Feline Ophthalmology Part 2: Clinical presentation and aetiology of common ocular conditions

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In this, the second part of our series on feline ophthalmology, emphasis is placed on the clinical appearance of the common conditions which regularly present in practice. Performing the thorough examination outlined in Part 1 will detect abnormalities. Using the information in Part 2 should help the clinician to correctly identify a condition, allowing treatment and prognosis to be given.

Conjunctivitis

This condition is manifested as congestion or oedema (chemosis) of the conjunctiva. It may be accompanied by an ocular discharge. Secondary conjunctivitis may occur with other ocular conditions, such as uveitis and glaucoma. In cats, conjunctivitis is frequently caused by an infectious agent, and multiple agents may be involved. Resident ocular bacterial flora is present; therefore, a bacteriology swab does not always produce a result with a causative organism. *Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus* spp and *Corynebacterium* spp are commonly isolated commensal organisms from the normal feline conjunctiva and eyelids (Gerding and Kakoma, 1990).

Figure 1: Kitten with FHV-1 infection, with typical conjunctivitis and nasal discharge.

Many upper respiratory tract pathogens cause conjunctivitis. Feline herpesvirus-1 (FHV-1) can infect domestic and wild cats (Maggs *et al.*,1999a,b). The virus is considered to be responsible for nearly half of all upper respiratory tract infections in these species. Replication occurs in the epithelium of the conjunctiva, the nasal mucosa, tonsils and turbinates. Replication is more limited in the corneal epithelium and produces characteristic dendritic ulcers, which are almost pathognomonic for FHV-1 infection. These are small branching superficial ulcers, most easily seen with fluorescein or rose bengal stains. Ocular signs (**Figure 1**) include bilateral conjunctival hyperaemia and ulceration and superficial corneal neovascularisation. Other signs include fever, malaise, anorexia, rhinitis, sneezing, ptyalism and keratoconjunctivitis. Eighty per cent of recovered cats remain latently infected, and viral reactivation can cause recrudescent infections in 45% of these animals (Gaskell and Povey, 1977). Stromal involvement may occur as an immune response to the viral antigen. Upper respiratory signs are usually minimal or absent with recrudescent disease. FHV-1 disease syndromes include keratoconjunctivitis sicca (KCS), corneal sequestration, eosinophilic keratitis, ophthalmia neonatorum, symblepharon (**Figure 2**), uveitis and periocular dermatitis. Symblepharon is an acquired conjunctival adhesion, which is rare in species other than cats. Primary disease caused by FHV-1 is common, but often self-limiting. Recrudescent disease is less common, but very frustrating.

Figure 2: Symblepharon – the conjunctiva is adhered to the lateral cornea, with some pigmentation.

Feline calicivirus usually causes a less severe disease, with ocular signs usually limited to conjunctivitis, mucoid discharge and, sometimes, chemosis. Many cases have oral ulceration, and frequently there is a serous oculonasal discharge. Polyarthritis may be present.

Chlamydiophila felis (formerly *Chlamydia psittaci* var. *felis*) can cause unilateral or bilateral conjunctivitis with marked chemosis and mucoid

to mucopurulent discharge. Corneal and upper respiratory tract involvements are not common.

Bordetella bronchiseptica causes a mild upper respiratory tract infection with oculonasal discharge and conjunctivitis. A severe form of the disease can occur, especially in combination with other upper respiratory tract pathogens, causing lymphadenopathy, sneezing, coughing and adventitious lung sounds.

Mycoplasma felis may be isolated from normal eyes, or it may cause a pseudomembranous conjunctivitis with conjunctival pallor.

Lipogranulomatous conjunctivitis is a not uncommon condition in older cats, which is often not noticed and has had little representation in the literature. Lipogranulomas are found distributed under the palpebral conjunctiva on upper and lower eyelids, and appear to be associated with the meibomian glands (**Figure 3**). They present as single or, more commonly, multiple, raised, cream-white, smooth nodules within the palpebral conjunctiva. They may be present in one or more lids, and they are more commonly present in non-pigmented eyelids (Read and Lucas, 2001). The lesions may lead to chronic discomfort.

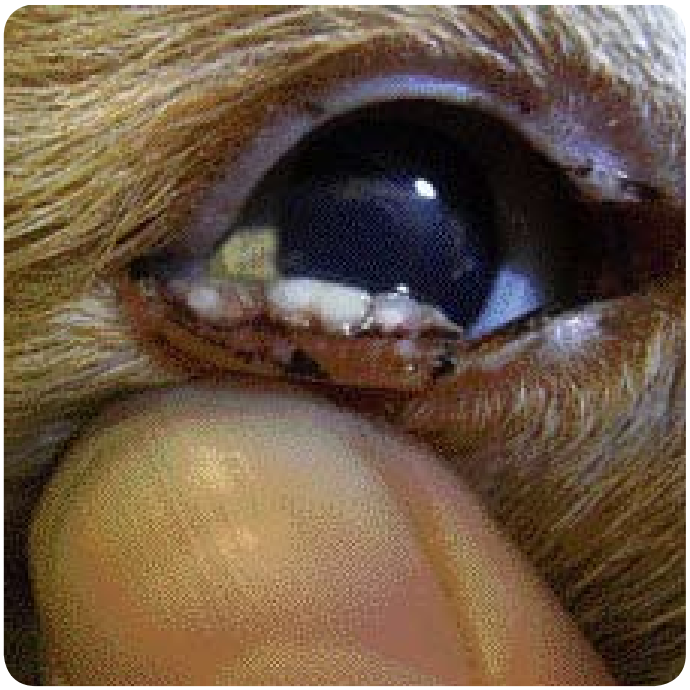

Figure 3: Lipogranulomatous conjunctivitis – multiple white nodules in the palpebral conjunctiva of the lower lid.

Nictitating membrane

Usually, the normal feline third eyelid is hidden, with the leading edge barely noticeable. Prominence of the third eyelid is noted in cats with a number of conditions. Aetiology of unilateral and bilateral protrusions is presented in **Table 1**. Interestingly, loss of the retrobulbar fat pad, debility and dehydration do not cause protrusion of the nictitating membrane to the same extent as they do in dogs.

Lacrimal system

The cat does not blink as frequently as the dog, and the corneal tear film is more stable. However, keratoconjunctivitis sicca (KCS) can occur in cats and is commonly overlooked. Schirmer tear tests should be performed in cases of conjunctivitis or surface ocular disease. Normal values are 5mm to 11mm in one minute, but they need to be interpreted in conjunction with evidence of ocular surface disease. Clinical presentation depends on the stage at which the disease is presented and varies, from conjunctival hyperaemia with superficial keratitis to severe ulcerative keratitis, with mucoid to mucopurulent discharge. Causes can be neurogenic (e.g., dysautonomia), drug induced (e.g., atropine administration) or idiopathic, or can be due to trauma, FHV-1, symblepharon or mucin abnormalities. Apart from quantitative KCS, qualitative KCS can also cause corneal disease, thought to be due to a deficiency in tear mucin (Cullen *et al.*, 1999)

Table 1: Protrusion of the third eyelid in cats

Unilateral	**Globe factors**	**Sympathetic innervation: Horner's syndrome**
		Size: microphthalmos
		Position: enophthalmos, e.g., Horner's syndrome
		exophthalmos, e.g., retrobulbar space-occupying lesion
	Third eyelid factors	**Prolapse of the gland of the third eyelid (TE)**
		Eversion of the cartilage of the TE
		Foreign body behind the TE
		Inflammation, e.g., bulbar conjunctivitis
		Symblepharon
		Neoplasia
		Trauma
Bilateral		**Haws syndrome with chronic diarrhoea *(Figure 4)***
		Dysautonomia (Key-Gaskell syndrome)
		Drug-induced, e.g., phenothiazine sedatives
		Tetanus

Figure 4: Bilateral prolapse of the nictitating membranes in a case of Haws syndrome in a cat with chronic diarrhoea.

Cornea

Corneal ulceration is commonly the result of trauma (**Figure 5**). The position of the ulcer may give clues as to its aetiology. Central lesions may be due to exposure (e.g., facial nerve paralysis, exophthalmos, KCS, lagophthalmos). Perilimbal lesions may be caused by abnormalities of eyelids or cilia, while medial lesions may indicate a foreign body behind the third eyelid.

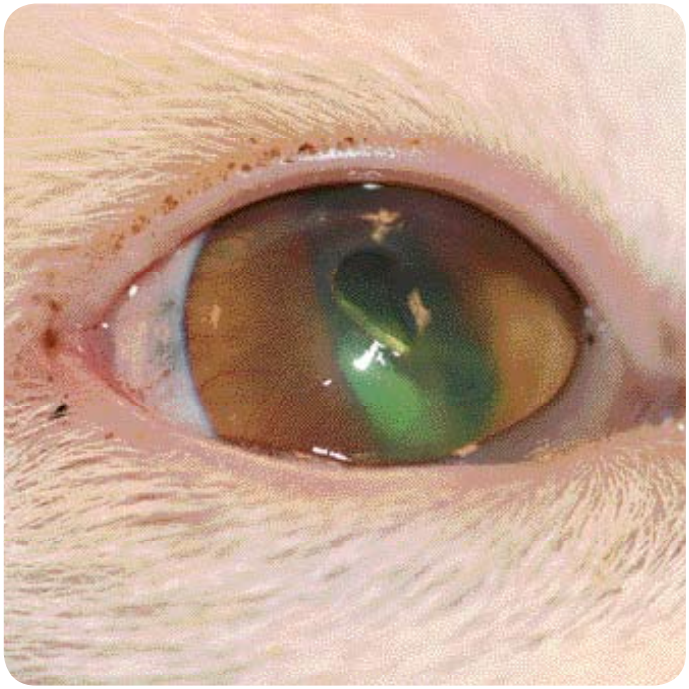

Figure 5: Corneal ulceration in the right eye of a domestic short-haired cat with a concurrent uveitis, exhibited by the focal scattering of opacities on the ventral aspect of the corneal endothelium (keratic precipitates), in this case secondary to phacoemulsification surgery one week previously.

All cases of conjunctival and ocular surface disease should be stained with fluorescein dye. There are frequently signs of unilateral ocular discomfort, such as blepharospasm, epiphora and, sometimes, photophobia. The corneal epithelial deficit may be very superficial (usually the most painful presentations) or it may involve the deeper stroma. Occasionally, they can progress to a descemetocoele (**Figure 6**), in which the elastic Descemet's membrane may be protruding visible from the back of the cornea into the corneal facet. Infectious organisms may contribute to the disease, as many ulcerations are caused by claw injuries.

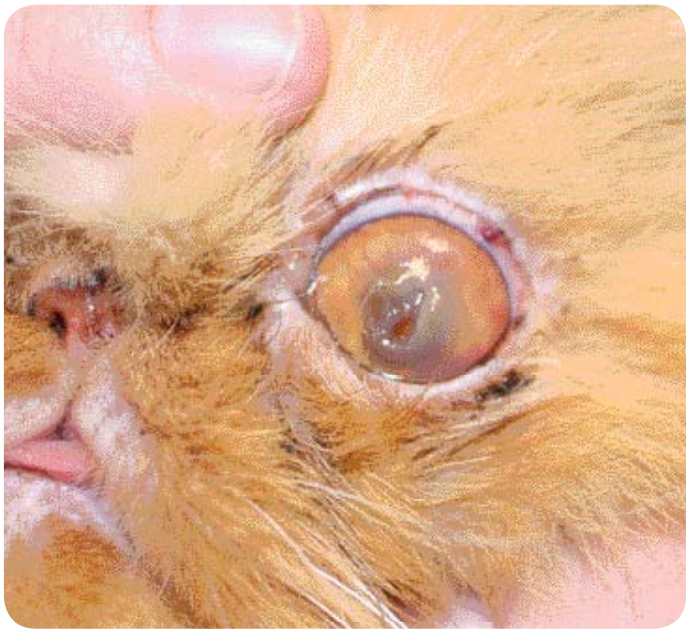

Figure 6: A melting ulcer in the left eye of a ten-year-old Persian cat with corneal neovascularisation, oedema and a central deficit exposing Descemet's membrane.

Figure 7: Early sequestrum formation in the left eye of a British Blue cat, visible as a focal black area on the cornea.

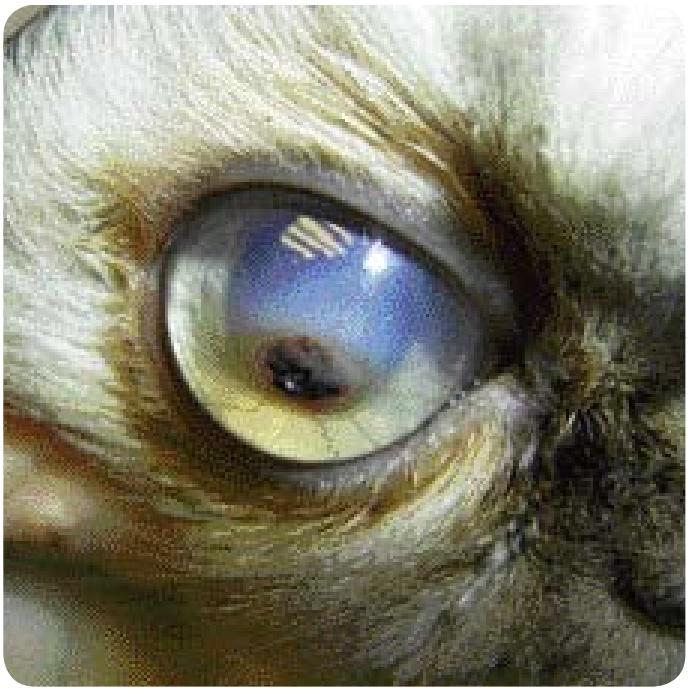

Figure 8: Black necrotic corneal stroma (sequestrum) with corneal oedema and neovascularisation.

Corneal sequestrum (**Figures 7 and 8**) formation is a condition almost unique to the domestic cat, and it has a characteristic clinical appearance (Featherstone and Sansom, 2004). Any breed may be affected, but there is a breed predisposition for Persian, Birman, Burmese, Himalayan and Colour-point cats (Morgan, 1994). Sequestra can occur in one or both eyes and in cats of all ages. Cats present with a history of ocular discomfort. There is a small to extensive oval to circular lesion, usually in the central or paracentral cornea, with variable pigmentation causing an amber to black discoloration (Featherstone and Sansom, 2004). Many cases are negative for FHV-1 infection, and it is more likely that corneal sequestrum formation in feline patients is the culmination of chronic damage caused to the cornea by a variety of ocular diseases.

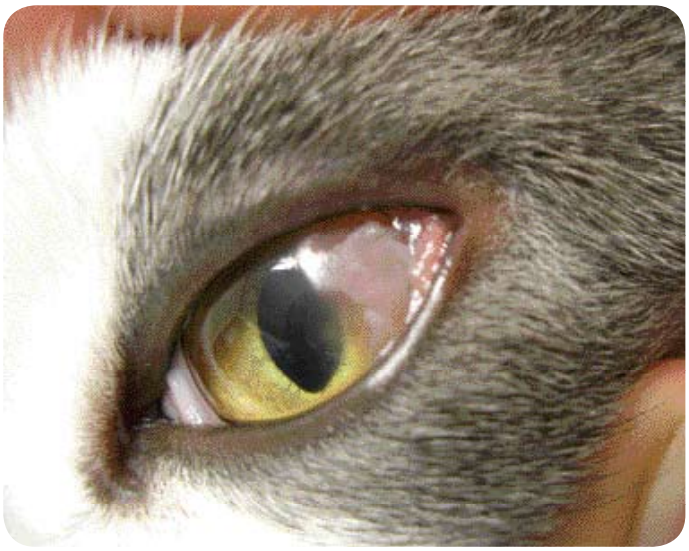

Figure 9: Typical presentation of eosinophilic keratitis with opacity in the lateral aspect of the cornea.

Eosinophilic keratoconjunctivitis (**Figure 9**) is a common immune-mediated condition in the cat. The cornea is primarily affected, with slowly progressive infiltrative disease (Allgoewer *et al*., 2001). A white plaque is usually proud of the cornea, resembling cottage cheese. There is usually associated corneal neovascularisation and conjunctivitis, and there may be multifocal pinpoint ulcers. Typically, the lesion is on the dorsolateral cornea, but it may present in any position on the cornea. The aetiology of the condition is not known (Morgan *et al*., 1996). A study by Nasisse *et al*. (1997) showed that 76% of corneal scrapings were positive for FHV-1 DNA by PCR assay. Association with feline eosinophilic granuloma complex has not been confirmed.

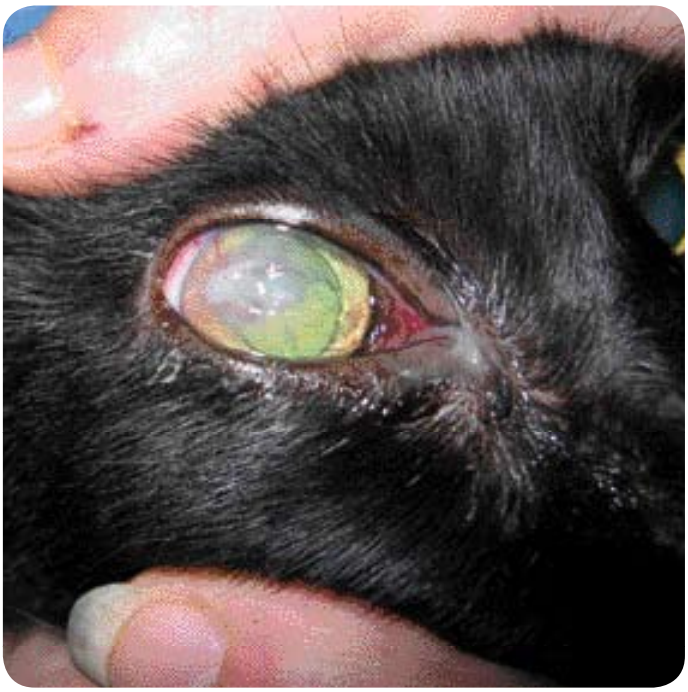

Figure 10: Exposure keratopathy in the right eye of a twelve-year-old domestic short-haired cat unable to blink due to exophthalmos caused by a retrobulbar squamous cell carcinoma.

Exposure keratopathy (**Figure 10**) may occur when there is interference with normal eyelid functions. An inability (**See Table 1**) to blink is termed lagophthalmos, and it may be caused by a number of conditions. Physical impediment to complete closure of the lids may occur with abnormal size of the globe, seen in glaucoma-associated globe enlargement (buphthalmos), or due to abnormal position of the globe, as is the case with exophthalmos caused by a retrobulbar space-occupying mass. Denervation of the lids occurs with palsy of the facial nerve, which may occur due to trauma or to aural surgery. However, compensation is usually adequately provided by the retractor oculi muscle, which is innervated by the abducens nerve. Activation of this mechanism allows the third eyelid to sweep across the ocular surface. Distribution of the tear film is impeded by removal of the third eyelid.

Calcium keratopathy (**Figure 11**), or corneal calcification, occurs most commonly in corneal degeneration, but it may occur with systemic hypercalcaemia.

Figure 11: Calcium keratopathy.

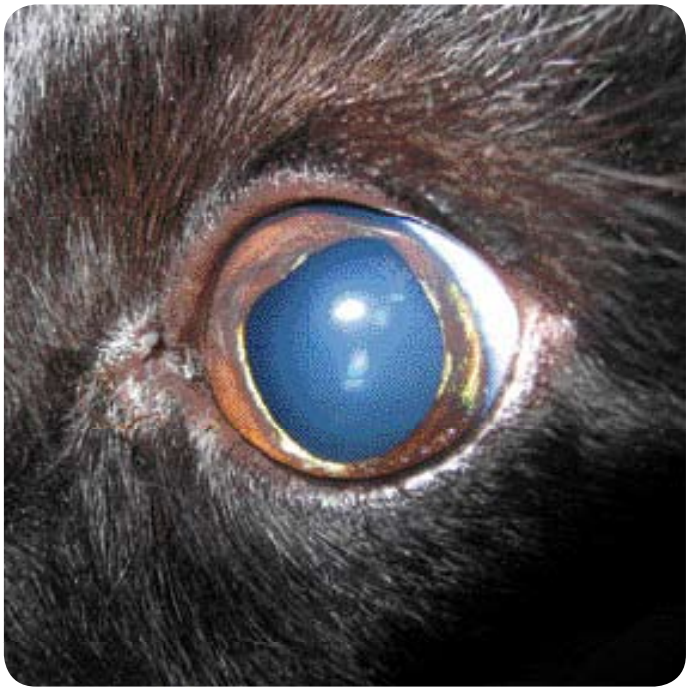

Figure 13: Diffuse iris melanoma with raised pigmented areas in multiple areas of the peripheral iris.

Uvea

It is not unusual to be presented with a cat with discrete foci of pigmentation visible on the surface of the iris (**Figure 12**). These are known by many names, including iris melanocytoma, iris freckles or nevi and benign melanosis. The cause is unknown, although it may be an aging change. When on the anterior surface of the iris, these areas need to be monitored. Although at this stage they may be considered harmless, they do tend to slowly grow and sometimes coalesce.

Figure 12: Multiple iris freckles.

However, invasion into the deeper layers of the iris may occur, resulting in a diffuse iris melanoma (**Figure 13**), which is the most common primary intraocular tumour in the cat. These are best examined with a slit lamp, as the drainage angles need to be assessed and ocular ultrasound is indicated. A cautious approach is prudent, especially if the lesions appear raised, if the pupil is misshapen (dyscoria) or if there is a glaucoma or uveitis. Early enucleation may be warranted as survival time in cats with extensive melanoma is greatly reduced (Kalishman *et al*,. 1998). Specialist advice should be sought.

Inflammation of one or more components of the uveal tract (iris, ciliary body and choroid) is termed uveitis (**Figure 14**). In general, the feline eye responds less intensely to inflammation than does the canine eye. This can result in a greater diagnostic challenge in the early stages of disease. Clinical presentation of uveitis can vary

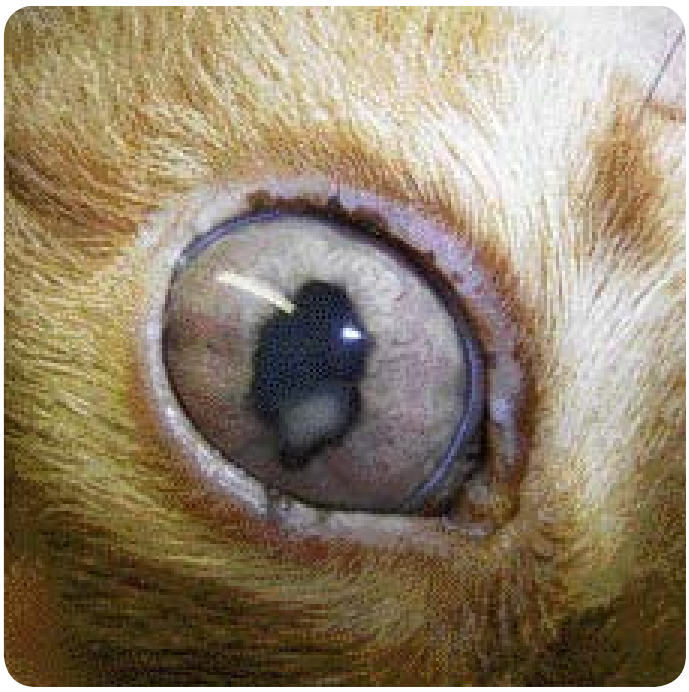

Figure 14: Swelling (iritis) and reddening (rubreosis iridis) of the iris causing a dyscoria (distorted pupil) and focal accumulation of inflammatory cells visible on the corneal endothelium. This is a case of uveitis.

greatly, depending on whether the disease is acute or chronic and on which part of the uveal tract is involved. Some of the following signs are found: ocular pain, redness, corneal oedema, keratic precipitates, aqueous flare, pre-iridal fibrovascular membrane formation, 'muddy' colour of iris, lymphoid nodules, synechiae, pupillary dilation, lens luxation/sub-luxation, cataract, low intraocular pressure, poor pupillary response to light and impaired vision (Hopper and Crispin, 1992). Pre-iridal fibrovascular membranes are easy to see in the cat, and are a frequent finding with chronic uveitis. Synechiae may be differentiated from persistent pupillary membranes. In both situations, the iris becomes adherent to the corneal endothelium, the anterior surface of the lens, or to the peripheral cornea involving the iridocorneal angle. Persistent pupillary membranes arise from the iris colarette, which is at the junction of the pupillary and peripheral zones of the iris, whereas synechiae usually arise from the iris margin. Causes of uveitis may be infectious or non-infectious. Infectious causes are much more common in the cat than in the dog.

Lens

The term cataract refers to lens or lens capsule opacification. Cataracts may be seen easily using the technique of distant direct ophthalmoscopy. In a darkened room, with the lens wheel set to zero, the tapetal reflex is observed from arm's distance. Opacities show up as black areas, and may be either focal or diffuse. Closer examination will reveal whether the opacity is in the lens, in front of it, or behind it. Anisocoria (pupils of unequal size) may also be appreciated using this technique. Most feline cataracts are acquired, usually as a secondary event to trauma (either blunt or penetrating), chronic anterior uveitis or chronic glaucoma. Diabetic cataracts are rare in the cat, because of low aldose reductase activity in the lens.

Lens luxation or subluxation (**Figure 15**) most commonly occurs as a secondary event following trauma, chronic anterior uveitis, chronic glaucoma or cataract formation. An aphakic crescent is usually visible where the fundic reflection may be seen in an arc shape peripherally behind the iris. This may be visible to the naked eye, and it is more noticeable on distant direct ophthalmoscopy. Glaucoma or uveitis may be the cause or the result of the condition.

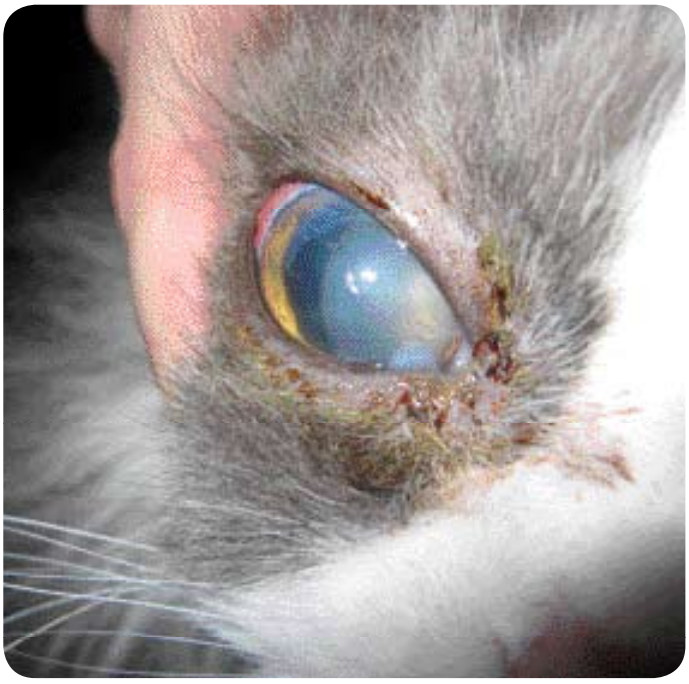

Figure 15: Aphakic crescent visible due to posterior lens luxation.

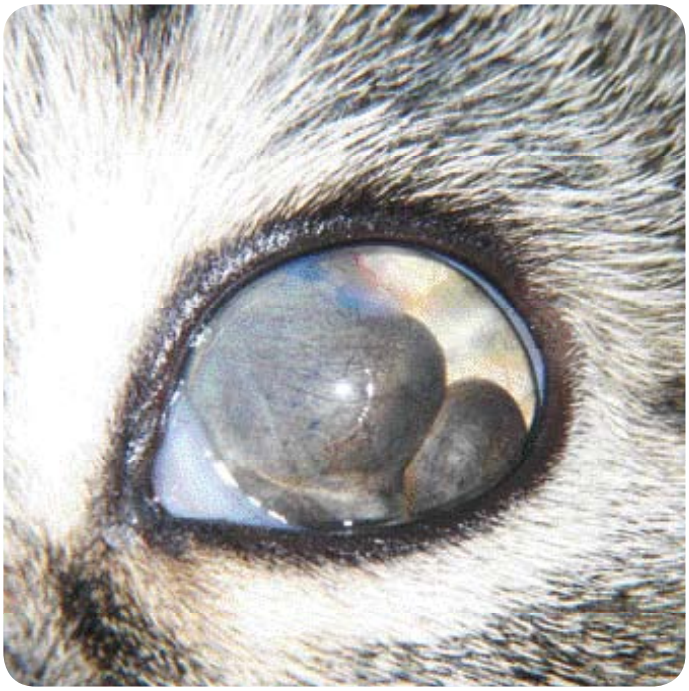

Figure 16: Gross distortion of the iris, which is bulging forward due to cystic expansion of the iris. This is a response to a ruptured lens and lymphoplasmacytic uveitis, consequent to severe blunt trauma two years previously.

Penetrating injuries are commonly the result of a cat scratch. Damage thus induced can cause a cataract, phacoclastic uveitis, or uveitis associated with *Pasteurella multicoda* or *Bartonella henselae* infections introduced by the cat claw. The prognosis is poorer if the lens epithelium has been breached. The release of lens material within the feline eye, along with severe trauma, are thought to be possible causes for the much later development of intraocular spindle cell sarcoma (**Figure 16**). The sarcomas are aggressive, and they can invade the optic nerve and grow towards the brain. These post-traumatic sarcomas develop years later and, for this reason, a badly damaged feline eye with concurrent lens damage should be enucleated. Intraocular sarcoma following trauma has not been reported in any other species.

Fundus

It is essential to be very familiar with the appearance of a normal fundus. Compared with the dog, fewer variations exist in the cat, the main variation depending on colour dilution. The normal cat has a very large tapetal fundus, containing a small, round, grey optic disc, from which three pairs of arteries and veins arise. Choroiditis may exist due to infection, or as a component of uveitis. Infectious causes include feline leukaemia virus (FeLV) and the causal agents of feline infectious peritonitis (FIP), toxoplasmosis, cryptococcosis and histoplasmosis.

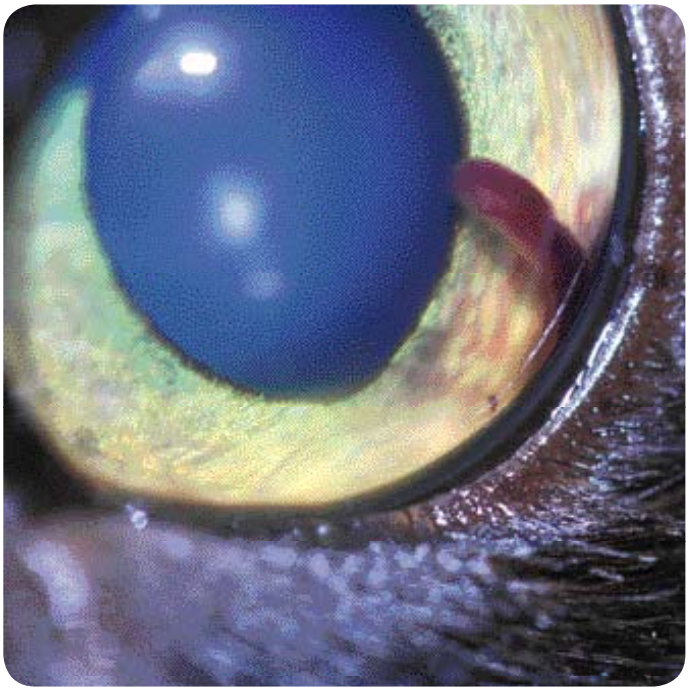

Figure 17: Iris aneurysm; an early sign of systemic hypertension.

Hypertension in the cat occurs with a sustained systolic blood pressure of over 170mmHg. Ocular hypertensive changes include iridal haemorrhage (**Figure 17**), hyphaema (bleeding from iridial capillaries into the anterior chamber), mydriasis, tortuosity of retinal vasculature, retinal haemorrhage, bullae or detachment and blindness. The cause of hypertension needs to be investigated to discover if it is a primary condition, or secondary to a systemic problem, such as chronic renal failure, hyperthyroidism, hyperaldosteronism etc. Taurine deficiency initially presents as feline central retinal degeneration, with the earliest lesions commencing as an increased reflectivity lateral to the optic disc. As taurine deprivation worsens, the lesion spreads to create a band of increased reflectivity corresponding with the area centralis of the fundus. These animals usually present with a cardiomyopathy. Fortunately, the condition is rare nowadays, as pet food manufacturers have included sufficient taurine in feline formulations. Intra-retinal haemorrhage may be apparent in anaemic individuals. There have been reports of blindness in cats (Gelatt *et al.*, 2001) following enrofloxacin doses in excess of 5mg/kg, particularly by parenteral route. Signs include mydriasis, blindness and slow pupillary light reflexes. Initially the fundus looks normal, but it progresses with time to end-stage degeneration with tapetal hyper-reflectivity, vascular attenuation and a mottling of the non-tapetal fundus.

The orbit

The cat has an incomplete bony orbit and a close relationship with the masticatory muscles, the caudal molar tooth roots, the paranasal sinuses and the zygomatic salivary glands. Facial trauma may involve fractures of bones making up the orbit or nerve damage. Oculomotor nerve (cranial nerve III) damage may result in disruption of the nerve supply to the iris sphincter muscle, leading to mydriasis, also referred to as internal ophthalmoplegia. The post-synaptic parasympathetic axons are in the short ciliary nerves, of which the cat has only two. Thus, a partial parasympathetic lesion may cause a D-shaped or reverse D-shaped pupil, as it is hemi-dilated. If the motor component of the nerve is also damaged, the resulting external ophthalmoplegia causes paralysis of extraocular muscles leading to a ventrolateral strabismus with upper lid drooping (ptosis). These eyes are capable of vision. Facial nerve palsy is usually due to trauma, and manifests as an inability to blink (lagophthalmos) with the third eyelid compensating by sweeping across the eye, distributing the tear film. This method is adequate in cats with normal conformation, but in brachycephalic cats the prominent globes may suffer an exposure keratitis, leading to keratitis and ulceration. Keratoconjunctivitis sicca may occur due to disruption of lacrimomimetic stimulation by the facial nerve.

Attali-Soussay *et al.* (2001) documented typical presentation of a retrobulbar tumour as unilateral exophthalmos (84%), protrusion of the nictitating membrane (28%) and several signs secondary to exophthalmos such as conjunctival hyperaemia (40%) or exposure keratitis (20%). Other clinical signs included fundus abnormalities (20%) such as retinal detachment, vasculature modification or oedema, and neuro-ophthalmologic effects (16%) such as lack of menace response, pupillary light or corneal reflexes. They found that 88% of these tumours were malignant. In a different study on 21 cases of feline orbital neoplasia, 85% were found to be secondary tumours and only 15% were identified as primary neoplasia (Gilger *et al.*, 1992). The more common types of orbital neoplasia were found to be lymphosarcoma, and oral/nasal/adnexal squamous cell carcinoma.

Glaucoma

Glaucoma is a collection of neuro-destructive diseases, which results in the intraocular pressure being too high to allow normal functioning of the retina and optic nerve. Normal intraocular pressure is 12mmHg to 26mmHg. Characteristic signs include globe enlargement (buphthalmos), episcleral and conjunctival hyperaemia, corneal oedema, mydriasis, pain and loss of vision (Ridgeway and Brightman, 1989). The lens may be secondarily luxated or subluxated due to stretching of the ciliary zonules, which suspend the lens. The condition occurs less frequently in cats than in dogs. While primary glaucoma can occur, especially in the Siamese breed, the most common glaucoma in cats is secondary glaucoma (Blocker and van der Woerdt, 1999). This is most commonly due to idiopathic lymphocytic–plasmacytic anterior uveitis (**Figure 18**), or due to blockage or obliteration of the ciliary cleft by diffuse iridal melanoma or other neoplasms (Wilcock *et al.*, 1990).

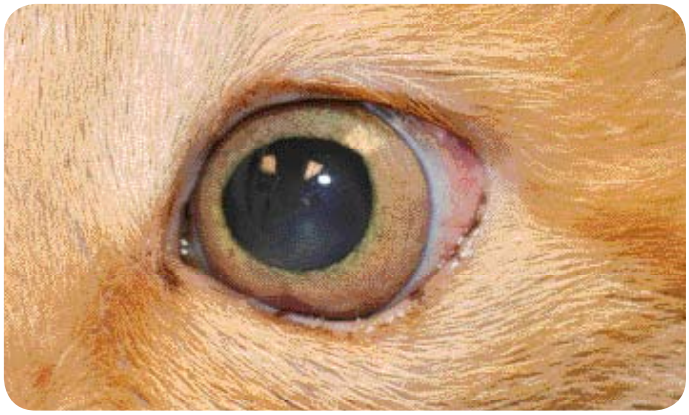

Figure 18: Rubreosis iridis and dilated pupil (mydriasis) in this case of glaucoma, secondary to chronic lymphocytic–plasmacytic uveitis.

Systemic disease

The primary presenting sign for a systemic disease may be ocular (Miller and Johnson, 1989), and this must be borne in mind when investigating an eye case. The eye is a window through which many systems can be directly examined, such as the vascular, nervous and endocrine systems. Thus, diagnostic aids can be obtained for many medical conditions. Examples of viral disease with ocular signs include FeLV, FIV (feline immunodeficiency virus), FIP, FHV-1, calicivirus, Haws syndrome, panleukopenia virus, and poxvirus. Another condition that is readily diagnosable on funduscopic examination is hypertension. There may be hyphaema, retinal detachment or serous exudation under the retina appearing as dull, elevated, hyporeflective areas of retina. Retinal haemorrhages may be small or extensive, depending on the extent of damage.

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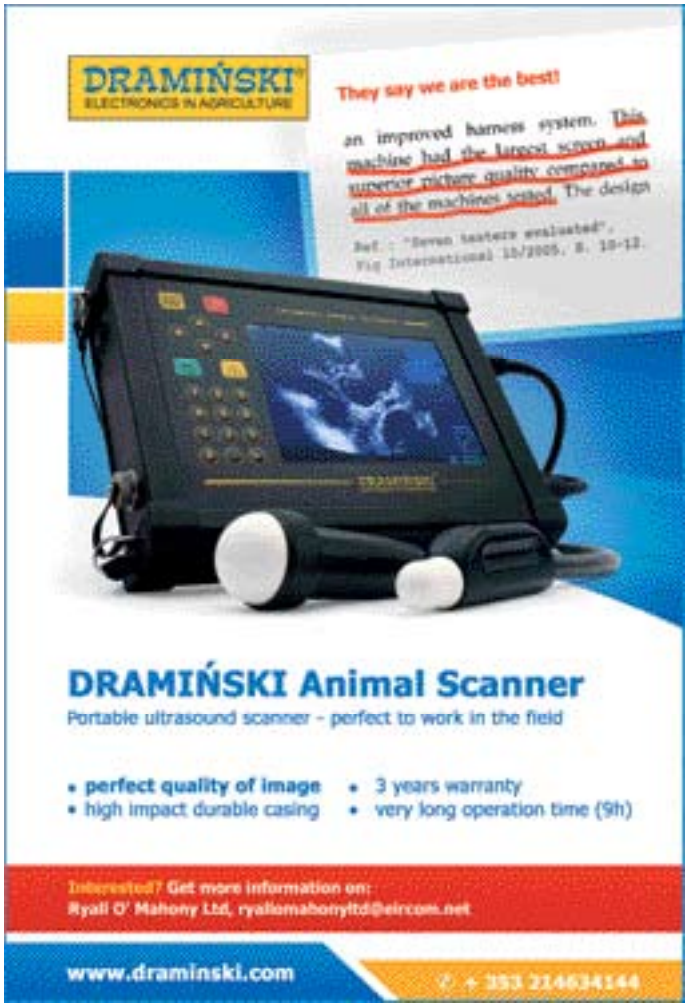

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