Uveitis is a general term used to describe inflammation of the intraocular vascular uveal tract, which comprises the iris, ciliary body, and choroid. Anterior uveitis refers to inflammation in the anterior segment of the eye affecting the iris and ciliary body. Posterior uveitis describes inflammation of the choroid, while chorioretinitis indicates adjacent retinal involvement. Panuveitis describes inflammation of all uveal tissues.

Causes of uveitis are numerous and often elusive. Thorough ocular examination enables diagnosis of uveitis and determination of whether it is unilateral or bilateral; it can be asymmetrically bilateral. Physical examination and additional diagnostic tests are often needed to help identify the underlying cause. Targeted ophthalmic therapy must be aggressive to control intraocular inflammation while any underlying cause is treated. Treatment is often prolonged, commonly continuing for 2 to 4 weeks past the resolution of clinical signs. Educating clients on the potential complications of uncontrolled uveitis (cataracts, glaucoma, loss of vision, pain) greatly increases compliance with therapy and follow-up visits to maximize success.

### CLINICAL SIGNS AND FINDINGS

**Acute Changes**

Observable signs and detailed examination findings in patients with uveitis can be numerous (BOX 1 AND FIGURES 1–7). Performing a thorough ocular examination that includes systematic evaluation of all ophthalmic structures...

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**FIGURE 1.** Canine eye with blastomycosis-induced uveitis manifesting as a “red eye” with episcleral blood vessel injection and rubeosis irides. Note the hazy view of the iris due to corneal edema and aqueous flare.

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**DETECTIVE WORK**

Managing uveitis centers on controlling inflammation, reducing pain, and preserving vision, but identifying the underlying condition requires sleuthing skills.
for possible abnormalities is critical (see **DIAGNOSIS** on p. 37).

Although no single patient has every potential clinical sign and finding associated with uveitis, the ability to recognize pertinent clues aids in accurate diagnosis. The number and severity of ophthalmic findings commonly correlate with degree of ocular pain; however, animals with neoplasia-induced uveitis or only posterior uveitis may not show signs of ocular pain unless concurrent secondary glaucoma is present. Secondary glaucoma can occur with acute or chronic uveitis depending on the cause and severity and rapidly leads to irreversible vision loss if not recognized and treated appropriately.

**Chronic Changes**

Ocular changes secondary to uveitis may develop in a short time or with chronicity (**BOX 2**). Some changes can affect vision or lead to irreversible blindness. Iris hyperpigmentation, pigment deposits on the anterior lens capsule ("footprints of synechia"), and chorioretinal scars, visible as well-defined hyperreflective lesions in the tapetal fundus or depigmented lesions in the nontapetal fundus, may provide evidence of past uveitis even if active signs of inflammation are absent.

**CAUSES OF UVEITIS**

When considering diagnostic tests and treatment for uveitis, it is helpful to determine whether the cause is ocular or nonocular. In some cases, such as uveitis secondary to immune-mediated disease or neoplasia, this distinction may depend on the specific underlying disease.

**Primary Ocular Causes**

Reflex uveitis is a common result of corneal ulceration or abscessation that stimulates corneal nerves,

| BOX 1 Clinical Signs and Examination Findings Associated with Uveitis |
|-----------------|------------------|-----------------|
| **General**    |                  |                  |
| Blepharospasm  | Third eyelid elevation due to enophthalmos |                  |
| Rubbing at the eye | Photophobia      | Epiphora         |
| Decreased vision or blindness |                  |                  |
| **Ocular surface** |                  |                  |
| "Bloodshot eye" due to episcleral and conjunctival vascular injection (**FIGURE 1**) |                  |
| Corneal edema (localized or diffuse; **FIGURES 1 AND 2**) | Dense peripheral corneal neovascularization |
| **Intraocular anterior segment** |                  |                  |
| Keratic precipitates (most notable ventrally)* (**FIGURE 2**) |                  |
| Aqueous flare* (**FIGURES 1 AND 3**) | Hypopyon* |                  |
| Hyphema (**FIGURE 4**) | Fibrin clots or strands* (**FIGURES 4 AND 5**) | Iris hyperemia or "rubeosis irides"* |
| Iris swelling (**FIGURES 5 AND 6**) | Iris color change (especially visible with lighter irides) | Iridal hemorrhage |
| Iridal hemorrhage | Peripheral anterior synechia* | Posterior synechia* |
| Dyscoria (**FIGURES 5 AND 6**) | Miosis and/or resistance to pharmacologic dilation | Low intraocular pressure |
| Secondary glaucoma |                  |                  |
| **Intraocular posterior segment** |                  |                  |
| Vitreal cells* | Vitreal hemorrhage |                  |
| Vitreal degeneration | Subretinal exudates causing hyporeflectivity in tapetal fundus or white-yellow discoloration in nontapetal fundus* (**FIGURE 7**) |                  |
| Retinal hemorrhage (**FIGURE 7**) | Retinal detachment (**FIGURE 7**) | Optic neuritis |

*Finding specific to uveitis.

| BOX 2 Potential Sequelae of Uncontrolled Uveitis |
|-----------------|------------------|-----------------|
| Cataract formation (usually capsular and outer cortex) | Iris bombeé due to complete circumferential posterior synechia | Lens subluxation or luxation |
| Phthisis bulbi | Pre-iridal fibrovascular membrane | Retinal degeneration |
FIGURE 2. Diabetic miniature schnauzer eye with lens-induced uveitis due to rapidly developing diabetic cataracts. Slit lamp examination demonstrates corneal edema as evidenced by the large space between where the beam first hits the fluorescein stain in the corneal tear film and the numerous keratic precipitates present on the endothelial surface. Iris and lens details were difficult to visualize owing to hazy ocular media.

FIGURE 3. Aqueous flare visible with slit lamp examination. Note the beam of light traversing the anterior chamber between the cornea and lens.

FIGURE 4. Large anterior chamber fibrin clot and hyphema hiding a miotic pupil.

FIGURE 5. Histoplasmosis causing uveitis in a cat. (A) Eye at initial presentation with episcleral blood vessel injection, rubeosis irides, localized peripheral iris swelling dorsolaterally, and a fibrin clot in the anterior chamber obscuring the miotic pupil. (B) Eight days after initiation of treatment for uveitis and histoplasmosis, episcleral injection and iris changes are reduced; fibrin is resolved. (C) Six months after initial diagnosis, uveitis recurred because the clients prematurely stopped fluconazole treatment 2 to 3 months prior. Note dorsal and dorsolateral iris swelling, rubeosis irides, dyscoria due to posterior synechia dorsolaterally, and a central fibrin clot adhered to the lens.
triggering an axonal reflex to release prostaglandins inside the eye. Simple corneal ulcers may cause mild anterior uveitis (e.g., relative miosis, trace aqueous flare), while infectious keratitis can cause more profound uveitis (e.g., severe miosis, hypopyon, anterior chamber fibrin). Necrotizing scleritis is an uncommon cause of reflex uveitis.

Trauma may involve a penetrating injury (e.g., cat claw) or blunt insult (e.g., tennis ball impact). In both cases, the uveitis severity depends on the force of injury, degree of trauma, and structures involved. If the lens capsule is ruptured during penetrating trauma, more aggressive treatment is needed, including broad-spectrum systemic antibiotic therapy, to help prevent globe-threatening endophthalmitis.

Primary ocular causes of uveitis related to immune-mediated disease include lens-induced uveitis, which may be due to protein leakage through an intact lens capsule with advanced cataract or to lens capsule rupture. Pigmentary uveitis and pigmentary cystic glaucoma (“golden retriever uveitis”) are also examples of immune-mediated primary ocular disease; steroid-responsive retinal detachment with or without anterior uveitis is also considered to be in this category.

Nonocular Causes
Systemic infections are common nonocular causes of uveitis. Septicemia or endotoxemia from any cause may lead to uveitis.

Neoplastic causes of uveitis include primary intraocular tumors, such as uveal melanoma, iridociliary adenoma/adenocarcinoma, and feline post-traumatic ocular sarcoma. Metastatic neoplasia is a nonocular cause of uveitis, lymphosarcoma being the most common example. Other malignant sarcomas, carcinomas, transmissible venereal tumors, and histiocytic proliferative disorders have also been reported as causing uveitis.

Nonocular immune-mediated causes of uveitis include systemic autoimmune diseases: uveodermatologic syndrome, immune-mediated thrombocytopenia, and immune-mediated vasculitis.

Vaccine-induced uveitis is much less common with the canine adenovirus (CAV)-2 modified live virus vaccine than the previous CAV-1 vaccine. When vaccine-induced uveitis occurs, it typically manifests 10 to 14 days after immunization and more commonly affects puppies or young adult dogs. Fortunately, most cases improve over 2 to 3 weeks with appropriate treatment for immune-mediated disease.

Idiopathic uveitis is the most common diagnosis for uveitis from a nonocular cause (40% to 60% of cases). It is a diagnosis of exclusion; other causes must be ruled out through thorough ocular examination, physical examination, and adjunctive diagnostic testing. In middle-aged to older cats with idiopathic uveitis leading to secondary glaucoma, ocular histopathology after enucleation may identify immune-mediated lymphocytic-plasmacytic uveitis. Cats matching this clinical picture should be strongly considered to have...
this type of uveitis, and chronic treatment may be needed.

**DIAGNOSIS**

A diagnosis of uveitis should begin by gathering history from the owner. Inquire about environmental and travel history, when the ocular signs were first noted, if there have been any changes in signs over time, and whether the owner has noticed any possible systemic issues (FIGURE 8). Signalment can be a very important clue with numerous ophthalmic diseases, so always consider the patient’s age and breed (TABLE 1).

A thorough examination to document ophthalmic lesions consistent with uveitis and to allow for tracking of progress is critical. Start by noting the presence and severity of general signs, then move in to consciously evaluate and qualify any ocular surface changes, anterior chamber infiltrates, iris changes, pupillary abnormalities, vitreal infiltrates, and fundic lesions.

Perform a Schirmer tear test (STT), measure intraocular pressure (IOP), and perform fluorescein staining on every patient, as the results may alter treatment decisions. For example, in an actively inflamed eye, IOP should be <10 to 15 mm Hg; higher values are inappropriate and should raise suspicion of secondary glaucoma.

**Ophthalmic Examination**

The ophthalmic examination should be used to identify any primary ocular causes (e.g., corneal ulcer, known trauma, advanced cataract, pigmentary uveitis). Consciously assess all ocular structures, including the ventral anterior segment, to look for keratic precipitates on the underside of the cornea or hypopyon settled in the anterior chamber. If third eyelid elevation makes the ventral aspect of the eye challenging to examine, try pointing the patient’s nose down to rotate the eye up and/or have an assistant lift the hind end of the patient off the table slightly, which usually causes the patient to bring the eye forward and the third eyelid to retract.

**FIGURE 8.** Diagnostic algorithm for uveitis. CBC = complete blood count; FNA = fine-needle aspiration; PE = physical examination.
It is strongly recommended to dilate the pupils with 1% tropicamide to allow posterior segment examination of both eyes if IOP is acceptably low. Posterior segment changes can provide clues to clinical severity, help dictate needs for systemic treatment, and guide diagnostic testing (e.g., presence of subretinal granuloma warrants fungal testing in endemic areas), and findings may influence visual prognosis.

The direct ophthalmoscope is also useful to assess for aqueous flare, which is visible evidence of blood–aqueous barrier breakdown. To evaluate for aqueous flare, select the smallest focal circular beam of light and hold the ophthalmoscope 5 to 10 mm in front of the cornea while grossly viewing from the side (45° to 90° angle) in a very dark examination room. A normal eye will show the light beam hitting the cornea, a void in the anterior chamber, then light hitting the anterior lens capsule and coursing through the lens to end at the posterior lens capsule. An eye in which the light beam continues through the anterior chamber (like a headlight beam in fog) to connect the cornea and lens has aqueous flare (FIGURE 3), with the degree of flare generally proportional to the severity of uveitis. In patients with systemic hyperlipidemia, even mild uveitis causes lipemic aqueous humor, which appears as pronounced white aqueous flare; patients that are suspected to have this issue should have fasting triglyceride and cholesterol testing performed to document current blood levels and plan concurrent systemic management. Treatment of uveitis typically results in rapid resolution of the lipemic aqueous humor.

After the systematic ocular examination, perform a complete physical examination and consider diagnostic tests to help determine an etiologic diagnosis and guide treatment. BOX 3 lists common diagnostic differentials for patients with uveitis. If the ophthalmic examination does not identify an obvious cause, underlying systemic issues should be investigated, with any abnormalities found on physical examination warranting further evaluation.

### Diagnostic Tests

#### Laboratory Tests

A complete blood count, serum chemistry profile, and urinalysis should be performed to look for signs of systemic inflammation or underlying disease that may suggest a cause (e.g., thrombocytopenia and tick-borne disease) or that could alter treatment (e.g., azotemia or elevated liver enzyme.

<table>
<thead>
<tr>
<th>TABLE 1 Signalment Clues to Differential Diagnosis in Patients with Uveitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>SIGNALMENT</td>
</tr>
<tr>
<td>Younger animals</td>
</tr>
<tr>
<td>Older animals</td>
</tr>
<tr>
<td>Young purebred cats</td>
</tr>
<tr>
<td>Young adult Arctic Circle breeds (Akita, husky)</td>
</tr>
<tr>
<td>Golden retrievers</td>
</tr>
<tr>
<td>German shepherds, mixed-breed</td>
</tr>
<tr>
<td>German shepherds, Australian shepherds, Labrador retrievers, and mixed-breed Labrador retrievers</td>
</tr>
<tr>
<td>Bernese mountain dogs</td>
</tr>
</tbody>
</table>

### Table 1 Signalment Clues to Differential Diagnosis in Patients with Uveitis

- **Younger animals**: Infectious disease
- **Older animals**: Neoplasia
- **Young purebred cats**: Unexplained uveitis
- **Young adult Arctic Circle breeds (Akita, husky)**: Uveodermatologic syndrome
- **Golden retrievers**: Pigmentary uveitis, pigmentary cystic glaucoma
- **German shepherds, mixed-breed**: Steroid-responsive retinal detachment
- **German shepherds, Australian shepherds, Labrador retrievers, and mixed-breed Labrador retrievers**: Usually bilateral; more common in dogs that have no identifiable underlying health problems or systemic disease. Concurrent anterior uveitis and/or vitreal hemorrhage may be present.
- **Bernese mountain dogs**: Systemic histiocytosis
levels would contraindicate systemic nonsteroidal anti-inflammatory drug [NSAID] use).

Additional tests for specific infectious agents should be performed as warranted by examination findings, signalment, and endemic area (BOX 4). These may be serologic, polymerase chain reaction, or urine antigen tests. Cats with uveitis may have multiple infectious etiologies, as some diseases (FeLV, FIV) predispose to other systemic infections.

**Imaging**

Thoracic radiography, abdominal radiography, and/or abdominal ultrasonography may be warranted based on history or physical examination findings, such as coughing, respiratory noise, or palpable abdominal masses. Even in animals with normal physical examination findings, imaging studies may reveal evidence of systemic infectious disease or neoplasia affecting internal organs and can provide valuable diagnostic sampling opportunities (e.g., aspiration of liver or splenic nodules).

**Cytology/Biopsy**

Sampling of external structures based on physical examination findings is a complementary and potentially very useful diagnostic tool. Fine-needle aspiration and cytology should always be performed on enlarged peripheral lymph nodes and is beneficial for suspicious subcutaneous masses. In endemic areas for fungal agents, impression smears should be performed on all draining skin wounds for cytologic evaluation. Skin lesion biopsy is warranted in young Arctic Circle breeds with uveitis if uveodermatologic syndrome is suspected and can be performed even in the absence of visible dermatologic changes.

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**BOX 3 Diagnostic Differentials in Patients with Uveitis**

- **Conjunctivitis or keratoconjunctivitis sicca**: Can cause a “bloodshot eye” with ocular discharge; however, intraocular examination is normal. STT results are low.
- **Glaucoma**: IOP is elevated. The pupil of the affected eye is dilated compared with the other eye.
- **Anterior lens luxation**: A clear lens in the anterior chamber may be confused with aqueous flare axially; however, even with a lens occupying the majority of the anterior chamber, the peripheral anterior chamber is clear. Endothelial contact damage from the lens may cause focal corneal edema, while elevated IOP due to secondary glaucoma causes diffuse corneal edema.
- **Corneal endothelial dystrophy or degeneration**: Leads to corneal edema, which can be focal or diffuse, but conjunctival hyperemia is generally absent, eyes are nonpainful, and there are no intraocular changes (e.g., pupils are symmetrical, IOP is normal).
- **Horner’s syndrome**: The narrowed palpebral fissure due to ptosis and enophthalmos must be differentiated from active squinting. Miosis is the only intraocular change, and signs resolve after topical phenylephrine application.
- **Systemic hypertension**: May cause hyphema, retinal hemorrhages, and/or serous retinal detachment. Measure blood pressure, perform laboratory testing, and start antihypertensive therapy. Treat hyphemic eyes with topical and/or systemic corticosteroids until blood clears and while monitoring for secondary glaucoma.

**BOX 4 Diagnostic Tests in Cats and Dogs with Uveitis**

**Cats**

- FeLV/FIV
- Coronavirus (if positive and high globulins on chemistry profile, may suggest feline infectious peritonitis)
- Toxoplasma (IgG and IgM)
- Bartonella
- Fungal agents (Cryptococcus neoformans, Histoplasma capsulatum, Blastomyces dermatitidis, Coccidioides immitis)

**Dogs**

- 4DX test
- Extensive tick disease panel
- Brucella (for breeding animals)
- Leptospira spp
- Toxoplasma/Neospora
- Fungal agents (Blastomyces dermatitidis, Coccidioides immitis, Cryptococcus neoformans, Histoplasma capsulatum, Aspergillus spp)

*Depending on the endemic area.
Additional Tests

Ocular ultrasonography is noninvasive and may be beneficial to look for an intraocular mass, retinal detachment, or other abnormality if cloudy ocular media preclude posterior segment exam. Ocular sampling is more invasive. It can be performed if other systemic diagnostic tests are unrewarding and infection or neoplasia is suspected; however, cytology of aqueous humor is usually nondiagnostic unless uveitis is due to lymphosarcoma.\textsuperscript{9,10} With suspected fungal infection, posterior segment sampling is more beneficial than aqueous humor, as ophthalmic involvement starts in the choroid. Fungal organisms are more commonly detected on vitreal cytology or subretinal aspiration (the latter may be considered on blind eyes with exudative retinal detachment when searching for an etiologic diagnosis).

Irreversibly blind, painful eyes should be removed for diagnostic and palliative benefit. Enucleation of a nonpainful, end-stage eye for histopathology of the globe may reveal the cause of uveitis, afford improved treatment planning to manage systemic and/or contralateral ophthalmic disease, and offer prognostic benefit.

Topical therapy alone may be sufficient for mild anterior uveitis, but systemic therapy is necessary for patients with severe anterior uveitis, posterior uveitis, or systemic disease. Appropriate treatment of underlying disease (when possible) is paramount, as insufficient management will result in recurrent or persistent uveitis (FIGURE 5).

Topical Therapy

Corticosteroids

These agents are the treatment of choice for uveitis and should be started immediately after diagnosis, even in patients with suspected systemic infection. Ophthalmic prednisolone acetate 1% or dexamethasone 0.1% achieves therapeutic levels in the aqueous humor and should be prescribed q6h or q3h depending on uveitis severity. Following clinical improvement, treatment frequency can be tapered over several weeks. Abrupt treatment cessation may result in recurrence of uveitis.

In the author’s experience, subconjunctival depot steroid injection using triamcinolone acetonide 4 mg or methylprednisolone 4 mg may be considered as adjunct initial therapy in severe cases or patients that are impossible to treat topically, but is rarely needed if topicals are used effectively.

Topical steroid use is contraindicated with corneal ulceration or infectious keratitis, given epithelial toxicity and immunosuppression that could delay wound healing and potentiate corneal infection. Sodium phosphate steroid forms and other ophthalmic formulations (e.g., hydrocortisone, betamethasone) have poor corneal penetration and should not be used to treat uveitis.

NSAIDs

Diclofenac 0.1%, flurbiprofen 0.03%, ketorolac 0.5%, and other NSAIDs are weaker than topical corticosteroids but may be used concurrently in severe uveitis cases or as a first choice if corticosteroid use is contraindicated (e.g., primary uveitis patient with concurrent corneal ulceration or a uveitic cat with ocular herpesvirus recrudescence).

Treatment frequency is typically q12h or q6h depending on uveitis severity. Q24h or q12h use may be continued long term if uveitis recurrence is possible or as a precautionary measure in cataract

Corticosteroids are best for immune-mediated disease and posterior uveitis and when intraocular hemorrhage is present.

TREATMENT

Immediate symptomatic management of uveitis is important to prevent adverse sequelae. Anti-inflammatory medications are imperative and should be gradually tapered to treat for 2 to 4 weeks past the resolution of clinical signs (e.g., normal IOP, resolved aqueous flare). A mydriatic drug is commonly indicated to improve comfort and prevent pupil adhesions. Specific therapy for underlying conditions should be initiated when appropriate.
patients at risk for lens-induced uveitis (e.g., diabetic or advanced cataracts) even if cataract surgery is not desired. Cats with suspected lymphocytic-plasmacytic uveitis will also benefit from chronic treatment. Patients receiving any long-term therapy should be monitored every 3 to 6 months.

**Mydriatic Drugs**

These agents are used to dilate the pupil, prevent posterior synechia, reduce pain from ciliary muscle spasm, and help stabilize the blood–aqueous barrier.

The treatment of choice is atropine sulfate 1% applied q24h to q8h depending on uveitis severity, then tapered to q48h after maximal pupil dilation. Ointment or solution may be used for dogs. To prevent significant salivation or vomiting caused by bitter drops draining to the nose, only ointment should be used for cats. Do not use atropine in patients with inappropriate normal IOP (10 to 20 mm Hg) in an inflamed eye or with overt secondary glaucoma, as pupil dilation will further crowd the iridocorneal drainage angle to decrease aqueous humor outflow and worsen the glaucoma. Avoid or use sparingly in patients with keratoconjunctivitis sicca because the parasympatholytic effect can decrease tear production.

Tropicamide 1% has a shorter duration of action and is less potent than atropine but can be used when pupil movement is desirable or as a safer drug choice in patients with low-normal but inappropriate IOP. Apply q12h to q6h, but discontinue if the IOP becomes elevated, and do not use in patients with overt secondary glaucoma.

**Systemic Therapy**

Systemic therapy is necessary for most patients with uveitis because topical treatments do not benefit the posterior segment. Corticosteroids are best for immune-mediated disease and posterior uveitis and when intraocular hemorrhage is present. Doses vary based on the suspected cause (TABLE 2). Anti-inflammatory prednisone is typically used unless systemic infectious diseases are ruled out. Immunosuppressive prednisone may be necessary when severe inflammation compromises vision. Depending on the cause and patient’s starting dose, begin drug tapering after 1 to 2 weeks.

In dogs with steroid-responsive retinal detachment, treatment will need to be more protracted. Rule out systemic disease, then initiate immunosuppressive corticosteroids and treat for at least 2 months beyond complete retinal reattachment. The prognosis for vision is good, although recurrence is possible.

NSAIDs are useful in cases of mild anterior uveitis (including reflex uveitis) or when systemic infection is suspected. Options for dogs are numerous; a few examples are included in TABLE 2. Options for cats are more limited. Short-term use of meloxicam, robenacoxib, or ketoprofen could be considered at labeled doses in cats, but be aware of possible renal impact.

### TABLE 2 Systemic Therapy for Patients with Uveitis

<table>
<thead>
<tr>
<th>DRUG</th>
<th>SPECIES</th>
<th>DOSAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CORTICOSTEROIDS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prednisone (anti-inflammatory)</td>
<td>Dogs</td>
<td>0.5–1 mg/kg q24h*</td>
</tr>
<tr>
<td>Prednisone (immunosuppressive therapy)</td>
<td>Dogs</td>
<td>1–2.2 mg/kg PO q24h*</td>
</tr>
<tr>
<td>Prednisolone</td>
<td>Cats, dogs that do not respond to prednisone</td>
<td>0.5–1 mg/kg PO q24h or q12h depending on severity*</td>
</tr>
<tr>
<td><strong>NSAIDs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carprofen</td>
<td>Dogs</td>
<td>2.2 mg/kg PO q12h</td>
</tr>
<tr>
<td>Meloxicam</td>
<td>Dogs</td>
<td>0.2 mg/kg loading dose, then 0.1 mg/kg PO q24h</td>
</tr>
<tr>
<td>Firocoxib</td>
<td>Dogs</td>
<td>5 mg/kg PO q24h</td>
</tr>
</tbody>
</table>

* Doses recommended by author.
Never use systemic NSAIDs in conjunction with systemic corticosteroids, given the risk of gastrointestinal ulceration. Systemic NSAIDs are safe to use with topical corticosteroid and/or topical NSAID formulations. Avoid NSAIDs in patients with intraocular hemorrhage, as they may promote further bleeding.

Exceptions to Standard Therapy
Reflex uveitis due to corneal ulceration or abscessation needs appropriate primary corneal disease treatment including a systemic anti-inflammatory medication; however, topical corticosteroids and topical NSAIDs are contraindicated. Cataract surgery can be considered to address lens-induced uveitis if retinal function is adequate to restore vision. Primary intraocular neoplasia warrants enucleation if metastatic potential exists (e.g., feline post-traumatic ocular sarcoma) or if the mass is extensive and/or causing secondary glaucoma. Ocular lymphosarcoma warrants systemic chemotherapy along with appropriate topical treatments.

FOLLOW-UP
A follow-up examination is recommended 3 to 7 days after starting treatment, then every 1 to 2 weeks after improvement is noted to track status during gradual treatment taper. Signs of ocular discomfort should subside quickly with appropriate treatment. Additional desired findings on clinical examination include normal IOP, appropriate pupil size, clearing ocular media, and resolving color changes (e.g., sclera appears white, iris color returns to normal). Anterior chamber inflammation will clear more readily than vitreal haze due to variable turnover rates. A final examination should take place once the patient is off all medications to ensure no signs of recurrence.

PROGNOSIS
The prognosis for uveitis resolution depends on the cause. Primary ocular causes have greater chance for resolution. Systemic causes increase the need for a prolonged therapeutic course. Recurrence is common with poor client compliance or insufficient treatment dosing/duration (FIGURE 5). In all cases, the goals of management are controlling inflammation, reducing pain, preserving vision, and resolving the underlying condition if identified. **TVP**

References
Managing Uveitis in Dogs and Cats

LEARNING OBJECTIVES
After reading this article, readers will be able to recognize clinical signs and findings that suggest uveitis, list possible causes, consider pertinent diagnostic tests, and plan treatment and patient follow-up.

TOPIC OVERVIEW
This article provides an overview of uveitis in dogs and cats, including clinical signs and findings of intraocular inflammation, causes, diagnostic tests, treatment, and follow-up.

1. Which clinical finding is not consistent with uncomplicated uveitis?
   a. corneal edema
   b. episcleral blood vessel injection
   c. constricted pupil
   d. elevated intraocular pressure

2. Untreated uveitis can lead to
   a. blindness
   b. globe rupture
   c. corneal squamous cell carcinoma
   d. spontaneous resolution

3. The most common metastatic neoplastic cause of uveitis is
   a. lymphosarcoma
   b. mammary adenocarcinoma
   c. pulmonary adenocarcinoma
   d. oral melanoma

4. Which structure is not part of the uveal tract?
   a. choroid
   b. ciliary body
   c. cornea
   d. iris

5. Which dog breed is predisposed to pigmentary uveitis and pigmentary cystic glaucoma?
   a. Akita
   b. Boston terrier
   c. Golden retriever
   d. German shepherd

6. To allow optimal posterior segment evaluation, which agent is recommended for pupil dilation during clinical examination?
   a. atropine
   b. dexamethasone
   c. prednisolone acetate
   d. tropicamide

7. Aqueous flare is best visualized in the
   a. cornea with the examination room lights on
   b. anterior chamber with the examination room lights off
   c. vitreous after pupil dilation
   d. retina adjacent to blood vessels

8. Which cytologic sampling procedure should be performed in a uveitis patient with anorexia, lethargy, and enlarged peripheral lymph nodes?
   a. aqueous humor sampling
   b. peripheral lymph node aspiration
   c. vitreal aspiration
   d. subretinal aspiration

9. Which of the following treatment scenarios is appropriate?
   a. concurrent oral corticosteroid and oral NSAID therapy in a dog with uveitis
   b. use of atropine in a cat’s uveitic eye with an IOP of 25 mm Hg
   c. topical prednisolone acetate and oral NSAID therapy in a dog with anterior uveitis
   d. topical corticosteroid therapy in an eye with reflex uveitis due to corneal ulceration

10. To help prevent lens-induced uveitis, a diabetic miniature schnauzer with cataracts should be started on an(n) _____ twice daily.
    a. topical NSAID
    b. oral tetracycline antibiotic
    c. topical atropine
    d. oral corticosteroid

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