PEER REVIEWED

Clinical Approach to the CANINE RED EYE

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The acute red eye is a common clinical challenge for general practitioners. Redness is the hallmark of ocular inflammation; it is a nonspecific sign related to a number of underlying diseases and degree of redness may not reflect the severity of the ocular problem.

Proper evaluation of the red eye depends on effective and efficient diagnosis of the underlying ocular disease in order to save the eye's vision and the eye itself.[1,2]

SOURCE OF REDNESS

The conjunctiva has small, fine, tortuous and movable vessels that help distinguish conjunctival inflammation from deeper inflammation (see **Ocular Redness** algorithm, page 16).

- **Conjunctival hyperemia** presents with redness and congestion of the conjunctival blood vessels, making them appear more prominent, and is associated with extraocular disease, such as *conjunctivitis* (**Figure 1**). If severe intraocular inflammation is present, conjunctival hyperemia can also occur in conjunction with episcleral injection.[1]
- **Subconjunctival hemorrhage** appears as amorphous areas of deep red below the conjunctiva, obscuring the view of the individual vessels. Subconjunctival hemorrhage occurs in *over-restraint*, *traumatic injury*, *clotting disorders*, and *strangulation* (**Figure 2**).[1]
- **Episcleral injection** causes redness because of congestion of the deep episcleral vessels, and is characterized by straight and immobile episcleral vessels, which run 90° to the limbus. Episcleral injection is an external sign of intraocular disease, such as anterior uveitis and glaucoma (**Figures 3 and 4**). Occasionally, episcleral injection may occur in diseases of the sclera, such as *episcleritis* or *scleritis*.[1]
- **Corneal Neovascularization**
  - » **Superficial:** Long, branching corneal vessels; may be seen with *superficial ulcerative* (**Figure 5**) or *nonulcerative keratitis* (**Figure 6**)
  - » **Focal deep:** Straight, nonbranching corneal vessels; indicates a *deep corneal keratitis*
  - » **360° deep:** Corneal vessels in a 360° pattern around the limbus; should arouse concern that *glaucoma* or *uveitis* (**Figure 4**) is present[1,2]
- **Hyphema or hemorrhage** within the eye appears as either a:
  - » Settled line of dull to bright red in the anterior chamber
  - » Diffuse redness filling the entire chamber (**Figure 7**).

  Hyphema can result from *clotting disorders*, *severe blunt trauma*, or *uveitis*, and can be associated with *systemic hypertension*.

Figure 1. *Ventral palpebral conjunctival hyperemia and chemosis secondary to allergic conjunctivitis (**A**); note small, fine, tortuous vessels and lymphoid vessels (**B**).*

DISEASES & DIAGNOSTICS

All red eyes must be evaluated for 3 key ocular diseases that may cause vision loss in an eye (**Table 1,** page 14):

1. **Corneal ulceration**
2. **Glaucoma**
3. **Uveitis**

A few basic diagnostic procedures can quickly assess whether these diseases are present; they should be performed in the following order for all patients with ocular signs:

1. **Schirmer tear test (STT):** Aids in diagnosis of conditions associated with decreased tear production, such as keratoconjunctivitis sicca (KCS), and should be performed before any medications are administered to the ocular surface
2. **Fluorescein stain:** Is critical for diagnosis of corneal ulceration[1,2]
3. **Tonometry:** Is critical for diagnosis of glaucoma and uveitis[1,2]

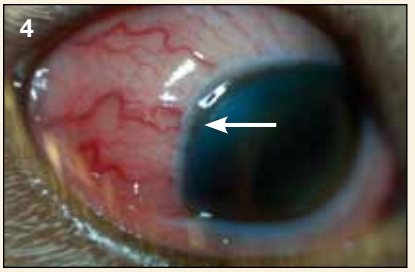
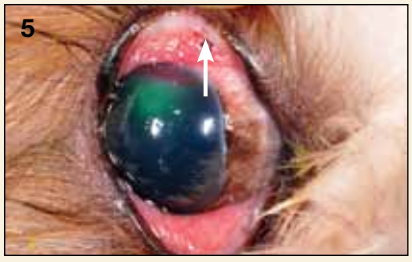


Figure 2. *Subconjunctival hemorrhage; note diffuse redness with no obvious congestion of bulbar vessels*
Figure 3. *Episcleral injection associated with glaucoma secondary to an anterior luxated lens; note presence of episcleral vessels 90° to the limbus.*
Figure 4. *360° perilimbal deep corneal vascularization (**arrow**); note episcleral injection (straight, nonmoveable, perilimbal, episcleral vessels approximately 90° to limbus).*



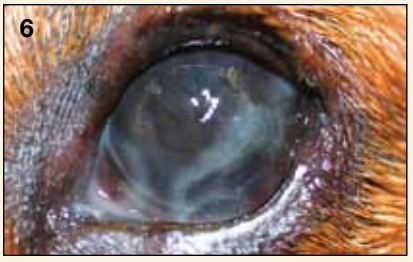


Figure 5. *Palpebral and bulbar conjunctival hyperemia and chemosis associated with superficial ulcer secondary to an ectopic cilium (**arrow**). Superficial corneal neovascularization is present in the dorsal cornea; note long and branching corneal blood vessels.*
Figure 6. *Superficial corneal neovascularization and melanosis, in association with adherent and tenacious mucopurulent discharge secondary to KCS; note long, branching corneal blood vessels, which confirms their superficial location.*
Figure 7. *Hyphema secondary to anterior uveitis; note diffuse bright red color and clot obscuring the pupil.*

Once an examination and these diagnostics are completed, the eye's condition can be classified as:

- Extraocular (conjunctival or corneal)
- Intraocular (glaucoma or uveitis)
- Ocular manifestation of systemic disease.

CORNEAL ULCERS

Causes of Red Eye

Corneal ulcers result in corneal vascularization, which appears as a "red eye."

Corneal blood vessels are an indication of chronic disease and, generally, take 1 to 3 days to proliferate on the corneal surface. Uncomplicated corneal ulcers typically heal in 3 to 5 days; ulcers that do not heal in this time period must be closely evaluated for confounding factors. Underlying disease that can impede healing include:

- KCS (low STT values, rapid tear breakup time)
- Adenexal disease (entropion, distichia, ectopic cilia)
- Chronic corneal exposure (lagophthalmos, exophthalmos).

They are often associated with other signs of corneal melanosis (or "pigmentation"). Corneal vascularization can occur with nonulcerative corneal disease, but this article strictly focuses on ulcerative disease.

Clinical Note: ***Blepharospasm*** *is seen with most forms of corneal disease but is a common and nonspecific sign of pain associated with many ocular diseases.*

Diagnosis & Classification

Once an ulcer has been identified with positive fluorescein staining, further classification allows proper therapeutic interventions and prevents catastrophic complications related to lack of treatment.

Corneal ulcers are classified as superficial or deep:[1]

- **Superficial corneal ulcer:** Has even and superficial fluorescein stain uptake, with no visible loss of stroma,[3] and presence of long, branching vessels over the cornea (**Figure 5**).
- **Deep corneal ulcer:** Has an irregular surface, with loss of corneal stroma, and presence of focal, fine, nonbranching vessels (**Figure 8**, page 15).

Superficial non-healing ulcers:

- Indicated by superficial staining with diffuse borders due to stain under running a nonadherent epithelial lip that develops *secondary to abnormal wound healing.*
- Can be associated with KCS (**Figure 6**), adnexal disease, chronic corneal exposure, or foreign bodies (**Figure 9**, page 15).[3]
- Others, however, are believed to occur without concurrent disease and are, therefore, associated with primary corneal disease and referred to as spontaneous chronic corneal epithelial defects (SCCEDs).[3]

Corneal malacia (melting ulcer):

- Presents with visible defects in the corneal surface; corneal malacia[4] appears as soft, gelatinous cornea around

TABLE 1. THREE KEY OCULAR DISEASES THAT MAY CAUSE VISION LOSS

DISEASE	CAUSE OF RED EYE	DIAGNOSTICS	TREATMENT
Corneal Disease or Ulceration	Corneal vascularization **Superficial corneal lesion:** Presence of long, branching vessels over the cornea **Deep corneal disease:** Presence of focal, fine, nonbranching vessels	• Slit-lamp examination • Fluorescein staining • Cytology • Culture & sensitivity	**Superficial ulcer:** • Treat underlying disease • Topical atropine • Topical antibiotic • Oral NSAID **Superficial nonhealing ulcer:** • Treat underlying disease • Debridement of ulcer • Grid keratotomy or DBD
Glaucoma	Episcleral injection Deep corneal vascularization (360° perilimbal pattern) **Primary glaucoma:** Usually associated with a narrow or closed filtration angle **Secondary glaucoma:** Often seen with uveitis or anterior lens luxation	• Tonometry • Gonioscopic examination • High-resolution US or US biomicroscopy	**Medical:** • Prostaglandin analogue drops • IV mannitol • Oral CAIs **Surgical:** • Laser cyclophotocoagulation & anterior chamber shunts • Endoscopic laser cyclophotocoagulation • Enucleation or evisceration
Anterior Uveitis	Episcleral injection Deep corneal vascularization (360° perilimbal pattern) Hyphema (blood in anterior chamber) Iris neovascularization	• Tonometry • Ocular US **Dx of Underlying Disease:** • CBC & serum biochemistry • Urinalysis • Tick-borne disease titers • Thoracic & abdominal radiographs • Abdominal US	• Topical atropine 1% • Topical prednisolone acetate 1% and NSAIDs • Systemic NSAIDs • Systemic steroids (only if infectious causes ruled out)

CAI = carbonic anhydrase inhibitor; CBC = complete blood count; DBD = diamond burr debridement; IV = intravenous; NSAID = nonsteroidal anti-inflammatory drug; US = ultrasound

the edges of the ulcer and in the ulcer bed (**Figure 8**) or stromal loss, both of which are due to activation of matrix metalloproteinases.

- Infiltration of the corneal stroma with white blood cells (WBCs; visible as creamy or yellow corneal opacity) often occurs in conjunction with corneal melting or stromal loss; this infiltration is considered highly suggestive of bacterial or fungal infection.

Deep corneal ulcer (descemetocele):

- Indicated by complete stromal loss and exposure of descemet's membrane
- Corneal stain uptake will occur in the walls but not the floor of the ulcer, producing a characteristic donut-shaped region of fluorescein stain retention.
- Severe condition in which the eye is in grave danger of perforation[4]; urgent referral to a specialist should be recommended to the client.

Clinical Note: ***Ulcers with greater than 50% stromal loss and malacia*** *require more aggressive medical management; sometimes surgical correction is necessary.*

Treatment

Topical therapy for superficial ulceration is geared toward preventing infection and alleviating pain. Unless the ulcer is infected, topical drugs do not promote healing; therefore, patients should be evaluated for underlying ocular disease (eg, KCS) and treated accordingly and concurrently. Routine corneal cytology is indicated to rule out low-grade infection.

Clinical Note: ***Superficial and uncomplicated ulcers*** *should heal in 3 to 5 days.*

Management of **superficial uncomplicated corneal ulcers** consists of:[4]

- *Topical atropine* once or twice daily until dilation is achieved to control ciliary muscle spasm and ocular discomfort. Atropine reduces tear production and should be decreased in frequency or discontinued after clinical effect. Most uncomplicated ulcers only require 2 to 3 days of treatment.
- *Topical broad-spectrum bactericidal antibiotic*, such as neomycin and bacitracin in combination with polymyxin B (ointment) or gramicidin (solution), three times daily.
- *Oral nonsteroidal anti-inflammatory drug* (NSAID) for additional comfort.

Simple ulcers should be rechecked within 5 days; therapy should be continued until resolution of the ulcer.

Management of **superficial non-healing ulcers** consists of:[3]

- *Treatment of concurrent disease*
- *Medical management*

Figure 8. *Deep stromal ulcer with a central descemetocele secondary to corneal malacia and stromal loss; 360° superficial corneal neovascularization is also present.*

Figure 9. *Foreign body posterior to the third eyelid* ***(A);*** *note hyperemia and chemosis of palpebral, bulbar, and third eyelid conjunctiva. Presence of superficial corneal ulcer following removal of foreign body and fluorescein staining* ***(B).***

Figure 10. *Episcleral injection and conjunctival hyperemia, chemosis, mydriasis, and diffuse corneal edema associated with primary glaucoma.*

Figure 11. *Anterior uveitis with diffuse corneal edema and secondary glaucoma; note 360° deep perilimbal deep neovascularization, diffuse edema, and slight mydriasis*

Figure 12. *Iris neovascularization (rubeosis iridis), episcleral injection, and miosis secondary to anterior uveitis.*

» As outlined for uncomplicated ulcers
» Do not use atropine in patients with KCS

Management of **SCCEDs** associated with primary corneal disease:

- *Medical management* as outlined for uncomplicated ulcers
- *Debridement of ulcer* with a cotton tip applicator
- *Contact lenses* to provide comfort during the healing process, if needed
- *Grid keratotomy* or *diamond burr debridement* if ulcer fails to heal (see **Diamond Burr Debridement**)

Treatment of **deep and melting ulcers** consists of:

- *Topical atropine* 2 to 3 times daily until dilation; do not use atropine in patients with KCS
- *Topical broad-spectrum antibiotic* Q 1 to 4 H; fluoroquinolones, such as ofloxacin, have good broad-spectrum efficacy
  » Corneal ulcers should be carefully evaluated with cytology to guide initial antibiotic therapy
  » Culture and sensitivity are indicated to confirm antibiotic choice
- *Compounded 50 mg/mL cefazolin* is indicated if gram-positive organisms are present; only used in conjunction with other antibiotics
  » Cefazolin eye drops can be compounded by reconstituting a 1-g vial of injectable cefazolin with 2.5 mL of sterile water; shake the mixture until dissolved and add it to a 15-mL bottle of artificial tears.
  » Keep refrigerated and discard after 10 days[5]
- *Topical protease inhibitors*, such as autologous serum, N-acetyl-cysteine, or EDTA, are applied Q 2 H until corneal malacia and stromal loss are controlled. Serum can be obtained in private practice via venipuncture and centrifugation, with sterile preparation and storage. Oral doxycycline (10 mg/kg Q 24 H) also acts as a proteolytic inhibitor[9-12]
- *Oral NSAID* for additional comfort.

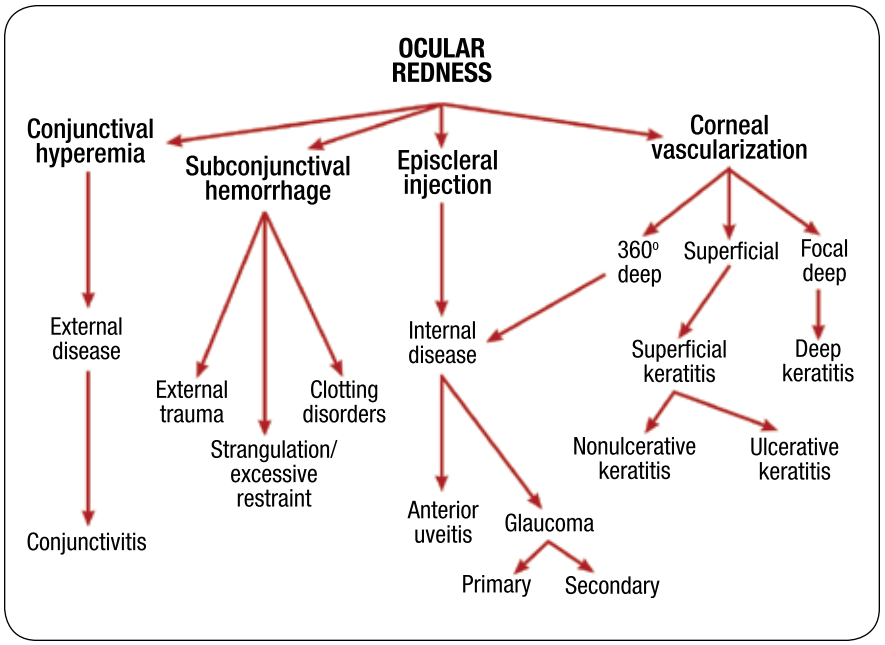
Patients should be re-evaluated within 24 hours for signs of improvement. Signs of improvement include:[4]

- Increased pupil dilation
- Smoothing of the epithelial margin
- Reduction of:
  » Corneal and episcleral/conjunctival blood vessel perfusion

DIAMOND BURR DEBRIDEMENT (DBD) is a relatively new treatment modality that:

- Provides safe and effective therapy for SCEDDs (no need for grid keratotomy)[6,7]
- Has a low risk of injury to the deeper corneal layers[8]
- Does not require extensive specialized training
- Is an affordable treatment solution.

However, only use grid keratotomy and DBD for superficial ulcerations *without stromal loss* and make sure *no infectious process* is present prior to performing either procedure.



» Corneal edema
» Mucopurulent discharge
» Signs of pain (blepharospasm and epiphora)
» Stromal loss, malacia, and WBC infiltration.

Referral & Advanced Therapy

It is prudent to refer all patients with deep stromal ulcers, descemetoceles, and ruptured eyes to an ophthalmologist for surgical evaluation in order to save the globe and vision. Surgical interventions include conjunctival flap, corneal graft, or corneal-conjunctival transposition.[4,5]

Recently, bioscaffold materials (ACell, acell.com) have shown promise for corneal ulcer treatment. These materials help promote healing of deep corneal ulcers and, when combined with a conjunctival flap, can be used for surgical repair of descemetoceles or penetrating corneal injuries.[13]

Another recent study has shown that the use of amniotic membranes can decrease scarring, promote healing of corneal injuries, and provide anti-inflammatory properties.[14]

GLAUCOMA

Causes of Red Eye

In a patient presenting with glaucoma, redness of the eye is due to episcleral injection, with deep corneal vessels that form a 360° perilimbal pattern if the condition is chronic.

Clinical Note: ***Pain, corneal edema, and disturbance of vision*** *may be present with glaucoma.*

Diagnosis

The only diagnostic sign of glaucoma is increased intraocular pressure (IOP).

- IOP is measured by applanation (Tono-Pen, reichert.com; I-pen Vet, imedpharma.com) or rebound (TonoVet, icaretonometer.com) tonometry.
- Normal IOP in the dog varies between 10 to 20 mm Hg
  » **Applanation tonometry:** Mean IOP ± standard deviation (SD) in the dog is reported as 17 ± 4 mm Hg[15]
  » **Rebound tonometry:** Mean IOP ± SD in the dog is reported as 10.8 ± 3.1 mm Hg (range, 5–17 mm Hg)[16]
- A recent study has shown that inexperienced personnel can obtain comparable intraocular values in dogs using either applanation or rebound tonometry.[17]

Clinical Note: ***Minor variations in IOP*** *are noted secondary to diurnal variations, corneal scarring and pigmentation, and stress related to the* white coat effect.[18-20]

Classification

Primary glaucoma (**Figure 10,** page 15) in dogs is almost always unilateral and often associated with a narrow or closed filtration angle.

- Once glaucoma becomes severe, episcleral injection is the predominate cause of redness.
- Increased IOP typically results in slow pupillary light responses and mydriasis in the affected eye.
- Buphthalmia (enlargement of the globe) occurs in patients with chronic glaucoma, but not in those with acute glaucoma or ocular hypertension.

Secondary glaucoma is often seen with uveitis (**Figure 11**, page 15) or anterior lens luxation (**Figure 3**).

Referral for gonioscopic examination or advanced imaging is required to classify type of glaucoma based on the iridocorneal angle morphology. While only the most superficial parts of the iridocorneal angle can be visualized with a goniolens, the entire ciliary cleft can be visualized with advanced imaging techniques, such as high-resolution ultrasonography or ultrasound biomicroscopy.[21,22]

Treatment

Initial medical therapy for acute primary glaucoma is aimed at rapidly reducing IOP.

- *Ophthalmic solutions containing prostaglandin analogues*, such as latanoprost 0.005% or travoprost 0.004% (if available), should be used to rapidly decrease IOP; latanoprost 0.005% is administered Q 1 to 2 H until IOP decreases.[23]
- *Intravenous mannitol* (1–2 g/kg, slowly administered over 15–20 min) is used as an alternative to topical prostaglandin analogues or in patients that do not respond to topical therapy within 2 hours.
  » Withhold water for 4 hours after administering mannitol.[24]
  » Adverse effects may occur in dehydrated or systemically ill animals, especially those with cardiac or renal disease; therefore, patient monitoring is critical when administering any osmotic diuretic, such as mannitol.
- *Oral carbonic anhydrase inhibitors* (CAIs), such as

methazolamide (2–5 mg/kg Q 8–12 H), decrease aqueous humor formation in the ciliary body.

If secondary glaucoma associated with uveitis, hyphema, or lens luxation is suspected, latanoprost should not be used due to its miotic effects, especially if the lens is in the anterior chamber.[17]

Combinations of medications to maintain lower IOP within the normal or acceptable range for dogs are often needed.

- *Topical CAIs*, such as dorzolamide or brinzolamide, are recommended Q 8 H.[26]
- *Topical beta-blockers*, such as timolol maleate 0.5% or betaxolol 0.5% (Q 8–12 H) are insufficient to control IOP when used alone but have a mild additive effect in lowering IOP when used in conjunction with a topical CAI.[27]
- If needed, a *prostaglandin analogue* may be used Q 12 H to help normalize IOP.[28]

Oral CAIs, such as methazolamide (2–5 mg/kg Q 8–12 H) have been recommended to help maintain IOP.[29] However, a recent study demonstrated that, in dogs with glaucoma, addition of an oral CAI did not improve reduction of IOP over the decrease achieved with a topical CAI (dorzolamide) alone.[30] In addition, when compared to a topical CAI alone, combination therapy with a topical and oral CAI was no more likely to provide long-term control in dogs with primary or secondary glaucoma.[30-32]

Clinical Note: ***Referral should be considered*** *early in the disease process, especially if IOP fails to respond to medical management.*

Glaucoma is ultimately a surgical disease. Medical therapy will typically become ineffective within a year. Surgical intervention may prolong vision but has a relatively poor long-term success rate.

- *Laser ablation of the ciliary body (cyclophotocoagulation)* or *anterior chamber shunts*, such as gonioimplants, used alone or together, offer longer periods of IOP control than medical management alone.[33]
- *Endoscopic laser cyclophotocoagulation* provides a longer period of control when combined with a gonioimplant, medical therapy, and frequent examinations.[34-36]
- *Enucleation or evisceration* is a humane option in most cases of blindness since the goal of therapy—if the eye is blind—should be pain relief.

Prevention

Generally, primary glaucoma begins as a unilateral disease but, eventually, the other eye develops it as well. Treating the "second" eye with a prophylactic medication even if IOP is within the normal range is critical in order to delay onset of disease.

- *Topical dorzolamide; beta blockers, such as timolol or betaxolol; and demecarium bromide* are common prophylactics.[31,37]
- When a topical CAI is applied prophylactically, glaucoma develops, on average, in 196 days.[31] Published time to onset of glaucoma for demecarium bromide and betaxolol when used prophylactically is 31 months and 30.7 months, respectively.[37]

ANTERIOR UVEITIS

Causes of Red Eye

When anterior uveitis is present, redness of the eye is due to episcleral injection, with 360° deep corneal vascularization if the disease is chronic (**Figure 4 and 11**). Other causes of redness related to anterior uveitis include

TABLE 2. CAUSES OF CANINE ANTERIOR UVEITIS[41,42]

TYPE	DISEASE
Autoimmune	Immune-mediated thrombocytopenia Immune-mediated vasculitis Lens-induced uveitis (secondary to cataracts) Pigmentary uveitis (golden retrievers) Phacoclastic uveitis (secondary to lens capsular rupture) Retinal detachment syndrome (steroid responsive) Uveodermatologic syndrome (Vogt-Koyanagi-Harada-like syndrome)
Infectious	**Algae:** *Prototheca* species **Bacteria:** Leptospirosis, *Bartonella* and *Brucella* species **Mycoses:** *Blastomyces* and *Cryptococcus* species, *Coccidioides immitis*, *Histoplasma capsulatum* **Parasitic:** *Angiostrongylus vasorum, Dirofilaria immitis* **Protozoa:** *Toxoplasma gondii, Leishmania donovani, Neosporium caninum* **Tick-borne disease:** *Borrelia burgdorferi, Ehrlichia canis* and *platys*, *Rickettsia rickettsii* **Viruses:** Canine adenovirus (types 1 and 2), canine distemper virus
Neoplastic/ Paraneoplastic	Histiocytic proliferative disease Hyperviscosity syndrome Lymphosarcoma Metastatic tumors Primary intraocular tumors: Adenomas, adenocarcinomas, melanomas
Other	Endotoxemia Idiopathic (approximately 50%–60% in dogs) Metabolic: Coagulopathies, hyperlipidemia, systemic hypertension Radiation therapy Reflex uveitis: Episcleritis, keratitis (ulcerative and nonulcerative), scleritis Trauma

hyphema (blood in the anterior chamber [**Figure 7**]) and rubeosis iridis (iris neovascularization; **Figure 12**, page 15).

Clinical Note: *Subtle and early uveitis may cause only mild ocular redness; be careful not to misdiagnose it as conjunctivitis.*

Diagnosis

Other **intraocular clinical signs** of anterior uveitis include:

- Aqueous flare (cloudiness of the aqueous humor)
  - » Identified with a small focal light or slit beam
  - » The fluid in the anterior chamber should be crystal clear; when flare is present, the light is visualized as it passes through the aqueous (Tyndall effect)
- Fibrin within the anterior chamber
- Hypopyon
- Keratic precipitates
- Synechiae.

Nonspecific **clinical signs** include:

- Blepharospasm
- Epiphora
- Miosis
- Ocular discharge.

With anterior uveitis, IOP is generally low, unless the animal has developed secondary glaucoma. As a rule, IOP less than 10 mm Hg, or a difference of 5 to 10 mm Hg between eyes, suggests uveitis.[1,2]

All patients with anterior uveitis should have the following performed:

- Complete blood count and serum biochemical profile
- Urinalysis
- Titers for tick-borne diseases
- Thoracic (3 views) and abdominal radiographs
- Abdominal ultrasound.

Clinical Note: ***Ocular ultrasound*** *can help determine extent of ocular disease if anterior segment changes are severe and the posterior segment cannot be visualized.*

Systemic Disease

Uveitis, whether unilateral or bilateral, is most often due to a systemic cause. Systemic causes of uveitis include infectious, autoimmune, and neoplastic disorders (**Table 2**). Uveitis is considered nonsystemic if there is evidence of:

(continued on page 40)

(continued from page 18)

- Cataracts (lens-induced uveitis)
- Corneal ulceration
- Intraocular neoplasia
- Other external evidence of trauma.

As for any other inflammatory conditions, underlying etiology must be determined in order to institute proper therapy. However, this determination is often impossible even with thorough diagnostic investigation.

Treatment

Treatment should be directed at addressing the primary cause and decreasing pain and inflammation.

- *Atropine 1%* Q 6–8 H may be used to relieve miosis and pain; it should not be used in patients with normal or elevated IOP in the face of uveitis as it will exacerbate glaucoma.
- *Prednisolone acetate 1% ophthalmic suspension* (Q 4–6 H[38]) and/or *topical NSAIDs*, such as diclofenac sodium 0.1% or flurbiprofen sodium 0.03%, can be used alone or in combination to reduce intraocular inflammation.
- *Systemic NSAIDs*, such as IV meloxicam (0.2 mg/kg)[39] and oral firocoxib (5 mg/kg),[40] may be beneficial in controlling inflammation associated with uveitis; however, concurrent disease and hepatic and renal function should be considered before using systemic NSAIDs.
- *Systemic glucocorticoids* should only be used to treat uveitis after infectious causes and neoplasms requiring chemotherapy, such as lymphoma, have been ruled out.

Clinical Note: ***Referral to an ophthalmic specialist*** *is appropriate for management of severe or resistant cases of uveitis.*

OTHER CAUSES OF RED EYE

This article has focused on causes of red eye that are most likely to threaten vision. However, it is important to remember that there are other causes for the apparently "acute" red eye, including more insidious disorders that are perceived as acute by the owner. These include:

- **Blepharitis associated with lid lacerations, pyogranulomatous blepharitis,** and **infectious blepharitis**, which often present as a cause of acute ocular redness
- **Conjunctivitis** (KCS, allergic conjunctivitis) and **prolapsed gland of the third eyelid** ("cherry eye")
- **Nonulcerative keratitis**, such as chronic superficial keratitis (pannus), qualitative tear film abnormalities, and other keratopathies without ulceration
- **Orbital diseases** (cellulitis, abscess, neoplasia), which routinely present with conjunctival hyperemia and chemosis.

IN SUMMARY

Correct diagnosis and treatment of the red eye are important to prevent loss of vision, the globe, or, in some cases, loss of life. Corneal ulcers, uveitis, and glaucoma must all be considered and can be diagnosed by performing a thorough ophthalmic examination, STT, fluorescein staining, and tonometry. Each of these diagnoses requires prompt and specific treatment in order to ensure a positive outcome. ■

CAI = carbonic anhydrase inhibitor; DBD = diamond burr debridement; IOP = intraocular pressure; KCS = keratoconjunctivitis sicca; NSAID = nonsteroidal anti-inflammatory drug; SCCED = spontaneous chronic corneal epithelial defects; SD = standard deviation; STT = schirmer tear test; WBC = white blood cell

Figures courtesy of University of Georgia teaching collection

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For article references, go to **todaysveterinarypractice.com**.

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