# Diseases of the Feline Fundus

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Care must be taken not to incorrectly incriminate anterior segment disease (i.e., uveitis, keratitis or immature cataracts) as the cause of blindness when the posterior segment is also involved. If a tapetal reflex can be seen when the pupil is totally dilated, anterior segment disease cannot be the cause of total blindness.

The pupillary light reflex (P.L.R.) should be evaluated in all cases of acute blindness. A P.L.R. should be present even in an eye with a dense cataract or severe keratitis. Total lack of P.L.R. in a normal, visual cat may be due to internal ophthalmoplegia. In most cases, blindness and fixed dilated pupils indicate involvement of the retina and/or optic nerve. But a positive P.L.R. may be present and the blindness due to retinal detachments. This is commonly the case in early retinal detachments associated with hypertension in cats.

A very bright focal beam may also be used to evaluate the dazzle reflex. The bright focal beam will produce a rapid blink and frequently a head jerk. Some individuals may only demonstrate a twitching of the ears in response to the bright light. This reflex requires a functional retina and optic nerve. It is a subcortical reflex with the efferent arm via the facial nerve. This reflex would be positive in an animal with blindness due to a visual cortex lesion.

**Feline generalized retinal atrophy**

Feline generalized retinal atrophy has been seen in young adult cats 3-6 years of age with a history of acute blindness. There is no history of nyctalopia (as in P.R.A.) or other systemic diseases. The P.L.R. is absent or severely depressed and the tapetum has a generalized brilliant hyperreflectivity. Retinal vessels are severely attenuated or absent and there is a lack of hemorrhage, exudate or pigment changes.

Experimental taurine deficient cats will develop feline central retinal degeneration (FCRD) that progresses to a feline generalized retinal atrophy. Cases of FCRD are usually found as a coincidental finding as a bilateral area of hyperreflectivity temporal and slightly superior to the optic disc in the area centralis. Even in severe case where the leision extends horizontally superior to the discs, vision is not clinically affected. Since taurine is added to feline commercial diets, it is difficult to explain spontaneous cases of taurine deficiency. Also, the age of individuals with spontaneous disease is not consistent. Most cases of feline generalized retinal atrophy are young adults whereas cats with FCRD are usually older. Additionally, cases of generalized atrophy have developed without other signs of taurine deficiency. It is possible it is not a problem of dietary deficiency but poor absorption and/or utilization of adequate dietary taurine. Early cases show cone disorganization and dysfunction and advanced cases progress to generalized photoreceptor degeneration.

**Fluoroquinolone toxicity**

In cats, ophthalmologists have reported acute blindness following the administration of oral and injectable enrofloxacin (Baytril® Bayer). The lesions were first described as ‘SARDS-like' in their appearance with tapetal hyperreflectivity and various degrees of vascular attenuation. Clinical signs may appear within days of instituting the antibiotic. Early signs include dilated pupils and rapid vision loss. The mechanism of action is not proven. Studies have shown that the rod photoreceptor is the initial target cell with rapid progression of outer retinal degeneration of the entire nuclear, photoreceptor and retinal pigmented epithelial cell layers. The retinal toxicity is both dose and concentration dependent. Since the recommended dosage of Baytril® has been reduced to 5mg/kg daily, best in divided doses, the incidence of the disease has reduced. Other quinolones have been incriminated but enrofloxacin has the greatest incidence. This is most likely due to the higher recommended dose initially, the popularity of the first drug of its class and the off label IV dosing. Once present, blindness is irreversible.

**Progressive retinal atropphy (P.R.A.)**

The information and incidence of inherited retinal diseases in cats is lacking compared to the dog. This is true not only in the variety of retinal diseases, but also in the variations of the most common disease, progressive retinal atrophy (PRA).

The most common form of PRA in the cat is a rod-cone degeneration inherited as an autosomal recessive gene referred to as rdAc (retinal degeneration in Abyssinian cats). This genotype has been observed in more than 40 cat breeds in North America and Europe. The disease has been studied the most in the Abyssinian. The fundus in affected individuals appears normal until 1.5 - 2 years of age. However, by 8 months of age, affected individuals have reduced visual function as determined by electroretinography and visual testing. The end stage of retinal atrophy, i.e. severe hyperreflectivity and attenuation of vessles, occurs at 2-4 years after the initial ophthalmoscopic signs. There is some variability in individuals and breeds. Some cats show ophthalmoscopic signs at 4–5 months and the end stage at 3-7 years. The breeds in which the rdAc gene has been identified are listed in Table 1.

**Table 1** **Feline Breeds With PRA**

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| Abyssinian/Somali | Tonkinese | Maine coon cat |
| Abyssinian (UK) | American shorthair | Manx |
| Abyssinian (Australia) | Angora | Norwegian forest cat |
| Abyssinian/Somali (Scandinavia) | Birman | Persian |
| American curl | Bobtail | Peterbald |
| American wirehair | Bombay | Ragdoll |
| Bengal | British shorthair | Russian blue |
| Balinese/Javanese | Burmese | Scottish fold |
| Colorpoint shorthair | Chartreux | Selkirk rex |
| Cornish rex | Devon rex | Siamese (‘Appleheads’) |
| Munchkin | Egyptian Mau | Sphynx |
| Ocicat | Exotic | Tennessee rex |
| Oriental shorthair | Havana | Thai |
| Siamese | Himalayan | Turkish angora |
| Singapura | Korat | Turkish van |

A second progressive retinal atrophy seems to be limited to the Abyssinian breed. This is an early onset retinal dystrophy with the symbol Rdy. It is inherited as an autosomal dominant trait with a homozygous dominant being lethal. At 4-5 weeks of age, affected kittens will have slow PLR; at 6 weeks, nystagmus and at 7-8 weeks of age ophthalmoscopic changes are present in the area centralis. At 3 months of age, the fundic leisions are severe and visual impairment occurs by the 4 month of life. Since the area centralis is the location of early changes both ophthalmoscopically and electroretinographically, the condition has been termed a cone-rod dystrophy. Since neither rods or cones reportably never develop normally, some may call this a rod-cone dysplasia.

Two additional retinal diseases have been described, both inherited as an autosomal recessive trait. An early-onset photoreceptor disorder has been identified in the Bengal cat. These affected individuals are blind within the first year of life. In the Persian breed, neither the rods nor cones develop to maturity. Clinical signs are present at 2-3 weeks of age and affected individuals are blind at approximately 16 weeks.

**Hypertensive retinopathy**

Systemic hypertension is seen in both dogs and cats, but the highest incidence is in the geriatric cat. Hypertensive retinopathy is the most common cause of posterior segment blindness in the geriatric cat and possibly even if you consider all cats. Primary or essential hypertension is rare. Most cases are secondary to renal disease, hyperthyroidism, hyperlipidemia, pheochromocytoma, Cushing's syndrome or diabetes mellitus. Renal disease and hyperthyroidism are the two most common etiologies.

The measurement of systemic blood pressure is becoming routine in cats. The reliability of the values is dependent on proper technique and equipment including the right size cuff. For this reason, the definitive diagnosis is best made by the internist who has experience with the instrumentation. The systolic pressure in cats is usually less than 180mmHg. Systolic pressures greater than normal with compatible lesions are considered diagnostic by most internists.

Cardiac disease, especially ventricular hypertrophy, is not uncommon secondary to the hypertension. This should be evaluated and treated since it would contribute to the overall well being of the patient.

The eye may be the first organ to show clinically apparent signs of systemic hypertension. Hypertension should be considered the primary differential in all geriatric cats with any form of intraocular hemorrhage. Most cases are presented to the ophthalmologist due to acute blindness in one or both eyes or for severe unilateral or bilateral anterior uveitis. The primary component of the anterior uveitis is the hyphema. Secondary glaucoma may be caused by solid blood clots occluding the pupil. Severe vitreal hemorrhage may preclude examination of the fundus. Severe retinal detachments and tears are not uncommon. Intraretinal, preretinal, and subretinal hemorrhages are usually present. The disease is frequently asymmetrical but almost always bilateral.

On routine physical examination of geriatric cats, fundus examination may reveal focal retinal hemorrhages and edema, which do not functionally impair vision. These cases should also be evaluated for hypertension.

Hypertensive vasculopathy has been frequently diagnosed in enucleated globes that had not previously been diagnosed as being associated with systemic hypertension. The diagnosis is based in part on the presence of focal retinal edema and destruction. Also common is the variation in caliber of retinal and choroidal vessels. Various arterioles will have a thickened vacular wall and decrease luminal diameter.

Cats with systemic hypertension induced ocular disease require treatment for both the hypertension and the specific ocular disease. Treatment of hypertension must first be directed at the primary etiology. Depending on the etiology, therapy may consist of one or

more of the following: low salt diets; diuretics; beta-blockers, i.e., propranolol (Inderal®), atenalol (Tenormin®); angiotension-converting enzyme inhibitors, i.e., captopril (Capoten®), enalapril (Vasotec®); calcium channel blockers, i.e., diltiazem (Cardizem®); amlodipine besylate (Norvascov®) and vasodilators, i.e., nitroglycerin ointment (Nitrol®), oral nitroglycerin or isosorbide nitrate (Nitrobid®).

In cases with anterior uveitis, topical anti-inflammatory drugs, i.e. corticosteroids and antiprostaglandins, are indicated. Dexamethasone or prednisolone acetate is the corticosteroids of choice. Topical antiprostaglandins or nonsteroidal anti-inflammatory drugs are available as flurbiprofen (Ocufen® Allergan and generic ) and diclofenac (Voltaren® Ciba Vision). In cases of severe miosis where pupillary blockage is threatened, topical atropine ointment is also indicated.

Secondary glaucoma is a severe complication usually caused by extensive hyphema and blood clots occluding the pupil. Treating as an anterior uveitis may resolve the problem. In several cases, tissue plasminogen activator (TPA) has been injected into the anterior chamber to dissolve large clots that block the outflow of aqueous. Unfortunately, enucleation has been necessary in non-responsive cases.

Systemic prednisolone has been used in cases of severe retinal involvement. If not contraindicated by pre-existing disease, the anti-inflammatory action helps reduce the retinal edema and subretinal transudate associated with the retinal detachment.

Improvement of vision and ocular signs is seen following control of the hypertension but may take up to six to eight weeks. Vitreal hemorrhage clears very slowly. Some patients may regain vision despite the fact the blood pressure does not drop to the preferred level. Other cats may not regain vision due to severe retinal involvement that results in total retinal atrophy. These animals do well in spite of blindness if their systemic disease is controlled.

**Chorioretinitis**

The etiology of chorioretinitis may be traumatic, infectious, neoplastic, toxic or immune mediated. In severe cases, total retinal detachments are possible. In other cases, very few lesions are found yet the animal is clinically blind. This may indicate a severe involvement of the outer retinal layers that is not ophthalmoscopically detectable.

Total retinal detachments due to trauma are seldom seen. Retinal hemorrhage is possible but seldom results in total blindness. A more common sequela to trauma is focal linear or comma shaped areas of retinal edema. These areas are in the outer layers of the retina and do not elevate the retinal vessels. In the non-tapetal area, they appear gray with pigmented margins. In the tapetum, they are seen as abnormal dark pigmented areas with gray margins. Lesions can be found in any location, but are most frequently seen in the peripapillar area and at the tapetal and non-tapetal junctions. These individuals are functionally blind. If treated early at the time of trauma with systemic prednisolone at a dose of 1mg/lb divided b.i.d.. vision may improve.

Infectious diseases with posterior segment involvement are common in veterinary medicine, if only you look. Involvement of the choroid and retina may precede both systemic manifestations and involvement of the anterior segment, i.e., anterior uveitis. Severe involvement of the anterior segment may prevent visualization of the posterior segment. Lesions are seldom symmetrical and initially may be only unilateral.

Admittedly, many lesions are not pathognomonic. However, taking into consideration your region of the country, other systemic signs and finally serology and cytology, a diagnosis can be reached by first looking into the eye. Keeping in mind there is a wide range of overlap, there are certain characteristics of the different lesions that might favor one etiology over another. As a general rule, infectious diseases result in more perivascular exudate and subretinal exudate than the traumatic, metabolic, toxic, or immune mediated diseases. Hemorrhage is seldom a major component in infectious diseases, except Ehrlichiosis.

The systemic mycoses (histoplasmosis, blastomycosis, cryptococcosis, and coccidioidomycosis) can all cause anterior and posterior segment involvement. In the posterior segment, chorioretinitis and optic neuritis are usually found in the advanced cases. Large subretinal white to cream colored granulomas are common in blastomycosis, coccidioidomycosis and cryptococcosis. This same lesion may be found with histoplasmosis, but in most cases histoplasmosis results in areas of abnormal pigment proliferation in the tapetum surrounded by retinal edema.

Toxoplasmosis is more commonly found in the cat associated with anterior uveitis than the dog. Circular areas of retinal edema with no perivascular changes may be found in the tapetum. Those focal areas may also be peripapillar. White perivascular exudates have also been found.

Feline infectious peritonitis should be considered, especially in young cats with anterior uveitis and chorioretinitis. Perivascular exudates, retinal edema and detachments with intraretinal hemorrhage are the common lesions in F.I.P.

Primary ocular tumors seldom cause acute blindness, especially due to chorioretinitis. Lymphosarcoma and FeLV have been associated with posterior segment involvement resulting in retinal hemorrhage and detachments.

**Optic neuritis**

The same etiologies for chorioretinitis can cause optic neuritis. Seldom is the optic nerve involvement the only fundic lesion found. Cases have been seen of acute blindness following blunt trauma to the globe such as a baseball. Traumatic proptosis of the globe frequently results in optic neuritis that may be the actual cause of blindness. Cases have been reported of acute blindness in the

remaining eye days following a unilateral enucleation. Excessive traction on the globe being enucleated resulted in damge to the optic chiasma and retrograde atrophy in the contralateral optic nerve.

Lymphosarcoma and cryptococcosis have been diagnosed as the cause of optic neuritis and acute blindness in the cat. The fundic lesions consisted primarily of neovascularization of the optic nerve and peripapillar edema. These cases are usually diagnosed by cytology from a cerebrospinal tap.

Meningiomas have been diagnosed as the cause of acute blindness. Blindness has preceded other neurological signs by 2-18 months. In one individual, the primary lesion was neovascularization of the optic disc. In other cases, linear areas of abnormal tapetal coloration and retinal edema have been present in the peripapillar area with minimal obvious involvement of the optic nerve.