Feline uveitis: A review of its causes, diagnosis, and treatment

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Uveitis is a common and painful ocular disease in cats that can eventually lead to blindness. Uveitis often occurs secondary to an acquired ocular or systemic disorder; however, in many instances the underlying cause is not identified despite extensive diagnostic testing. As such, it presents a great challenge for practitioners with respect to diagnosis and treatment. In this article, we review the pathophysiology, clinical presentation, causes, and treatment of uveitis in cats.

ANATOMY AND PATHOPHYSIOLOGY

The uvea, or vascular tunic of the eye, is composed of the iris and ciliary body anteriorly and the choroid posteriorly. The iris divides the anterior ocular compartment into anterior and posterior chambers and controls the quantity of light entering the posterior segment through the pupil. The ciliary body provides nourishment for and removes wastes from the cornea and lens via the production of aqueous humor. The choroid is the main source of blood and nutrition for the outer layers of the immediately adjacent retina.

Uveitis is defined as any condition that involves uveal tract inflammation. Damage to the uveal tissue or vasculature causes the release of tissue factors and chemoattractants, resulting in vasodilation and changes in vascular permeability. These changes lead to a breakdown of the blood-aqueous barrier or the blood-retinal barrier, allowing protein and cellular accumulation.

Classifying uveitis can help you determine what diagnostic steps will be required when presented with a cat that has uveitis. Anatomically, uveitis is classified as anterior if the iris and ciliary body are inflamed (iridocyclitis). Inflammation of the choroid is termed posterior uveitis or choroiditis. A full ophthalmic examination is required to determine which portions of the uveal tract are involved. Additionally, uveitis can be classified etiologically as being related to an underlying ocular disorder or secondary to a systemic disease process. Ocular disorders are typically unilateral and readily identified on complete ophthalmic examination. Systemic causes of uveitis often result in bilateral ocular involvement.

OPHTHALMIC EXAMINATION AND FINDINGS

A thorough ophthalmic examination is required to diagnose uveitis. Perform fluorescein staining to rule out the presence of a corneal ulcer, and measure intraocular pressure by applanation tonometry after applying topical proparacaine.
An anterior segment evaluation is best performed by using a hand-held slit lamp. Alternatively, the anterior chamber can be evaluated with a direct ophthalmoscope by using either the small spot or slit setting. Place the direct ophthalmoscope close to the eye, and focus it on the cornea. View the eye perpendicular to the light source to evaluate the anterior chamber for flare, cellular accumulation, or changes of the iris face. To evaluate for opacities of the aqueous, lens, or vitreous, you may also use retroillumination. To do so, stand at an arm's length from the patient, and obtain a tapetal reflex with a hand-held transilluminator. Opacities will block or diminish the tapetal reflex.

Perform the fundic examination by using direct or indirect ophthalmoscopy. Indirect ophthalmoscopy requires a focal light source (Finoff transilluminator) and a hand-held lens. The most commonly used hand-held lenses are 20 or 28 diopters. The larger-number diopter lenses make it easier to visualize the fundus when the pupil is small. The image obtained by indirect ophthalmoscopy is less magnified than with direct ophthalmoscopy, but the larger field of view obtained makes it a better method of screening eyes for posterior segment involvement.

Table 1: Possible Ocular Signs and Complications in Cats with Uveitis

Clinical signs associated with uveitis in cats can vary widely and may not be as prominent as signs seen in other species (Table 1). Pain is often associated with acute uveitis and is observed clinically as blepharospasm, photophobia, enophthalmos, third eyelid elevation, or epiphora.2,3

**Anterior uveitis**

Aqueous flare, which is diagnostic of anterior uveitis (Figure 1), occurs secondary to the breakdown of the blood-aqueous barrier and increase in aqueous humor protein concentration.2,3 Purulent material or blood may also accumulate in the anterior chamber, resulting in hypopyon or hyphema, respectively (Figure 2).2

Figure 1. A slit lamp photo of a patient with anterior uveitis and resultant aqueous flare (arrowheads). Breakdown of the blood-aqueous barrier results in increased protein concentration of the aqueous humor, and the resultant turbidity (scattering of light) can be seen on oblique illumination of the anterior chamber. (Photo courtesy of Dr. Ellison Bentley.)

Inflammation of the corneal endothelium impairs the chloride-dependent active pump mechanism responsible for transporting fluid out of the stroma and results in corneal edema.4 Inflammatory cells within the aqueous humor may aggregate and deposit upon the corneal endothelium, resulting in keratic precipitates that are typically present on the ventral half of the cornea (Figure 3).2,3 Additionally, corneal vascularization can occur secondary to chronic inflammation.2
Intraocular pressure is often decreased in patients with uveitis because of prostaglandin-mediated inflammation and edema of the ciliary body resulting in impaired aqueous humor formation or increased aqueous humor outflow through the uveoscleral route.

Changes in the iris can also occur with anterior uveitis. In response to the effects of prostaglandins and other inflammatory mediators on the iris sphincter muscle, miosis may be observed. The miosis is typically associated with ciliary muscle spasm, which contributes to ocular pain. Iridal swelling or iritis results from vasodilation, increased iris vessel permeability, and cellular accumulation, which often causes a change in iris color. Cellular accumulations may also give the iris a grossly swollen appearance.

In patients with chronic uveitis and resulting miosis and iridal swelling, the pupil margin may adhere to the anterior lens capsule, resulting in posterior synechiae. In these cases, the pupil margin appears irregular and the pupil fails to respond appropriately to light and dilating agents. If the entire pupil margin is involved, anterior movement of the aqueous humor is inhibited, resulting in aqueous humor accumulation behind the iris. This accumulation appears clinically as iris bombé, or an anterior ballooning of the iris, which predisposes the eye to secondary glaucoma development. Additionally, in patients with chronic inflammation, the iris may appear red, also known as rubeosis iridis, secondary to neovascularization of the anterior iridal surface.
**Posterior uveitis**

Pupil dilation with tropicamide ophthalmic solution is required to evaluate the posterior segment. Upon dilation, it is possible to detect inflammatory cells in the anterior vitreous, known as *pars planitis*. This cellular accumulation occurs secondary to the infiltration of cells from the adjacent pars plana or pars plicata of the ciliary body.²,³ Dilated examination also allows for the detection of posterior uveitis. Posterior uveitis is often accompanied by retinal inflammation because of the close anatomical position of the structures.²,³ The breakdown of the blood-ocular barrier located at the retinal blood vessels and the retinal pigment epithelium allows inflammatory cells to migrate to the area and results in chorioretinitis.³ Clinically, edema, exudation, and hemorrhage within the vitreous, retina, and subretinal space may be observed.²,³

Because of the location of the retina and subretinal space over the tapetum, tapetal reflectivity may be diminished or appear gray (*Figure 6*).²,³ Retinal detachments may also occur secondary to severe inflammation.² Retinal detachments develop when the neurosensory retina separates from the underlying retinal pigmented epithelium. Detachments typically occur secondary to the accumulation of blood or exudates between these two layers. Further evaluation of the fundus may reveal vascular tortuosity, hemorrhage, or sheathing of retinal vessels by inflammatory cells, known as *perivascular cuffing*.²

**Diagnosis**

In cats with bilateral uveitis, a thorough medical history, physical
examination, complete blood count, serum chemistry profile, and urinalysis are necessary because of the potential for an underlying systemic disorder. Important historical information to obtain relates to the patient's environment (indoors vs. outdoors), use of flea preventives, travel history, history of trauma, duration of clinical signs, and the presence of any clinical signs often associated with systemic illness, such as inappetence and lethargy.

On physical examination, rectal temperature and mucous membrane color should be evaluated, and the cat should be examined for ectoparasites, ocular or nasal discharge, and lymphadenopathy in addition to undergoing a thorough thoracic auscultation and abdominal palpation. Additionally, serologic tests are available for many of the infectious disease processes that cause uveitis (see below). Diagnostic testing is also recommended in cases of unilateral uveitis for which a primary ocular cause cannot be identified, as systemic diseases may not always manifest with bilateral ocular signs.

Further diagnostic tests may be required if a diagnosis is not made with routine testing. Additional testing modalities are available on aqueous humor samples, including PCR tests for various infectious agents, cytologic examination, and bacterial culture and antimicrobial sensitivity testing. Samples are collected by aqueous humor paracentesis, which is performed under general anesthesia. Vitreous humor can also be sampled when other diagnostic test results are unrewarding, but there is a high risk of ocular hemorrhage and lens or retinal damage. Thus, this procedure is typically limited to patients that are blind or nearly blind. Procedures to acquire aqueous and vitreous humor can carry serious complications since structures within the eye may be inadvertently damaged. Thus, it is recommended that patients requiring such diagnostic tests be referred to an ophthalmologist.

TREATMENT

Uveitis in cats results from many ocular disorders or systemic diseases. Nonspecific therapy of uveitis is needed to minimize ocular inflammation, reduce pain, and prevent complications (see sidebar titled "Nonspecific therapy for uveitis"). Specific therapy is directed at the underlying ocular or systemic cause of uveitis.

OCULAR DISORDERS CAUSING UVEITIS

Uveitis can result from a variety of ocular disorders.

Corneal ulceration or trauma

Any event affecting the cornea, such as perforation or ulceration, can result in uveitis through the stimulation of an axonal reflex that is mediated by trigeminal nerve endings. Both blunt and penetrating trauma may result in uveitis. Penetrating ocular trauma may result in lens capsule rupture. Release of lens proteins causes severe inflammation (phacoclastic uveitis) of delayed onset after the traumatic event. With tears in the lens capsule > 1.5 mm, loss of the eye will often occur without early lens extraction. Additionally, penetrating trauma may result in the introduction of bacterial or fungal contaminants. Treatment with broad-spectrum oral antibiotics is recommended to reduce the risk of infectious endophthalmitis.

Neoplasia

Primary ocular neoplasms, iris melanoma being the most common, do not directly induce uveitis but instead mimic uveitis by producing such changes as tissue necrosis, hemorrhage, and edema.
Changes at tissue necrosis, hemorrhage, and glaucoma a diffuse iridal 

melanoma is a progressive neoplasm (developing over months to years) 

that presents as increased pigmentation of the anterior iridal surface 

(Figure 7). Metastasis has been reported in as many as 63% of cases. 

Affected cats should be monitored for the degree of iris and iridocorneal angle involvement, changes in pupillary shape, and the development of increased intraocular pressure as enucleation may be required. 

Figure 7. Chronic secondary glaucoma of the left eye resulting in anisocoria (arrowheads indicate pupil margins) and buphthalmia. Buphthalmia is demonstrated in the left eye by the increased width of the palpebral fissure and increased corneal diameter compared with the right eye. Note the diffuse, dark-brown color of the iris. Diffuse iridal melanoma was diagnosed on histologic examination. 

Trauma-associated sarcomas are primary ocular tumors that may present clinically with chronic uveitis, glaucoma, intraocular hemorrhage, or white to pink masses. These neoplasms are typically detected an average of five years after a traumatic ocular event and are highly malignant. An association with lens capsule rupture and the development of these tumors has been reported. 

Other neoplasms seen in cats include primary ciliary body adenomas and adenocarcinomas, but these neoplasms are rare. These nonpigmented tumors are often identified as focal growths originating from the ciliary body on dilated examination or on the basis of ocular ultrasonography. These primary ocular tumors must be differentiated from metastatic tumors such as lymphosarcoma, hemangiosarcoma, and adenocarcinoma. 

Enucleation or exenteration is the treatment of choice for feline ocular neoplasms, and evaluation for metastasis should occur not only at the time of diagnosis but in the years after enucleation or exenteration. 

Idiopathic or immune-mediated 

Despite a complete ophthalmic examination and systemic work-up, the cause of a patient's uveitis may not be identified. In some cases, histologic evaluation has demonstrated uveal lymphocytic-plasmacytic cellular infiltrates. This finding suggests that a proportion of feline uveitis cases may be secondary to an immune-mediated process. 

SYSTEMIC DISORDERS CAUSING UVEITIS 

Bacterial diseases 

Bartonella henselae is a fastidious gram-negative bacterium that infects a cat's endothelial cells and erythrocytes. Fleas are the principal vector for the organism, with flea feces serving as the most likely infectious substrate, which is inoculated through contaminated cat claws. Asymptomatic bacteremia can be prolonged, extending from weeks to months. It remains uncertain whether B. henselae is an etiologic agent of feline uveitis. Antibodies against the organism have been documented in the serum of cats exhibiting uveitis and healthy cats. In one report, the seroprevalence of B. henselae was higher in both healthy cats and cats without ocular disease compared with cats with
Healthy cats and cats without ocular disease compared with cats with uveitis. Serology is thus unlikely to aid in diagnosis.

It has been postulated that diagnosis could be achieved by culturing the organism from or performing special staining techniques on ocular tissue samples; however, *B. henselae* is difficult to culture, and current staining techniques are obscured by ocular pigmentation. Detection of the organism in aqueous humor by PCR testing has yielded positive results, but these results should be interpreted cautiously as the organism may be introduced into the sample secondary to hyphema or hemorrhage induced by anterior chamber paracentesis.

It is recommended that *B. henselae* infection be diagnosed based on eliminating other causes of uveitis as well as a positive antibody titer, a positive response to therapy, and a decrease in antibody titer after therapy.

Systemic therapy, in conjunction with nonspecific uveitis therapy (see sidebar titled "Nonspecific therapy for uveitis") involves long-term administration of azithromycin (10 mg/kg orally once daily for 21 days), doxycycline (10 mg/kg orally followed by water twice daily for six weeks), or rifampin (10 mg/kg orally once daily for 21 days).

**Viral diseases**

Four viral diseases in cats have been associated with uveitis—feline leukemia virus (FeLV) infection, feline immunodeficiency virus (FIV) infection, feline infectious peritonitis (FIP), and feline herpesvirus-1 (FHV-1) infection.

**FeLV and lymphosarcoma.** The retrovirus FeLV is transmitted both horizontally and vertically among cat populations. Two disease progressions are possible in cats infected with FeLV: 1) persistent viremia and progressive infection or 2) self-limiting, regressive infection. Numerous FeLV strains exist, some of which can lead to malignant transformation or cytopathic deletion of specific lymphocyte and hematopoietic cell populations.

A low incidence (< 2%) of ocular disease has been reported among cats infected with FeLV. Ocular lesions in cats with FeLV infection are unlikely to be the direct result of FeLV infection but rather neoplasia induced by the virus or related to secondary invasion of infectious agents caused by immunosuppression. The most common neoplastic disease secondary to FeLV infection is lymphosarcoma, which is a significant cause of uveitis in cats. Clinically, lymphosarcoma manifests as iridal thickening with associated flesh-colored lesions (Figure 8). These lesions are most commonly nodular but may be diffuse, with diffuse lesions appearing similar to uveitis secondary to other causes. Other ocular findings may include pink vascular corneal masses, hyphema, orbital disease, retinal degeneration, and hemorrhage.
The most commonly used FeLV testing method is a peripheral blood ELISA that tests for the presence of the p27 antigen. As previously discussed, cats can develop a self-limiting regressive infection, so a positive ELISA result should be confirmed with an immunofluorescent antibody test or a second ELISA performed three to four months after the first test. A second positive test result is highly suggestive that the patient is persistently infected. Identifying neoplastic lymphocytes on histologic examination of mass lesions or cytologic examination of aqueous humor is diagnostic of ocular lymphosarcoma; however, such samples may be unrewarding, and detection of neoplasia in other body systems may be necessary.

With regard to ocular lesions, a positive FeLV status should be evaluated with caution as not all cats infected with FeLV develop lymphosarcoma, and, as previously discussed, FeLV infection may result in uveitis secondary to other infectious diseases as a result of immunosuppression. Address ocular lesions with nonspecific therapy (see sidebar titled “Nonspecific therapy for uveitis”). In addition, systemic antivirals have been investigated and are thought to decrease the antigenemia in infected cats. These therapies, however, are associated with significant side effects, so the mainstay of systemic therapy remains good husbandry and supportive care. Patients with ocular lymphosarcoma should be treated with systemic chemotherapy agents since the disease manifestation is often associated with multicentric lymphosarcoma.

FIV. This lentivirus causes an acquired immunodeficiency syndrome in cats. Many modes of transmission are thought to be possible, including in utero and postpartum through milk; however, bite wounds are thought to be the predominant cause of viral inoculation. In patients infected with FIV, there is a progressive depletion of the CD4+ helper T lymphocyte population with a coinciding decrease in CD8+ T cells late in the disease process. Feline acquired immunodeficiency syndrome can occur months to years after primary infection with FIV and is associated with severe secondary infections, neoplastic diseases, and neurologic disorders. Ocular lesions seen in patients infected with FIV may include pars planitis, glaucoma, and chronic conjunctivitis, but anterior uveitis is the most frequent clinical finding. Ocular inflammation is thought to occur either directly in response to a cytopathic effect of the virus or secondary to immune stimulation by viral antigens in ocular tissue. Uveitis may also occur secondary to immunodeficiency and associated opportunistic infections with organisms such as Toxoplasma gondii.

Because FIV induces a persistent infection, a definitive diagnosis is most commonly achieved by detecting FIV-specific antibodies in blood through either an ELISA or rapid immunomigration-type assay. False positive results can be seen in cats that have received the FIV vaccine or in kittens < 12 weeks of age that have passively acquired anti-FIV antibodies from an infected or vaccinated mother. False negative results may also be observed in the acute phase of infection when the antibody response is undetectable. Treatment of ocular lesions should include nonspecific...
therapy of uveitis (see sidebar titled "Nonspecific therapy for uveitis"). Current systemic therapy is centered on good husbandry and supportive care, but antiviral chemotherapy and immune modulatory therapy are under investigation.21

**FIP.** FIP is caused by a highly fatal coronavirus that is shed mainly in feces and transmitted by ingesting or inhaling viral particles.25,26 Numerous feline coronaviruses exist, and it has been proposed that the FIP virus occurs secondary to spontaneous mutation of feline enteric coronavirus within an environment.26 Whether a cat develops clinical disease after viral exposure depends on the animal’s immune response. Vasculitis is a hallmark of the disease and occurs secondary to the activation and circulation of monocytes.27 The clinical syndrome in cats occurs as either an effusive or noneffusive form; the noneffusive form is most commonly associated with ocular lesions.25 Such lesions may be present in either the anterior or posterior segment of the eye and may include iritis, keratic precipitates, fibrin within the anterior chamber, hyphema, chorioretinitis, and retinal perivascular cuffing.25

Diagnosing FIP is difficult since no reliable diagnostic testing methods are available at this time. Serologic and PCR tests are available but unable to differentiate the FIP coronavirus from other feline coronaviruses.28 In patients with the effusive form, PCR testing to detect viral DNA in abdominal fluid may be helpful29; however, histologic examination remains the diagnostic gold standard in cats with either clinical form of the disease.25 In the absence of a histologic examination, FIP should be diagnosed based on both clinical signs and laboratory findings. Common abnormal serum chemistry profile findings include severely elevated serum globulin concentrations, elevated hepatic enzyme activity, and increased BUN and creatinine concentrations.25 Common clinical findings in addition to ocular lesions include ascites, thoracic or pericardial effusion, icterus, diarrhea, and neurologic signs.25 Although ocular lesions can be addressed with nonspecific therapy (see sidebar titled "Nonspecific therapy for uveitis"), systemic treatment is rarely successful but may include immunosuppressive and anti-inflammatory agents and feline interferon-omega.25

**FHV-1.** This DNA alpha-herpesvirus is widespread among the general cat population.30,31 The virus is shed in ocular, nasal, and oral secretions and transmitted mostly by direct contact, although indirect transmission can occur.30 After infection, about 80%31 of cats become latently infected carriers. Of these, about 45% of cats can have virus reactivation, resulting in clinical disease or asymptomatic episodes of viral shedding spontaneously or after periods of stress.30,31

The most common clinical ocular manifestations of FHV-1 are conjunctivitis and keratitis, but anterior uveitis has also been a suggested manifestation of the disease.32 One study demonstrated FHV-1 DNA in the aqueous humor of 12 of 86 cats with clinical signs of anterior uveitis that had negative test results for other known causes of feline uveitis.32 This study proposed that FHV-1 gained entry into the eye through axonal transport of virus, but this hypothesis has not been investigated.32 As previously discussed, FHV-1 may reactivate in times of stress, so it remains unclear whether the intraocular FHV-1 infection is a cause or result of feline uveitis.31 Additionally, FHV-1 can replicate in conjunctival and corneal tissue and could serve as a contaminant during anterior chamber paracentesis.

Acute ocular FHV-1 infection can typically be diagnosed with virus
isolation from conjunctival cytology samples. Other diagnostic tests available include serology and serum neutralizing antibody titers, fluorescent antibody testing on corneal or conjunctival smears, and PCR tests. Serologic tests are of limited value since most cats have been exposed to or vaccinated against FHV-1.

In addition to nonspecific therapy (see sidebar titled “Nonspecific therapy for uveitis”), topical antiviral medications, including trifluridine and idoxuridine solutions, are efficacious against FHV-1 conjunctivitis and keratitis when administered as one drop in the affected eye four to six times a day for two to three weeks. As most antiviral agents are virostatic, frequent application is needed. Combined with local irritation, these factors often result in poor owner compliance.

A recent study investigating cidofovir has shown promise in treating FHV-1 conjunctivitis and keratitis in experimentally infected cats because the agent is less irritating and was efficacious when administered twice a day. Famciclovir, an oral antiviral drug, effectively reduces the severity of systemic and ocular clinical signs in cats; however, dosing regimens remain varied and dosing recommendations are uncertain, ranging from 62.5 mg/cat once to twice a day to 125 mg/cat three times a day. Treatment with oral L-lysine (250 to 500 mg once or twice a day) has also effectively reduced the severity of conjunctivitis and decreased viral replication in cats with FHV-1 infection by serving as an arginine inhibitor and an arginase inducer.

**Mycotic diseases**

Cryptococcosis, blastomycosis, histoplasmosis, and coccidioidomycosis have all been associated with feline uveitis.

**Cryptococcosis.** Cryptococcosis is one of the most common mycotic diseases in cats. Basidiospores of *Cryptococcus neoformans* cause the disease; the mode of transmission is thought to be inhalation. Pigeon droppings serve as the principal reservoir for the yeast. Affected cats typically develop respiratory and cutaneous manifestations, but they may also exhibit neurologic signs secondary to direct extension of the organism through the cribriform plate. Ocular signs unrelated to neurologic disease are thought to occur secondary to hematogenous dissemination of the organism and include both anterior uveitis and chorioretinitis. Chorioretinal lesions vary in appearance from single to multifocal and pinpoint opacities to large circular lesions.

**Blastomycosis.** Blastomycosis is caused by *Blastomyces dermatitidis*, a dimorphic fungus often isolated from bat and pigeon feces. Infection occurs primarily by inhaling aerosols containing infective spores. Although the disease appears to be most prevalent in areas around water, such as the Mississippi, Missouri, and Ohio river valleys, it has been observed in solely indoor cats in dry geographic regions. Most affected cats present with systemic signs, including anorexia, lethargy, weight loss, and respiratory impairment. Central nervous system signs and cutaneous lesions may also be seen in a few cases. Ocular lesions may include severe aqueous flare, posterior synechiae, keratic precipitates, rubeosis iridis, severe retinal detachments with subretinal pyogranulomas, and intraocular pyogranulomas. Lesions described as pyogranulomatous chorioretinitis have also been reported in the posterior segment of the eye.

**Histoplasmosis.** Histoplasmosis is caused by *Histoplasma capsulatum*, a dimorphic fungus most commonly found in bat and bird feces.
Dimorphic fungus most commonly found in bat and bird feces.37,41 Infection occurs primarily by inhaling aerosols containing infective spores.37,41 Most affected cats present with vague systemic signs, including anorexia, lethargy, weight loss, fever, and anemia.37 Infrequent findings include pulmonary involvement, skeletal infection, and cutaneous lesions.37 Ocular involvement is thought to be more common with disseminated histoplasmosis than with other feline systemic mycoses.37 Ocular lesions may include mucoid ocular discharge, blepharospasm, conjunctivitis, granulomatous blepharitis, endophthalmitis, chemosis, anterior uveitis, chorioretinitis, retinal detachment, and secondary glaucoma.37,41,42

**Coccidioidomycosis.** Coccidioidomycosis is caused by *Coccidioides immitis*, a dimorphic fungus found in soil in the southwestern United States.37,43,44 Transmission occurs primarily by inhaling aerosols containing infective spores but can also occur after direct inoculation of the organism into the skin.37,43 Clinical signs typically include draining skin lesions, abscesses, subcutaneous granulomatous masses, and regional lymphadenopathy.37,43 Ocular lesions may include fibrinopurulent exudates in the anterior, posterior, and vitreous chambers; pyogranulomatous endophthalmitis; diffuse granulomatous chorioretinitis44; and retinal detachment.37

**Diagnosis and treatment.** The aforementioned mycotic diseases can be diagnosed based on clinical findings, the results of serologic testing, and demonstration of an organism by cytology or histology.37 In cats with ocular lesions, aqueous aspirates are rarely rewarding, but histologic examination of an enucleated eye37,39 or vitreous or subretinal aspirates37 is likely to demonstrate organisms.

In addition to nonspecific topical therapy for uveitis (see sidebar titled "Nonspecific therapy for uveitis"), azole antifungal therapy with or without adjunctive amphotericin B therapy has been effective.37,43,45 Fluconazole is the azole of choice in cats since it is associated with the fewest side effects7 and has good penetration into the eye. It is recommended in cases of cryptococcosis and coccidioidomycosis at doses of 25 to 50 mg/cat orally every 12 hours or 5 to 15 mg/kg orally every 12 to 24 hours.2,37 Histoplasmosis and blastomycosis have been effectively treated with itraconazole administered orally at 5 mg/kg every 12 hours, but reversible hepatotoxicosis can occur.4,37

Continue antifungal therapies with azoles for one month after clinical signs resolve.37 Therapy is typically long-term and may last six months or longer.37 Cats with severe disease should be adjunctively treated with parenteral amphotericin B.37 This medication is typically reserved for severely affected patients since it can be nephrotoxic. As such, blood urea nitrogen and serum creatinine concentration monitoring is recommended.37

**Protozoal diseases**

*Toxoplasma gondii* is an obligate intracellular coccidian parasite.46 Cats, the definitive host, acquire toxoplasmosis by ingesting *T. gondii* cysts in prey animals.46 Systemic signs of infection include vague clinical signs, such as lethargy, anorexia, weight loss, and weakness; short-lived signs, such as a self-limiting, small-bowel diarrhea; and more severe signs, such as ataxia, seizures, icterus, abdominal effusion, and cardiac arrhythmias.46 The seroprevalence of *T. gondii* infection in cats with uveitis has been reported as high as 80.2%.47 Both the organism’s DNA and antibodies to the organism have been detected in aqueous humor, confirming that *T. gondii* can directly infect the eye.47,48 Intracocular
confirming that *T. gondii* can directly infect the eye. Intraocular inflammation is thought to occur secondary to organism replication or intraocular hypersensitivity induced by exposure of antigen-specific intraocular lymphocytes to circulating *T. gondii* antigens. In addition to anterior uveitis, *T. gondii* can cause chorioretinitis and retinal vasculitis.

Several diagnostic methods are available, including serology, fecal examination, aqueous humor PCR testing, and aqueous humor antibody detection. But the only means to definitively diagnose the disease is to demonstrate the organism on ocular histologic examination. The latter may prove difficult as the organism has rarely been identified. In addition to nonspecific therapy for uveitis (see sidebar titled “Nonspecific therapy for uveitis”), cats infected with *T. gondii* should be given clindamycin hydrochloride at a dose of 12.5 mg/kg orally twice a day for two to three weeks. Clindamycin slowing the replication rate of the organism but is unlikely to clear it from the body.

**COMPLICATIONS**

Uveitis can lead to secondary glaucoma because aqueous humor flow through the pupil or out of the iridocorneal angle becomes impaired. Secondary glaucoma has been reported to occur in up to 50% of cats with uveitis secondary to systemic disease. Secondary glaucoma should be suspected in any eye with uveitis that has relatively normal intraocular pressure readings. Treat secondary glaucoma with carbonic anhydrase inhibitors and beta-blockers to decrease aqueous humor production.

**CONCLUSION**

Feline uveitis can occur secondary to ocular disorders or can be a manifestation of a systemic disease. The disease processes that can lead to uveitis, although discussed individually in this article, can occur concurrently in a patient. In many cases, ocular lesions are the first and only clinical signs of systemic disease. A thorough history and physical
and ophthalmic examinations are necessary to obtain a diagnosis. Early diagnosis and treatment can help preserve a cat's vision.

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