported previously in the dog. Further investigation following these preliminary results will include investigation of possible pressure-increasing effects of 1% tropicamide, phenylephrine and atropine as well as correlation of ocular hypertensive effects with pupil dilating effects and gonioscopic findings while mydriasis and intraocular pressure increase is occurring.

Figure 1: Mean changes in intraocular pressure in phase I study. (Filled circles: treated eye open circles: control eye)

Figure 2  Changes in intraocular pressure in clinical cases in phase II study between pre-treatment values and values at 40 minutes of mydriasis