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Ocular Manifestations of Systemic Disease in Cats

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Systemic feline diseases (parasitic, bacterial, fungal, viral, neoplastic, metabolic, vascular, and immune-mediated) are often associated with ocular symptoms. An ocular examination is an important diagnostic tool and should be part of any physical examination. Conversely, cats afflicted with systemic disease require periodic ocular examinations for prognostic information and to prevent vision threatening complications. Typical feline systemic diseases encountered by the practicing clinician are presented with their commonly associated ocular signs.

This paper is intended to review the ocular manifestations of systemic (parasitic, bacterial, fungal, viral, neoplastic, metabolic, vascular, and immune-mediated) diseases in the feline. The reader is encouraged to read other comprehensive reviews of these diseases.

Parasitic Disease

Toxoplasmosis is caused by the coccidial protozoa, *Toxoplasma gondii*, in which the feline is the definitive host. Exposure to *T. gondii* is congenital, via ingestion of sporulated oocysts (sporozytes) within cat feces, or via ingestion of tissue cysts (bradyzoites) within infected carrion. In primary toxoplasmosis, tachyzoites disseminate and replicate within the central nervous system, skeletal muscles, visceral organs, and the eye. Chorioretinitis is the most common ocular manifestation. Multifocal gray (hyporeflective) lesions in the tapetal fundus and fluffy white gray lesions in the nontapetal fundus are typically found. Other ocular manifestations include optic neuritis and anterior uveitis. Cellular necrosis of ocular tissue may result from intracellular growth of *T. gondii*. The role of toxoplasmosis in feline anterior uveitis is not completely understood.

The parasitic larval stages of dipterous flies (*Cuterebra*) have also been found within the feline orbit (ophthalmomyiasis externa) and globe (ophthalmomyiasis interna). Ocular abnormalities include uveitis, corneal edema, multiple curvilinear tracks within the tapetal and nontapetal fundi, retinal detachment, intraocular hemorrhage, and low grade chorioretinitis. *Cuterebra* larvae can also directly penetrate feline sclera causing severe uveitis, fibrin clot formation, and blindness. Other neurological signs and blindness can be caused by intracranial migration of these larvae.

Feline demodicosis is caused by parasitic *Demodex cati* and other unnamed *Demodex* sp. This rare disease affects the feline eyelid, periocular area, head, and neck. Variably pruritic lesions with patchy erythema, crusting, scaling, and alopecia are found. Generalized demodicosis is often found in Siamese and Burmese breeds associated with diabetes mellitus, feline leukemia, systemic lupus erythematosus, hyperadrenocorticism, or feline immunodeficiency.

Bacterial Disease

*Bartonella* are fastidious, flea-transmitted, hemotrophic, small, curved, Gram-negative bacteria. Human "cat scratch fever" is caused by *B. henselae*. Feline infection with *B. henselae* is
extremely common. Although 55 to 81% of cats are seropositive, clinical disease is rarely reported. A solitary case of feline unilateral uveitis associated with intraocular antibodies to *Bartonella* has been reported. This cat’s uveitis was unaffected by topical or oral steroids, but did respond to oral doxycycline.

Mycobacteria are aerobic, nonspore forming, nonmotile, and acid fast-staining bacteria. Generally mycobacteria cause internal tubercular granulomas (tuberculosis), localized cutaneous nodules (leprosy), or spreading subcutaneous inflammation. In the past, choroidal hemorrhages, subretinal granulomas, and retinal detachments were commonly associated with feline tuberculosis caused by *Mycobacterium bovis*. Today’s pasteurization of milk and the culling of *M. bovis* infected cattle have reduced the incidence of feline tuberculosis in the United States. Recently, ocular granulomatous lesions were found in a German cat infected with *M. simiae*. These lesions resolved with antibiotic (enrofloxacin, rifampicin, clarithromycin) therapy.

**Fungal Disease**

Systemic fungal infections are uncommon in cats. The dimorphic saprophytic fungi (*Cryptococcus*, *Histoplasma*, *Blastomyces*, and *Coccidioides*) can act as systemic pathogens. The infective route for these fungi is generally inhalation, but they can also be ingested or inoculated. Fungi can reach the eye hematogenously and then lodge within uveal vessels. It is believed that the fungi must be present within the eye to cause uveitis.

Cryptococcosis occurs worldwide and is the most common systemic fungal infection of cats. *Cryptococcus neoformans* is a saprophytic, round, yeast-like fungus (3.5-7 μm in diameter) with the ability to form large capsules. The fungi are commonly found in pigeon droppings, accounting for the high prevalence of cryptococcosis in urban settings.

The mode of *Cryptococcus* transmission is unclear but may rely on inhalation of aerosolized basidiospores. These spores can then erode the cribriform plate resulting in meningitis. Involvement of the optic nerves results in cryptococcal optic neuritis. Clinical signs of optic neuritis include blindness, or visual problems with either slow pupillary light responses or fixed dilated pupils. Cats with optic neuritis have inflamed hyperemic swollen optic nerve heads. The swollen nerves can be associated with increased tortuosity and congestion of the retinal vessels, peripapillary hemorrhages, and/or peripapillary retinal detachments.

The most frequent ocular manifestation of cryptococcosis is chorioretinitis. *Cryptoccus* spores spread to the eye from the nasal cavity via hemological dissemination. The most common ocular lesions are multiple, small, irregular, gray to black, punctate lesions surrounded by an abnormal fundus (Fig. 2). In severe cases, these fundic lesions can progress to retinal detachment. Anterior uveitis, secondary to lesions of the posterior segment, can also occur. Rarer ocular disorders include exophthalmos secondary to retrobulbar abscessation, Horner’s syndrome secondary to an aural mass, conjunctival swelling and thickening (or protrusion) of the nictitating membranes, and ocular discharge.

Histoplasmosis is the second most common systemic fungal infection of cats. The etiological agent of American histoplasmosis is the soil-borne, dimorphic fungus *Histoplasma capsulatum*. Most clinical cases occur in the Ohio, Missouri, and Mississippi river valleys and Texas. The most common ocular manifestation of feline systemic histoplasmosis is granulomatous chorioretinitis. Other ocular associations include discharge, blepharospasm, conjunctivitis, chemosis, retinal detachment, and secondary glaucoma. Conjunctival, mucus membrane, and skin granulomas may also develop.

Blastomycosis is caused by the dimorphic fungus *Blastomyces dermatitidis*. It is found in the Ohio, Missouri, and Mississippi river valleys, as well as Virginia, the Carolinas, and Georgia. A severe pyogranulomatous uveitis is most
often associated with feline blastomycosis.46-49 Other ocular manifestations include blindness, chorioretinitis, and anterior uveitis.49 Blindness can result from ocular or central nervous system involvement. The posterior segment of the eye is more commonly affected than the anterior segment.49

Coccidiomycosis is found only in the Southwestern United States, especially in Arizona.50 In a retrospective study of 48 cats with systemic coccidiomycosis, 6 cats had ocular lesions.50 The most common ocular manifestation is retinal detachment with concurrent uveitis and iritis. One isolated case of feline coccidiomycosis manifested as an intraocular granuloma with a lens capsule rupture.51

The most common feline fungal skin infection (dermatophytosis) affecting the eyelid is caused by *Microsporum canis*.52 Feline dermatophytosis presents as one or more irregular areas of alopecia with or without scaling.53 Other lesions occur on the head, pinnae, and paws. Young cats (<1 year of age) and longhaired Persians and Himalayans are predisposed to contracting dermatophytosis.52

**Viral Disease**

The feline immunodeficiency virus (FIV) is most likely transmitted by bites between animals.54,55 Feral colonies and roaming tomcats show the highest prevalence of infection.56 The hemological hallmark of FIV infection is a progressive depletion of CD4+ helper T cells, and in advanced stages a loss of CD8+ cells.57

The FIV causes ophthalmic disease through direct viral damage of ocular tissues, by initiating secondary immune phenomena, and/or by promoting opportunistic eye infections.37 The most common ocular manifestations of FIV include anterior uveitis, glaucoma, lens luxation, and pars planitis.38,59 Pars planitis is characterized by white punctate infiltrates in the anterior vitreous. Histologically, FIV-induced anterior uveitis is characterized by diffuse lymphocytic and/or plasmacytic infiltrates.57 Inflammation in response to FIV is more commonly associated with the anterior, rather than the posterior ocular segment.9 Coinfection with FIV also exacerbates ocular manifestations of other pathogens such as *T. gondii*.60

Feline leukemia virus (FeLV) is transmitted primarily through saliva where its concentration surpasses that of serum.62 Viremic cats live and shed virus for several years.62 Infection eventually leads to malignant transformation or cytopathic depletion of specific lymphocytic/hematopoietic cell populations.61

Infection with FeLV can cause infiltrative uveal, conjunctival, orbital, and/or corneal lymphosarcoma.63 Restricted iris motility is associated with invasion of the anterior uvea by this tumor.9 Alternatively, dyscoria or anisocoria can stem from the neurological effects of FeLV on the short ciliary nerves.63,64 In addition, FeLV-induced anemia may lead to secondary retinal hemorrhages.64,65

Feline infectious peritonitis (FIP) is a mutated form of the feline corona virus (FECV).66 This virus systemically replicates within macrophages.67 The resultant viremia deposits virus-laden macrophages within the endothelium of small blood vessels.68 In the absence of a strong T cell-mediated immune response, a profound complement-mediated pyogranulomatous vasculitis (effusive FIP) develops. Limited cell-mediated responses slow FIP replication and granuloma formation (noneffusive FIP).68 Ocular manifestations of FIP are more common in this noneffusive form.35,69

Bilateral granulomatous anterior uveitis, accompanied by chorioretinitis, is commonly associated with FIP infection.73 Virus-laden macrophages damage uveal vasculature allowing the exudation of white blood cells, red blood cells, and/or fibrin into the anterior chamber.69 Frequently large keratic precipitates and a fibrinous exudate are found within the anterior chamber.70 Virally induced vasculitis causes perivascular cuffing of the retinal vessels, retinal detachment, and retinal hemorrhage.71

Feline herpesvirus-1 (FHV-1) primarily replicates within the conjunctival, nasal, and pharyngeal epithelium.72 The virus is cytopathic during its replication, destroying the surface epithelia of the conjunctiva, cornea, and nasal passages.72,73 Primary infection of kittens is associated with upper respiratory and ocular disease.74 Dendritic ulcers of the corneal epithelium are pathognomonic for FHV-1 infection (Fig. 4).
4). Secondary bacterial infections within these ulcers may lead to ocular rupture and blindness. The virus can also damage lacrimal duct epithelium resulting in a permanent decrease in tear production.

It is estimated that 80% of cats will become latently infected with FHV-1 and nearly half will experience spontaneous reactivation and shedding of the virus. Reactivation of FHV-1 occurs sporadically and is associated with conjunctivitis, recurrent corneal ulceration, corneal sequestra, stromal keratitis, eosinophilic keratitis, and ulcerative eosinophilic dermatitis. Herpesvirus is associated with feline uveitis. Both intraocular anti-FHV-1 antibody production and aqueous humor FHV-1 DNA have been found in cases of feline uveitis not associated with other infectious agents.

Neoplastic Disease

Multicentric lymphoma is the most common metastatic tumor of the feline eye, and ocular anomalies can present the first signs of systemic lymphoma. Typical ocular presentation is a nodular iridal mass (Fig. 5). Other manifestations of ocular lymphoma include uveitis with or without glaucoma, and keratitis. Additional feline tumors that are known to metastasize to the eye include fibrosarcoma, squamous cell carcinoma, mammary adenocarcinoma, uterine adenocarcinoma, and adenocarcinomas of undetermined origin.

Pulmonary carcinoma is unusual in its capacity to colonize the vascular endothelium of choroidal retinal arteries. The resultant nonperfused areas of the retina can be viewed ophthalmoscopically. These appear as wedge-shaped tan discolorations of the tapetal fundus, with profoundly attenuated retinal vasculature.

Space-occupying orbital masses are usually manifested as exophthalmia. Feline orbital fibrosarcoma is unusual in causing enophthalmos. The tumor causes pressure necrosis of supportive orbital fat. The most common orbital neoplasia of the cat is squamous cell carcinoma invading from periorcular areas. Other feline orbital neoplasms include zygomatic osteoma, parosteal osteosarcoma, osteosarcoma, fibrosarcoma, undifferentiated sarcoma, and rhabdomyosarcoma.

Brain tumors can present a variety of ophthalmic signs including blindness, abnormal pupillary light reflexes, changes in vestibular ocular reflexes, and irregular ocular alignment. Acute visual loss can present as the sole sign of a tumor within the optic chiasm of the rostral and cranial brain fossa.

Vascular Disease

The correlation between systemic hypertension and retinal lesions caused by choroidal injury is well established in the feline. Causes of feline hypertension include renal disease, hyperthyroidism, diabetes mellitus, heart disease, anemia, hyperadrenocorticism, hyperaldosteronism, pheochromocytoma, and primary hypertension. Retinal, choroidal, and optic nerve blood flow is maintained by alternating vascular resistance within precapillary arterioles. Increased blood pressure can cause a breakdown of this autoregulation. Small focal intraretinal perivascular transudates form at sites of collapse and leakage of retinal arterioles. Over time, these areas coalesce and enlarged regions of intraretinal edema appear. As the hypertensive retinopathy advances, a severe disruption of pigmented retinal epithelium leads to subretinal edema, serous retinal detachment, and hemorrhage.

Other associated ocular changes include a narrowing and increased tortuosity of retinal arterioles, papilledema, and optic nerve atrophy. Systolic blood pressures greater then 160 or 170 mm Hg have been considered diagnostic for hypertension. However, not all cats that have an elevated systolic blood pressure will have signs of hypertensive retinopathy. Monkeys with acute spikes in blood pressure exhibit severe hypertensive retinopathy, whereas those with gradual rises in blood pressure to similar levels do not.

Figure 5 Feline eye with anterior uveal infiltrative lymphosarcoma.

The iris is infiltrated and distorted by two pink to red (hyperemic) nodular masses. These masses extend to fill the anterior chamber. (Image courtesy of Dr. A. Metzler.)

Figure 6 Fundic image of feline hypertensive retinopathy. There is a large subretinal hemorrhage and retinal detachment in the dorsal temporal tapetum. There is a perivascular transudate and increased tortuosity of the retinal vessels. (Image courtesy of Dr. J. Bowersox.)
Ocular manifestations of systemic disease

Aldose reductase is involved in the formation of secondary cataracts when glucose levels are elevated by diabetes mellitus. Diabetic cataracts do not develop in older cats as the level of aldose reductase within their lenses is reduced when compared with that of dogs. Younger cats (<4 years of age) have elevated levels of aldose reductase. Diabetes mellitus in younger cats is rarely described, and therefore, diabetic feline cataracts are uncommon. Secondary cataracts do occur in the feline secondary to hypocalcemia. Kittens with hyperparathyroidism and secondary hyperparathyroidism may develop cataracts.

Hyperthyroidism can cause systemic hypertension followed by secondary hypertensive retinopathy. However, the occurrence of ophthalmic lesions secondary to feline hyperthyroidism is rare. In a retrospective study of 100 hyperviscosity syndromes, 1/300 patients had retinal hemorrhages secondary to immune-mediated anemia or thrombocytopenia. Feline immune-mediated anemia is associated with hemobartonellosis or neonatal isoerythrolysis. Pure red cell aplasia of the bone marrow is linked to FeLV infection. In rare cases of feline thrombocytopenia, hemorrhaging does not even occur with platelet counts as low as 10,000 uL.

Conclusions

Ocular symptoms may be the first sign of a systemic disease. Ophthalmic examination associated with routine physical examination will help diagnose systemic problems. Ophthalmic signs must be considered in relation to history, physical examination, and other clinical symptoms. This will determine appropriate ancillary diagnostic testing.

The most common ophthalmic manifestations of systemic disease encountered by the clinician is uveitis. The reader is referred to excellent reviews of feline uveitis diagnostics for further information, as well as the feline uveitis review in this issue.

An early diagnosis of systemic disease prompted by ophthalmic examination can aid in the preservation of vision and life of the veterinary patient.

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