The Differential Diagnosis of "Red Eye" in Dogs. So Many Diseases - So Little Time!

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Introduction

"Red eye" is one of the most common ophthalmic presentations in clinical veterinary practice. Although relatively "minor" conditions are responsible for the majority of "red eyes", this presentation may signal more severe, vision-threatening or even life-threatening disorders. A logical, step-wise approach to the diagnosis of red eye is vital to ensure that serious ocular and systemic disorders are identified and treated promptly and appropriately.

Causes of Red Eye

These may be considered from an anatomic standpoint, in a logical progression, from external / adnexal diseases, to anterior segment and intraocular disorders (Table 1).

Table 1. Common causes of red eye in dogs.

Orbital disease	Abscess/ cellulitis Proptosis Neoplasia
Anterior segment & adnexal disease	Blepharitis Conjunctivitis Third eyelid protrusion Episcleritis / scleritis Conjunctival or scleral hemorrhage
Corneal disease	Keratitis - ulcerative Keratitis - non-ulcerative
Intraocular disease	Uveitis Glaucoma Hemorrhage Neoplasia

Clinical Approach to Differential Diagnosis of Red Eye

The importance of obtaining a thorough clinical history is emphasized. A careful clinical examination to localize the source of the red appearance; identify key features of disease and to exclude or diagnose serious intraocular disorders can be achieved in general clinical practice, within a limited time frame and using only simple diagnostic equipment.

Key features to evaluate: (See Table 2)

- Onset: Acute vs chronic?

- Pain: Signs include squinting, lacrimation, rubbing
- Globe size & position: Enlarged vs exophthalmos; reduced in size vs enophthalmos?
- Pupil size: Are pupil sizes equal, large, small? (evaluate in dim **and** bright light)
- Pupillary light reflexes: Both direct and consensual
- Vision: Menace, dazzle, obstacles, tracking
- Episcleral congestion vs conjunctival hyperemia: Congested conjunctival blood vessels are less worrisome than episcleral vessels. Conjunctival vessels remain mobile within the bulbar conjunctiva, extend into the fornix and tend to be more tortuous, bright red and branching vs the relatively straight and immobile, darker episcleral vessels
- Ocular discharge: May indicate infection if purulent Discharge adherent to the ocular surface is suggestive of dry eye (KCS).
- Corneal vascularization: Deep, straight circum-limbal vessels form a "brush border", appearing at the limbus and indicate deep corneal disease, and /or intraocular disease e.g., uveitis and glaucoma. These should be differentiated from branching, superficial vessels that originate in conjunctiva and cross over the limbus.
- Corneal edema: Blue, hazy, "steamy" appearance that may be focal or diffuse?
- Aqueous flare: Use small, focal, bright light beam to detect protein in anterior chamber.
- Lens abnormalities: Distant direct ophthalmoscopy can detect cataract, lens luxation / subluxation.
- Posterior segment changes: Attempt to evaluate for hemorrhage, chorioretinitis, retinal detachment

Ancillary Diagnostics

Additional basic supplies include: Schirmer tear test strips, fluorescein stain, Topical anesthetic, swabs for bacteriology, supplies for cytology (microscope slides and a blade for obtaining scrapes, stain (e.g., Diff-Quik). Additional instrumentation that may or may not be available include Tonometer, Ultrasonography, Goniolens.

Table 2. Differentiating common causes of red eye.

Clinical feature	Uveitis	Glaucoma	Keratitis	Conjunctivitis
Pain	++	++ (↓ if chronic)	+/-	Discomfort
Pupil size & PLR	Miotic /↓ PLR	Dilated (absent PLR)	Normal or miotic (if reflex uveitis)	Normal
Vision - affected eye	+/-	-	+/-	+
Episcleral congestion	+	+	-/+ (if deep keratitis/ulcer)	-
Conjunctival hyperemia	+	+	+	+
Ocular Discharge	Variable	Variable	Variable	Variable / "sticky" if KCS

Corneal vascularization	Deep	Deep	Superficial / deep (depends on disease)	-
Corneal edema	+	+	+/-	-
Aqueous flare	++	+/-	-/+	-
Lens	Synechiae, cataract or sublux ?	Luxation or subluxation? Cataract?	- (unless penetrating wound)	-
Posterior segment	Chorioretinitis/ retinal detachment/ hemorrhage	Optic disc cupping	Retinal detachment if trauma	-
IOP	↓ (or ↑ if 2° glaucoma)	↑ ↑ (may be normal if chronic / 2° to uveitis)	-/↓ (if reflex uveitis)	
Gonioscopy	+/- normal (if visible)	Opposite eye - pectinate ligament dysplasia?	Normal (if visible)	Normal

Speaker Information

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SA281

FELINE CONJUNCTIVITIS: IT'S ALL ABOUT HERPES & STRESS!

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INTRODUCTION

Feline herpesvirus (FeHV-1) is widespread in the domestic cat population, especially in colonies and catteries. More than 95% of the cat population has been exposed to the virus, and more than 80% are carriers.[1,2] Cats are infected after direct or indirect contact with sick or carrier animals; the infection occurs through the oronasal and conjunctival routes. Clinical signs of active disease include conjunctivitis and keratitis with ulceration (early, superficial dendritic ulcers may stain with rose bengal but not with fluorescein) and possible upper respiratory signs. Stromal vascularization and cellular infiltration characterize the immune response to viral particles in the cornea, either with or without active viral replication. Secondary bacterial infections, especially with *Chlamydia felis* and possibly with *Mycoplasma* spp., are frequent; and symblepharon is a common sequel. FeHV-1 may also play a role in the pathogenesis of corneal sequestration and eosinophilic keratitis.[1-3]

Cats that recover from the disease probably remain persistent carriers due to latent infection in the trigeminal ganglia. The latent disease is characterized by the absence of clinical signs, but viral shedding and reactivation can occur.[1,2] Stressful events, such as an unrelated illness, the introduction of a new pet or baby into the house, travel, or fighting, can serve as triggers of shedding and reactivation of latent infection. Treatment with corticosteroids can have similar consequences. The presence of a latent disease, as well as the confounding impact of an overly aggressive immune response, presents great therapeutic challenges to the practitioner. Drug availability, irritancy, and dosing frequency further complicate treatment of the disease.

PRINCIPLES OF TREATMENT

Antiviral medications are indicated in cats with active disease. The drugs are pyrimidine and purine nucleoside analogues, which interfere with the viral replication cycle by inhibiting target enzymes that incorporate nucleosides into nucleic acids. Compiling results of multiple studies[4-8] in vitro efficacy against FHV-1 is:

trifluridine > ganciclovir > idoxuridine > cidofovir > penciclovir > vidarabine > valacyclovir > acyclovir

Importantly, antivirals are virostatic and are only effective in treating actively replicating virus, achieving their effect by interfering with viral DNA replication. The drugs are not virucidal and therefore are unable to eradicate latent infection. Significant toxicity can occur with antiviral administration, due to the intracellular location of the virus and the inability of available medications to target viral, rather than host cell, replication selectively.

Virostatic drugs usually have to be administered frequently, and treatment is often continued for 10-14 days after remission of clinical signs. Furthermore, as the drugs are not virucidal,

owners should be warned of the possibility of shedding, reactivation, and recrudescent infection.[9]

There are two iatrogenic factors that may induce recrudescent FeHV-1 infection. One is corticosteroid treatment. Corticosteroids are contraindicated in all cases of primary ocular FeHV-1 infection because they will exacerbate active viral infection; thus, a self-limiting conjunctivitis may become a chronic corneal infection. In fact, any corticosteroid treatment in cats must be carefully considered, as most cats should be suspected of being potential FeHV-1 carriers. When such treatment is unavoidable (eg, in cases of eosinophilic keratitis), it should probably be combined with concurrent antiviral therapy. In this context it is important to note that because the FeHV-1 may be reactivated due to immunosuppression, the prognosis for the disease is poor in immunosuppressed patients (ie, FeLV- or FIV-infected) because the recurrence rate can become high.[1-3]

Another factor that may induce viral shedding and reactivation is stress, and events such as the introduction of a new animal to the household or traveling to cat shows may exacerbate the signs. For this reason, frequent treatment with multiple drugs may sometimes aggravate the clinical signs of the disease. If worsening of signs is noted, the clinician is advised to carefully consider reducing (or even ceasing) treatment rather than increasing it.

TOPICAL ANTIVIRAL MEDICATIONS

Trifluridine 1% (Viroptic), idoxuridine 0.1% (Stoxil) and vidarabine 3% (Vira-A) are variably effective against FeHV-1, with trifluridine having the highest efficacy as well as transcorneal penetration.[4-6] However, trifluridine also tends to be more irritating to cats and, in fact, sometimes induces hyperemia of the eyelids and conjunctiva that may mimic worsening of the disease. In such cases, other drugs should be considered. Furthermore, the drug requires frequent administration (5-6 times/day), which can increase stress and decrease compliance.[6,10] Idoxuridine and vidarabine are less irritating and are administered less frequently but are difficult to obtain because they are not widely available commercially, although they can be ordered from compounding pharmacies.[9]

Newer recommendations for topical antiviral administration in cats include cidofovir 0.5%.[7,11,12] Cidofovir, which is not commercially available as an ophthalmic preparation, has strong in vitro and in vivo efficacy against FeHV-1 infection, with treatment reducing severity of clinical signs and viral shedding. Importantly, cidofovir's beneficial effect has been demonstrated with twice-daily administration, a significant advantage compared to other topical antiviral medications. Cidofovir is less toxic than other antivirals due to its relatively high specificity for viral, rather than host, replication proteins. However, long-term safety studies have yet to be published; indeed, cidofovir has been associated with a dose-dependent nephrotoxicity in humans, and cat owners should be warned to protect themselves from exposure to cidofovir solutions. Ganciclovir (Zirgan) is commercially available as a 0.15% ophthalmic gel and has in vitro efficacy against FeHV-1 but has not been studied in clinical feline patients and may be toxic.[5,8] Even if found effective and nontoxic, it is not likely to be of any value in treating FeHV-1 in cats due to its high cost.

Antiviral therapy with acyclovir or valacyclovir, a prodrug of acyclovir, is the drug of choice for treating ophthalmic herpes disease in humans, but neither drug appears to suppress FeHV-1

replication. In fact, both drugs are contraindicated in cats as acyclovir is myelosuppressive in that species, and valacyclovir is extremely toxic and often fatal to cats.[13-15]

ORAL ANTIVIRAL MEDICATIONS

Famciclovir, a prodrug of penciclovir, is a very safe and effective oral drug for the treatment of FeHV-1.[16-22] Due to complex pharmacokinetics of famciclovir and penciclovir in cats and the variable target penciclovir concentrations needed to produce a clinical effect, the accurate oral dose is currently not definitively known. In clinical studies, oral administration of 90 mg/kg famciclovir three times daily for 3 weeks to cats with experimentally induced FeHV-1 disease improved outcomes for systemic, ophthalmic, clinicopathologic, virologic, and histologic variables and reduced viral shedding. In naturally infected cats, 62.5 mg orally once daily for 7 days then twice daily was reported to result in clinical improvement. However, a single dose of 125 mg or 500 mg famciclovir, administered at the time of admission to a shelter, did not lessen clinical signs or viral shedding in cats, even in the presence of appropriate plasma penciclovir levels.[16-22]

ADDITIONAL THERAPIES

It has been suggested that recurrent infection may be minimized by oral lysine supplementation.[23-25] It is hypothesized that lysine can be helpful in the treatment of FHV-1 infection because viral growth appears to be inhibited by high intracellular concentrations of lysine and low levels of arginine. Thus, long-term treatment (250-500 mg PO twice daily) may decrease shedding, viral replication, and severity of conjunctivitis and may be used for long-term maintenance therapy. However, results of clinical investigations are equivocal, perhaps due to the high levels of dietary arginine in cats. The efficacy of lysine in humans to prevent herpes simplex is associated with severely arginine-restricted diets. This is not possible with obligate carnivore patients, and the higher arginine content of feline diets may continue to hinder the success of lysine prophylaxis of FeHV-1.[23-26]

Interferons are components of the innate immune system that induce expression of antiviral proteins within host cells, thus creating an antiviral environment, and also exert immunomodulatory effects that further limit the impact of viral infections.[26-30] Recombinant human IFN-α and feline IFN-ω have been evaluated relative to anti-FVH-1 effect, with the feline version demonstrating greater in vitro efficacy. The drug can be given as low oral doses (25 units/d) or eye drops (10^3 to 10^6 units/mL) applied two to three times daily and has decreased the severity of clinical signs in experimentally infected cats. However, results of clinical field studies have been disappointing, possibly due to the timing of administration relative to infection and shedding, as well as uncertainty surrounding appropriate dosing protocols and handling of IFN preparations.[27-31]

Supportive therapy may include tear replacements, topical antibiotics, and debridement of necrotic corneal epithelium with a cotton-tipped swab to remove viral particles. Topical tetracycline is frequently added because coinfections with *Mycoplasma* spp. or *Chlamydia felis* are common. Eyes that are irritated by tetracycline may be treated with ciprofloxacin. In cases of respiratory diseases, oral azithromycin or doxycycline should be considered.[2,9]

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Horner's Syndrome:

Synonyms:
Oculosympathetic paralysis

Disease:
Signs:
1. Miosis - Small Pupil (Constriction)
2. Drooping Upper Eyelid (Ptosis)
3. Enophthalmos - Globe retracted caudally
4. Prolapsed Third Eyelid
≥ 3 signs to be Horner's

Preganglionic
Postganglionic: Most Common: Prognosis - Excellent - Many are Idiopathic - Not involve parts of CNS or Thorax

Clinical Signs:
Injury, Stretching or Inflammation Along Pathway
1. Lower Cervical and Upper Thoracic Nerveroots: Lameness concurrent or weakness of Front limb - Same side
2. Cervical Vagosympathetic Trunk: Neck masses, wounds or injury
3. Middle Ear: Otitis Media or Masses

Diagnosis:
Signs - above
Examination of Above Clinical Signs

Radiographs: Thorax:
Anterior Thorax:
R/o Fracture 1st Rib, Mediastinal Masses, other mass

Pharmacologic Testing:
1. Hydroxyamphetamine 1% OU
a. Dilation of affected pupil: within 1 hour: Preganglionic Lesions
Postganglionic Neurons not Impaired → can release stores of Norepinephrine → Dilate
b. Pupil not Dilate → Postganglionic (with injury - Norepinephrine stores depleted)
2. Phenylephrine 1% or Cyanephrine 0.01% OU
a. Hydroxyamphetamine used? Wait 1-2 Days
b. Preganglionic Lesion: No dilatation
c. Postganglionic Lesion: Dilation. Denervation Hypersensitivity (2 weeks to develop - wait to test)
d. Normal Eye should not change. Medication concentration too low.

Testing does not correlate to Prognosis for Recovery

Signalment:
Breed: Collie, Golden Retriever
Sex: None
Age: Mature, Middle-Aged (Mean age 8.6 years)

50% dogs Idiopathic
Usually Unilateral

Dx:
Radiography:
Thorax → Pulmonary Mass
Head/Skull → Osseous Bulla
Spine → Vertebral Bony Lesion
Otoscopy:
Fluid behind Tympanum, Ruptured Tympanic Membrane

Horner's Syndrome

Last updated on 3/21/2015.

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Synonyms:

Oculosympathetic paralysis

Disease description:

Horner's syndrome is a collection of signs, not a specific disease, that occur following sympathetic denervation to the eye. Signs consist of miosis, drooping of the upper eyelid (ptosis), enophthalmos, and prolapse of the third eyelid. At least three of these signs must be present to confirm Horner's syndrome. The presence of <3 signs could be compatible with a partial Horner's syndrome or some other disease. Horner's syndrome is most often a unilateral condition but signs can rarely be bilateral.[1-6]

Pathophysiology

Sympathetic pathways that innervate the dilator muscle of the pupil and the smooth muscles of the orbit and lids originate in the hypothalamus. The nerve fibers continue through the brain stem and cervical cord to the first and second spinal thoracic cord segments, where a synapse with cell bodies takes place. Fibers then continue in the cervical vagosympathetic trunk to synapse in the cranial cervical ganglia. The cranial cervical ganglion lies deep to the tympanic bulla and proximal positions of Cranial Nerves IX, X, XI, XII. Postganglionic fibers proceed through middle ear and bony orbit to innervate the iris dilator muscle and smooth muscles of orbit and lids.

Pre- and postganglionic lesions: If damage to the sympathetic pathway occurs anywhere between the brainstem and the sympathetic ganglion (located just behind the eye), the lesion is preganglionic in location, which could indicate a more serious etiology. If damage occurs in the sympathetic ganglion or beyond it, the lesion is postganglionic. Prognosis can be more favorable with postganglionic lesions and many of these are idiopathic in origin. Pharmacologic testing can be done to distinguish pre- from postganglionic lesions.

Clinical Signs

Injury, stretching or inflammation anywhere along the described pathway can result in Horner's syndrome. The most common sites of injury or disease that cause Horner's syndrome are:[1-6]
1) The lower cervical and upper thoracic nerve roots: look for concurrent lameness or weakness of the front limb on same side 2) The cervical vagosympathetic trunk: look for neck masses, wounds or injuries 3) The middle ear: look for otitis media or masses

Diagnosis

Physical Examination Findings: Diagnosis is made by finding the classical four signs of Horner's syndrome on physical examination. Thorough physical and neurological exams are then performed to rule out systemic abnormalities, as well as cranial nerve and other neurological deficits. All structures in the neck are palpated to search for lesions near where the vagosympathetic trunk lies. A complete eye exam is performed to rule out other causes of miosis, enophthalmos, and prolapse of the third eyelid. Otoscopy is done to look for evidence of middle ear disease.

Thoracic Radiography: Thoracic radiographs help rule out abnormalities in the anterior thorax, including fractures of the first rib, mediastinal masses, other masses, etc.

Pharmacologic Testing: Pharmacologic testing can be done to help localize the lesion to preganglionic or postganglionic sympathetic nerves.[17-18] When performing pharmacologic testing, the normal eye can be used as a control.
1) Hydroxyamphetamine 1% is applied (1-2 drops) in both eyes once.
a) Dilation of the affected pupil within 1 hour indicates a preganglionic lesion. In such cases, the nerve

endings of the postganglionic neuron are not impaired, so can release their stores of endogenous norephinephine, which causes the pupil to dilate.
b) If the affected pupil does not dilate, the lesion is more likely to be postganglionic. With injury to postganglionic nerve endings norepinephrine stores are depleted.

2) Phenylephrine 1% or epinephrine 0.01% is applied (1-2 drops) in both eyes once.
a) If hydroxyamphetamine was used previously, wait 1-2 days before doing this test.
b) No dilation in affected pupil indicates a preganglionic lesion.
c) Dilation in the affected pupil indicates a postganglionic lesion from denervation hypersensitivity. This hypersensitivity takes approximately 2 weeks to develop, so pharmacologic testing is not accurate if performed immediately after the onset of signs.
d) The normal eye should not change because the concentration of medication is too low to effect a change.

Results of pharmacologic testing do not correlate well with the prognosis for recovery. However, postganglionic lesions tend to have a more favorable prognosis than preganglionic ones, probably because many postganglionic cases are idiopathic in origin and do not involve other parts of the central nervous system or the thorax.

Disease description in this species:

Signalment

Most affected dogs are middle-aged (mean age 8.6 years), but Horner's syndrome can occur in dogs of all ages (range 1-17 years).[1] Unlike the cat where the origin of the Horner's syndrome can frequently be determined, up to 50% of cases in dogs may be idiopathic.[1] Many different breeds of dogs may develop idiopathic Horner's syndrome, but male golden retrievers and collies appear to be predisposed.[7-8] Horner's syndrome is usually unilateral but rare cases of bilateral Horner's syndrome have been documented.[9-11]

Etiology[1-5,9-16]

1) Trauma to the neck and anterior thorax: bite wounds, automobile accidents, jugular venipuncture and catheterization, cervical surgery, alcohol ablation of thyroid nodules
2) Other lesions in neck: thyroid and parathyroid neoplasms
3) Anterior mediastinal disease: thymoma, anterior mediastinal lymphoma, other masses, hematoma
4) Middle ear disease and surgery: otitis media, inflammatory polyps, tumors, bulla osteotomy, iatrogenic injury associated with ear cleaning with irritating substances when the ear drum is ruptured[1]
5) Diseases of the cervical spine and nerve roots: brachial plexus injuries or neoplasia, cervical vertebral trauma or neoplasia
6) Space-occupying lesions of the hypothalamus or cavernous sinus
7) Brainstem diseases: vascular, infectious, inflammatory or neoplastic (may be bilateral)
8) Diabetes mellitus: bilateral, rare
9) Idiopathic: Idiopathic cases have no identifiable cause and most are postganglionic in origin.[7-8,23] They account for up to 50% of canine cases.

Etiology:

Automobile injury
Bite wound
Brachial plexus injury
Ceruminal gland adenocarcinoma
Fibrocartilaginous embolism
Iatrogenic
Idiopathic, unknown
Intervertebral disc disease
Mediastinal neoplasia
Neoplasia
Otitis media
Parathyroid adenocarcinoma
Thyroid carcinoma
Trauma

Breed predilection:

Collie

Golden retriever

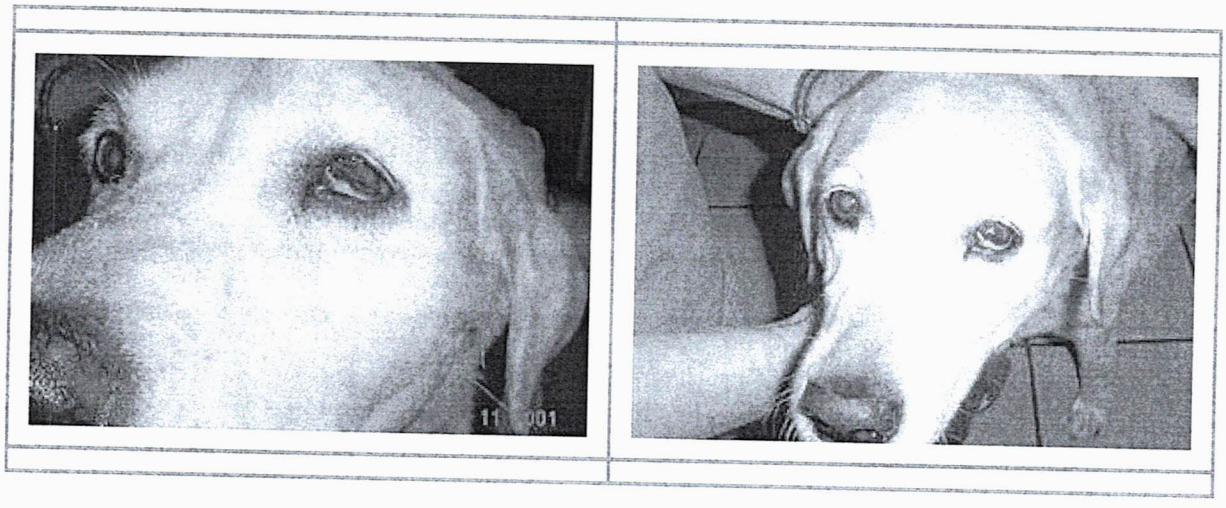
Sex predilection:
None

Age predilection:
Mature, middle-aged

Diagnostic procedures:	Diagnostic results:
Radiography of thorax	Pulmonary mass
Radiography of head/skull	Osseous (tympanic) bulla opacified
Radiography of spine	Vertebral bony lesion
Otoscopy	Fluid behind tympanum Tympanic membrane ruptured

Images:

Click on the image to see a larger view.



Treatment/Management/Prevention:

SPECIFIC THERAPY

1) The underlying cause should be identified and corrected if possible. Example: If otitis media is present, treat it.
2) No specific treatment exists for Horner's syndrome itself. Signs resolve if the nerve function recovers once the underlying disorder is treated.
3) A single case report exists of acupuncture treatment resulting in rapid resolution of signs.[19] Whether or not the improvement was co-incidental is unknown.

PROGNOSIS

1) Recovery is less likely with avulsion or irreparable damage of the sympathetic nerves (e.g. brachial plexus syndrome), central nervous system disease, anterior mediastinal neoplasia, and certain iatrogenic injuries (e.g. surgical trauma, arterial catheterization, etc.)
2) Postganglionic lesions have a more favorable prognosis than preganglionic ones.
3) Usually the prognosis is good for idiopathic cases as spontaneous recovery can begin in 7-8 weeks, with resolution in an average of 16 weeks (it can take as long as 6 months). If necessary, signs can be masked by topical treatments of phenylephrine eye drops 2-3 times daily which basically improves the appearance of the eye and eliminates the elevation of the third eyelid which can affect vision in severe cases. Caution must be taken with use of phenylephrine, however, because it is toxic to corneal epithelium.
4) Recovery may or may not occur. It can be partial or complete.[1-4]

Preventive Measures:

Use of ear cleaning solutions containing tannic acid should be avoided if the ear drum is not intact. Care must

be taken with jugular venipuncture and insertion of IV catheters. Careful dissection during bulla osteotomy and cervical surgery is also warranted.

Special considerations:

Other Resources:
Recent VIN Message Board discussions on Horner's syndrome
Client Handout on Horner's syndrome

Differential Diagnosis:

Miosis
1) Ulcerative keratitis
2) Uveitis
3) Drug-induced
4) Other neurologic diseases

Enophthalmos
1) Ocular pain
2) Loss of retrobulbar fat pad
3) Extraocular or masticatory muscle fibrosis

Third eyelid prolapse
1) Ocular pain
2) Enophthalmos or exophthalmos from other causes
3) Tetanus
4) Dysautonomia (usually bilateral)
5) Third eyelid or orbital neoplasia

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Feedback:

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If you have any questions about a specific case or about this disease, please post your inquiry to the appropriate message boards on VIN.

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Companion Notes

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Case report of Horner's syndrome due to a post-ganglionic lesion in a dog

Overview on Horner's syndrome

- consider in all animals with anisocoria where both pupils constrict with light
  - and anisocoria worsens in scotopic conditions
- non-specific accompanying signs
  (also seen when uveitis and keratitis are present)
  - ptosis (DROOPING OR FALLING EYELID)
  - enophthalmos (POSTERIOR DISPLACEMENT OF EYEBALL WITHIN ORBIT)
  - third eyelid prolapse
- in Horner's syndrome
  - norepinephrine is not released from pre-synapse
    - doesn't accumulate in the NM junction
      - doesn't stimulate mydriasis of iris dilator muscle
- in human medicine:
  - gold standard test to confirm the syndrome is response to topical cocaine
    - lack of pupillary dilatation in affected pupil
    - dilatation of the normal pupil
  - cocaine blocks norepinephrine reuptake at neuromuscular (NM) junctions (of the iris dilator muscle)
    - topical ophthalmic cocaine not available when this case dog presented
    - aproclonidine (weak α_1 agonist) can stimulate the iris dilator muscle
      - in Horner-affected eye that is adrenergic hypersensitive
        - can confirm some cases of Horner's syndrome
      - also not available at time of examination
- in author's experience, post-ganglionic lesions are the most common cause (PROGNOSIS: EXCELLENT)
  prognosis is excellent

- prognosis is excellent

Case report of a 9-year-old, neutered, sex:M dog

- historical signs, physical examination and testing from referring clinician (rDVM)
    - tentative diagnosis: anterior uveitis
- at referral for left enophthalmos, miosis, ptosis, 3rd eyelid prolapse and anisocoria
    - ophthalmic examination
        - present menace responses, bilaterally
        - present direct and consensual pupillary light reflexes, bilaterally
        - present palpebral and oculocephalic reflexes, bilaterally
        - anisocoria, left pupil smaller than the right
            - worse under scotopic conditions
    - Schirmer tear test: 27 mm/min in right and 25 mm/min in left
    - rebound tonometry: 13 mmHg in right and 8 in left
    - topical ophthalmic tropicamide (parasympatholytic agent), bilaterally
        - right pupil dilated completely within 20 minutes
        - left pupil dilated minimally, 1-2 mm
            - still mid-sized after 30 minutes
            - this subtle dilation with tropicamide is typical of Horner's syndrome
                - in article author's experience
                - blocks parasympathetic portion of oculomotor nerve
                    - in Horner's syndrome the pupil dilates slightly
                        - as "parasympathetic tone is removed"
                            (pupillary escape)
    - biomicroscopy and indirect ophthalmoscopy
        - incipient nuclear cataracts
        - anisocoria
    - negative fluorescein staining
    - tentative clinical diagnoses
        - left Horner's syndrome
        - bilateral incipient nuclear cataracts, incidental finding in many breeds
            - these do not progress
- re-presentation 2 days later for an adrenergic response test
    - topical 0.1% phenylephrine prepared
        - 1 drop placed on right and left pupils and time noted
            - within 20 minutes
                - left eye signs resolved
                    - enophthalmos, third eyelid prolapse and miosis
                    - left pupil hyper-responded and dilated
                        - indicating 1 of the following:
                            - post-ganglionic lesion
                            - third order Horner's lesion
                        - hyper-response develops ~ 1 month
                            (after lesion develops)
                            - due to denervation hypersensitivity
                                - in post-synaptic NM junction

- a compensatory change

- right pupil did not react

- remained responsive to light and mobile

"The lack of biomicroscopic and indirect ophthalmoscopic evidence of uveitis, or keratitis in this dog, further supported the tentative clinical diagnosis of Horner's syndrome."