

Unwrapping the Red Eye

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Red eye is one of the most common presenting complaints in veterinary medicine. Although it can feel overwhelming, a systematic approach can ensure that you never apply inappropriate treatment, even if you aren't 100% confident of your diagnosis. By ruling out diseases with very targeted therapy, you can identify diseases that require anti-inflammatory therapy.

OPHTHALMIC EXAM

The description "red eye" is pretty vague and encompasses diseases that result in conjunctival hyperemia and those that result in episcleral injection. In order to identify and localize redness, a few tips on exam technique are worthwhile. The key to proper ophthalmic examination is a good light source and a methodical approach. You don't need a slit lamp biomicroscope or a head-mounted indirect ophthalmoscope. 90% of what you need to see can be seen with a high intensity penlight (like the Welch Allyn halogen penlights) or a bright transilluminator.

Experienced clinicians will use the known breed and age predisposition to ocular disease to narrow the list of possible diagnoses even prior to examining the animal. Most ophthalmic diagnoses are at least initially anatomic (retinal detachment, corneal ulcer, etc.) rather than etiologic (e.g., systemic hypertension induced retinal detachment) and are based on direct inspection. I usually begin from the outside and work my way deeper into the eye and then consider what, if any, additional diagnostic tests are required. Be systematic and do not focus exclusively on the obvious.

Begin with a Finoff transilluminator (or a direct ophthalmoscope set at 0 diopters) at arm's distance and establish both tapetal reflexes. This allows you to detect unequally sized pupils (anisocoria) and whether any opacities are present in the ocular media between you and the tapetal reflection. Move the light source close to the eyes and check for a direct and consensual pupillary light reflex. Also use the light to directly and obliquely illuminate the eye. Retroillumination, in which a more anterior lesion is backlit by bouncing light off the tapetum or iris, also can be helpful. Be sure to inspect the eyelids, eyelid margin, the anterior surface of the third eye, the conjunctiva, the pre-corneal tear film (seen as a bright reflection from the ocular surface), the cornea, depth and clarity of the anterior chamber, the color size and shape of the iris/pupil, and the lens. The animal naturally wants to keep its eye on a horizontal plane; therefore, lesions involving the inferior cornea can be seen by pointing the animal's nose to the ground.

Surface vessels on the conjunctiva are branching and move with the conjunctiva. Engorgement of these vessels (conjunctival hyperemia) is indicative of surface disease. This is different than the deep vessels of the episcleral that exit the limbus at 90 degrees. These vessels do not move, nor do they blanch with phenylephrine. Episcleral injection is indicative of intraocular disease. Although these rules are helpful for localizing redness, keep in mind that clinical presentations can be murky.

CONJUNCTIVITIS AND KERATOCONJUNCTIVITIS SICCA (KCS)

The clinical signs of conjunctivitis include hyperemia (redness), chemosis (edema) and various degrees of discharge. Depending on the nature and severity of the conjunctivitis, discharge can range from serous to mucoid to mucopurulent. Pure conjunctivitis may affect the palpebral conjunctiva, the bulbar AND palpebral conjunctiva but never just the bulbar conjunctiva. This can help you distinguish conjunctivitis from episcleral injection associated with intraocular inflammations such as uveitis or glaucoma. Additionally conjunctival vessels are moveable, bright, red and tortuous as opposed to straighter episcleral vessels.

Keratoconjunctivitis sicca or dry eye is frequently overlooked in initial evaluation of the red eye as it requires a Schirmer tear test to be performed prior to any other ocular testing. KCS has a typical clinical appearance with hyperemia, chemosis and thick, ropey, mucoid discharge. This discharge is frequently difficult to wash off the surface of the eye. Additionally, the dryness causes chronic irritation to the surface of the eye resulting in varying degrees of corneal vascularization and pigmentation (unlike the other stated causes of conjunctivitis). The most common causes of KCS are bilateral while neurogenic KCS is almost exclusively unilateral and associated with a dry crusted nose.

A STT under 15mm/min concurrent with redness and discharge is sufficient to make a diagnosis of dry eye. Treatment remains lifelong at this point and consists of topical cyclosporine or tacrolimus. Optixcare is a great large volume gel that clients like and dogs find soothing. Gels and ointments have much longer contact times than aqueous drops and tend to work better for supplementing tears. Only in cases of neurogenic KCS, is the drug Pilocarpine indicated.

A subtle form of dry eye is qualitative tear film deficiency. The Schirmer tear test only assesses the aqueous portion of the tears (produced by the lacrimal and third eyelid glands). However, the mucus and oil components are equally vital to spreading the tear film, adhering it to the surface of the cornea and preventing it from evaporating too quickly. With deficiencies in either mucus (made by goblet cells of the conjunctiva) or meibum (made by the meibomian glands at the eyelid margin), the tears roll off the surface of the eye too quickly or evaporate leaving dry spots. In fact, the STT may be high as the body is making extra aqueous tears in response to the sensation of dryness. Thankfully, cyclosporine and tacrolimus improve this disease as well. These dogs will have hyperemia, serous discharge and a dull appearance to the surface of the eye with potentially small blood vessels growing onto the surface. The official test for this disease is the tear film break up time, but it is highly subjective, and I typically make a clinical diagnosis with an appropriate collection of clinical signs.

Infectious conjunctivitis is analogous to human pink eye which is a highly contagious bacterial infection of the conjunctiva. This is exceedingly rare in canine ophthalmology. For this reason, conjunctival culture and sensitivity and empirical therapy with a pure antibiotic in canine conjunctivitis is NOT INDICATED for the vast majority of cases. Staphylococcus and streptococcus species are common normal flora for the conjunctiva so any culture will be positive for some type of bacteria. Every type of canine conjunctivitis is best served with treatment with an anti-inflammatory with either cyclosporine, NSAID or steroid. If one is careful to screen for KCS, then conjunctivitis becomes an easily mastered disease process.

ULCERATIVE KERATITIS: CORNEAL ULCERS AND EROSIONS

General goals of treating corneal disease include quickly arriving at an accurate diagnosis as to the depth and severity of the lesion, determining the underlying cause of the lesions, ensuring mechanical integrity of the cornea, collecting the appropriate samples prior to therapy, preventing sepsis, and controlling intraocular pain and inflammation.

Corneal erosions and ulcerations are quite common and differentiating between the two is crucial for developing a treatment plan. Corneal erosions imply loss of the corneal epithelium only, with the stroma remaining intact. Corneal ulcers imply loss of corneal epithelium, basement membrane and variable amounts of corneal stroma including down to Descemet's membrane. Perforation is possible, particularly with deep and/or infected ulcers.

It is important to always try to determine the depth of the ulcer and identify an etiology. Oblique illumination is best to determine the depth. Epithelial defects tend to look like the skin has been peeled off a grape, and the epithelium may be loosely adherent. In ulcers there is a depression or frank crater, or a "bubble/blister" (often a descemetocoele). If perforation has occurred the defect may be filled with blood, fibrin, or uveal tissue. The most common causes of erosions/ulcers seen on an emergency basis are secondary to foreign bodies and trauma. Other causes of corneal problems are adnexal disease or poor conformation leading to exposure or mechanical injury to the cornea, both quantitative and qualitative tear film abnormalities, abnormalities in globe position leading to lagophthalmos and facial nerve paralysis causing lagophthalmia. Appropriate management of corneal disease includes a thorough ophthalmic exam to ensure that lid function is normal, tear function is normal, and that no other ocular abnormalities are present which could contribute to the formation or persistence of a corneal erosion.

NON-ULCERATIVE KERATITIS: CORNEAL PANNUS AND EOSINOPHILIC KERATITIS

There are a number of diseases of the corneal surface that do not involve corneal ulceration. Anything that causes chronic irritation to the surface of the eye results in keratitis (exposure, entropion, KCS). As described in the conjunctivitis section, KCS or dry eye presents with corneal disease as well as conjunctivitis. This should be identified with an appropriately administered Schirmer Tear Test. The other cause of keratitis is immune-mediated keratitis. In dogs this takes the form of chronic superficial keratitis aka pannus and in cats this takes the form of eosinophilic keratitis.

These diseases are identified by the presence of corneal vessels in the absence of corneal ulceration or a mechanical cause for chronic irritation (lid abnormalities). In dogs, pannus typically presents bilaterally with temporolateral areas of corneal vascularization and pigment. German shepherds are overrepresented in this disease process. Immunosuppression with topical steroids or cyclosporine is considered the gold standard care. In cats, immune-mediated keratitis frequently takes the shape of eosinophilic keratitis. This form of keratitis has corneal vessels combined with foci of white cells (often raised off the corneal surface). A corneal cytology confirming the presence of at least one eosinophil is all that is necessary to confirm the diagnosis of eosinophilic keratitis. The foundation of treatment for this disease is also immunosuppression (keeping in mind that local immunosuppression may activate latent herpesvirus infection).

UVEITIS

Uveitis is inflammation within the eye characterized by a break-down of the blood-eye barrier. Normal and aqueous humor should be completely and exquisitely transparent. A breakdown of the blood-eye barrier allows protein, white blood cells, blood or fibrin to be in the aqueous humor. The eye will look hazy, and "flare" will be present in the anterior chamber. Best examined in a dark room with the smallest focal spot on the direct ophthalmoscope head. Flare is the pathognomonic sign of uveitis but unfortunately the hardest to identify clinically. Other clinical signs of uveitis are hypopyon (pus in the anterior chamber), hyphema (blood in the anterior chamber, ciliary flush (360-degree short straight blood vessel growth on the peripheral cornea), miosis (small pupil), pain and an intraocular pressure under 10mmHg or a 10mmHg difference between eyes. As aqueous flare is so difficult to confidently identify, it is recommended that you look for two or more clinical signs (a red eye and a small pupil, a low IOP and a small pupil, a red eye and a low IOP) in order to make a working diagnosis of uveitis. Do not make the mistake of diagnosing uveitis with just one clinical sign such as a low IOP or episcleral injection.

Uveitis is most often caused by a systemic disease (even if only one eye is affected at the time of presentation) but there are a couple of primary ocular causes of uveitis. Rule out the primary ocular causes of uveitis: corneal ulcer; rapid onset cataract (will be almost mature), intraocular neoplasia (look for a mass), witnessed direct trauma. In the absence of a primary ocular cause for uveitis, the causes are systemic and fall into the three main categories of infectious, neoplastic and immune-mediated. Infectious causes include fungal, parasitic, protozoal, algal, viral, rickettsial and bacterial. Keep in mind that bacterial causes would imply sepsis so prophylactic therapy with a broad-spectrum antibiotic is NOT appropriate for empirical treatment of uveitis. The most common neoplastic cause of uveitis is lymphosarcoma, but almost any neoplasia can present with uveitis as a paraneoplastic lesion. Sixty percent of all uveitis cases will not have an identifiable cause and fall under the category of idiopathic or immune-mediated.

Regardless of the cause, appropriate treatment should be started as soon as possible. This includes topical steroids (as long as the cornea if fluorescein negative) and topical NSAIDS. Reserve systemic immunosuppression until a systemic workup has been performed. Patients should be monitored for

GLAUCOMA

Normal intraocular pressure in the dog is 15-25mmHg using both the tonopen and TonoVet. A diagnosis of glaucoma in veterinary terms must include an elevated intraocular pressure. Keep in mind that glaucoma almost always presents unilaterally and be suspicious of patients that have mildly elevated pressures in both eyes, especially if it isn't a typical breed (a breed list can always be quickly found via google).

When aqueous humor cannot flow properly through the eye, it backs up and increases the pressure within the eye. Fluid flow can be obstructed at the pupil (if there is no way for aqueous to get through the pupil) or at the iridocorneal angle (most common). Outflow through the iridocorneal angle can be obstructed by iris adhesions, congenitally abnormal drain structure, cells in the meshwork (blood, white blood cells, neoplastic cells) or rarely by problems that cannot be identified (open-angle glaucoma). The important thing to remember is that glaucoma is caused by a decrease in DRAINAGE and thus far the majority of our treatments address PRODUCTION. For this reason, our treatments are almost always built to fail. Identifying and quickly instituting pressure lowering medication is critical to managing this disease.

ORBITAL DISEASE: EXOPHTHALMOS

Orbital disease is an atypical cause of red eye wherein a space occupying mass in the orbit pushes the eye forward (exophthalmos) and the eye appears red due to increase scleral exposure and conjunctival hyperemia. An appearance of asymmetry should be clear when observing the patient while looking down at the top of the head. The abnormal eye should be pushed further out. The diagnosis is confirmed with manual retropulsion of the globe and in cases of cellulitis and neoplasia, the exophthalmic eye will be resistant to retropulsion. Be cognizant that exophthalmos can result in corneal exposure and ulceration or an incomplete blink. Treatment is focused on identifying the underlying cause of the orbital mass effect through image or blind exploration.

(EPI)SCLERITIS

Perhaps the strangest and most misdiagnosed cause of scleral injection is immune-mediated episcleritis or nodular granulomatous episcleritis (NGE). This disease can present as relatively diffuse scleral injection with or without a discrete mass on the sclera (underneath the conjunctiva). There is often a discrete line of limbal edema near the reddest area. This disease is frequently asymmetric and usually not painful despite severe redness. Biopsy is the gold standard of diagnosis but is not recommended for general practitioners. Instead, one must use a diagnosis of exclusion by running through the other causes of red eye and landing on episcleritis. This disease is treated with immunosuppression (topical steroids +/- cyclosporine or tacrolimus). This disease is controlled rather than cured and will frequently require lifetime medication.

	Keratitis	Conjunctivitis	Uveitis	Scleritis	Orbital	Glaucoma
Diagnostic	STT, fluorescein	STT, exam	Slitlamp Flare	Exclusion	Retro-pulsion	Tonometry
Pupil size	Normal to miotic	Normal	Miotic	Normal	Normal to mydriatic	Mydriatic
Pain	+ ulcer +/- without	+/-	+/-	-	+/-	+/-
Other signs	Corneal vessels, edema, pigment	Mucopurulent discharge	Low IOP, edema, hypopyon, ciliary flush		Pain opening mouth	Edema, buphthalmos, lens sublux, Haab's striae