FELINE OPHTHALMOLOGY

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Cats are increasingly popular as pets. By the end of the previous century, the number of cats exceeded the number of dogs in the USA.

Principally, in cats, similar ocular diseases as in other mammalian companion animals are seen. Yet, some ophthalmic diseases occur in felines that are not seen in other (small) companion animals. Examples of these are: the corneal sequestrum, and eosinophylic keratitis and conjunctivitis, blindness due to taurine deficiency. Some other eye diseases show a different expression, course, or different etiological or pathobiological signs in the cat as compared to, for example, the dog. Examples of this type comprise ocular herpes problems, iris melanoma and some types of retinal atrophy.

In this paper, the most important ophthalmological disorders in felines will be discussed, as well as some specific diagnostic and therapeutic aspects for cats. Cats are not small dogs!

Palpebral aplasia
In palpebral aplasia the margins of the eyelids are completely or partly undeveloped. This anomaly is congenital, most likely hereditary (recessively), and usually bilateral, affecting the lateral part of the upper eyelid. It occurs relatively frequent in Persian cats and Persian crossbreeds. Palpebrae aplasia may be associated with other congenital anomalies.

Affected kittens are often born with the palpebral fissure partly or fully open. There are frequently hairs in that area, that are directed toward the globe which predispose to chronic irritation of the corresponding area of the cornea. The eyelids cannot partly or completely close. As a result, parts of the cornea are exposed and cannot be protected by an adequate lid function, leading to lesions of chronic irritation (edema, neovascularization, pigmentation, ulceration and sequestration).

If there are few ectopic hairs in the area, these have to be permanently removed. If the cornea is only slightly irritated, administration of topical lubricants, to effect, on a daily basis will be sufficient. If the lesions are larger, a substitute eyelid should be created by blepharoplasty.

The breeder/owner should be informed that these anomalies may be hereditary, and that the parents and littermates should be examined carefully for these and other anomalies. Affected animals, and ideally also members of their immediate family should not be bred.

Dermoid
Dermoids are ectopic and abnormally developed islands of skin in or on the the cornea and conjunctiva, sometimes extending to the eyelids. In the cat, it is a rare anomaly. In the Burmese this condition most often bilateral, and is probably hereditary in this breed. An isle or fold of skin often disrupts the lid margin and is continuous with the conjunctiva. Blinking is abnormal and hairs generally grow toward the cornea causing chronic irritation, and resulting in edema, vascularization, and pigmentation.

Treatment consists of a very precise keratectomy with a rounded scalpel (e.g., Beaver 6400), beginning at the central margin and removing the dermoid and the superficial stromal layers into the direction of the limbus. Magnification of 5-10x is necessary to be certain that no hair follicles remain and that the cornea is not incised too deeply.

If the lids are affected, the affected parts are removed as well. In these cases, blepharoplasties are seldom necessary, as the fissure length is sufficient in most cases.

The prognosis is favorable. Parents and littermates should also be examined. Affected animals and ideally also members of their immediate family should not be used for breeding.

Lid neoplasia
Neoplasms of the eyelids are quite common in dogs, horses and cattle but rare in cats.

In the cat, lid neoplasms usually are malignant (squamous cell carcinomas, mastocytomas). Squamous cell carcinomas are more often seen in white cats (possibly in connection with UV hypersensitivity). In cats, eosinophilic granulomas must be considered in the differential diagnosis.

Carcinomas are usually flat, slowly growing, ulcerating defects at or near the margin of the eyelid. They may initially show as an hyperemic area, with some dark exudate. They are often accompanied by neoplasms on other parts of the body (e.g., ears, lips). Therefore, these parts have to be examined thoroughly. The diagnosis is established by means of biopsy or, in the case of small neoplasms, by direct radical excision.

Therapy: Neoplasias of the eyelids should be carefully surgically removed (blephaorplasty). The tumor itself should never be squeezed during surgery, otherwise causing artefacts for the pathologist. The tumor, in 5% formaldehyde, is at least stored, but better directly sent for histopathologic evaluation.

Treatment of larger tumors, and tumors with distinct aspects of malignancy, or recurrences consists of radical removal of the neoplasm. These methods usually require more extensive blepharoplasties. In malignant tumors, adjunctive therapy (cryo, hyperthermia, radiation, brachytherapie, immunotherapy, et cetera) is advisable in most cases. In all cases, a preoperative metastases detection is to be performed.

Conjunctivitis

In conjunctivitis exudate is mixed with the tear film. Viscosity increases and lacrimal drainage decreases, resulting in epiphora and tear stripe formation below the medial canthus. This reaction may be caused by wind, dust, or allergens. Other causes are acute infections such as in the upper respiratory disease in felines caused by agents as viruses, Mycoplasma and Chlamydophila This is followed by the production of more mucus and more desquamated epithelial cells. Production of purulent material is usually more prominent in bacterial and fungal infections. Purulent conjunctivitis often follows catarrhal conjunctivitis, caused by mechanical or infectious agents.

Catarrhal conjunctivitis is characterized by epiphora, redness, slight swelling, and mucous discharge. In the acute phase, epiphora is especially prominent. In the chronic phase (after 1-2 weeks), redness, swelling, and discomfort increases.

Conjunctivitis usually causes more itching than pain. The predominance of pain is a sign of corneal involvement. The pruritus may result in rubbing and a minor blepharospasm. Often some follicular activity will follow, increasing when the conjunctivitis persists. Having much more serious consequences for the eye are epithelial defects in the conjunctiva and cornea which may occur in upper respiratory disease in cats. When two opposite layers are affected, the surfaces may adhere and grow together (symblepharon). This process is often found in the lacrimal canaliculi, between the palpebral conjunctiva of the nictitating membrane and the lids, and between the palpebral conjunctiva and the cornea.

Therapy: Therapy in *acute* conjunctivitis consists of removal of discharge by irrigation, 1-4 times daily. The conjunctiva usually begins to regenerate in a matter of days. If there is no spontaneous improvement, broad spectrum ocular antibiotic drops or ointment can be prescribed. In cats, oxytetracycline or chlortetracycline ointment is often preferred.

In *acute purulen*t conjunctivitis, therapy consists of dissolution of the purulent exudate by acetylcysteine, followed by irrigation, at least 4 times daily. Antibiotic therapy (tipcal and/or systemic) may be prescribed as well. In acute conjunctivitis due to upper respiratory disease in the cat topical therapy includes acetylcysteine (decreasing the risk of symblepharon; see below).

Symblepharon

Symblepharon is an adhesion between parts of the conjunctiva or between the conjunctiva and the cornea. It is especially found in young cats as a complication of feline ocular herpesinfection (FHV-1). The virus and the secondary bacterial infection destroy the superficial layers of the epithelium. Together with the mucous discharge this easily results in conjunctival layers adhering and subsequently growing together. In particular, there are frequently adhesions between the walls of the lacrimal canaliculi and/or the palpebral conjunctiva of the nictitating membrane and the rest of the conjunctiva. Strangely enough, symblepharon is usually unilateral.

Signs of symblepharon include epiphora, tear-stripe formation, permanent protrusion of the nictitating membrane, and blepharospasm. The adhesions are found during examination of the conjunctival sac. If the cornea is involved there may be membranes of scar

tissue spreading over and loosely attached to the cornea.

Treatment consists of loosening the adhesions. Installation of a soft bandage contact lens covers the globe and may facilitate regrowth of the epithelium and thus prevent recurrence of adhesion. Still, recurrence is possible. Surgical treatment should only be performed after the underlying infectious disease has run its course.

Adhesions may be prevented when, during the acute phase of upper respiratory disease in cats, the adhesive mucous discharge is removed very thoroughly by topical acetylcysteine and irrigation.

Eosinophilic keratitis
Eosinophilic keratitis is a superficial keratitis in the cat in which the granulation component over the cornea is prominent. The etiology is not known but is believed to be related to (previous) herpes infection. It occurs fairly infrequently and can be unilateral or bilateral. A zone of opacity followed by pink granulation tissue invades the superficial layer of the cornea, usually from the lateral side. Sometimes the granulation is locally covered by white-yellow ("cottage cheese") necrotic plaques (staining fluorescein positive). The diagnosis is made on the basis of the finding of large numbers of eosinophils among other inflammatory cells by cytologic or histologic examination.
The therapy consists of topical treatment with dexamethasone drops (0.1%) 3-4 times daily until the signs disappear, after which maintenance administration to effect is adequate. Treatment with ß-irradiation (Sr^{90}), megestrol acetate (orally), or cyclosporin (topically) is also possible. Megestrol acetate can be used, but the progestagenic and diabetogenic side effects should be considered.

The prognosis is favorable, even if recurrences have to be suppressed repeatedly.

Intraocular haemorrhage
Intraocular haemorrhage may be caused by a number of underlying causes such as trauma, uveitis, coagulopathy, vasculopathy, intraocular neoplasia and systemic hypertension.

Especially in cats with intraocular haemorrhage aged older than 11-12 years, systemic hypertension should be considered as the most probable cause. A general physical examination is of great importance and measurement of the (systolic) blood pressure should be performed. Systolic pressure values over 170 mm Hg should be considered abnormal. If confirmed, examinations for underlying diseases (hyperthyroidism, chronic kidney failure, hyperaldosteronism, etcetera) should be performed.

Initial symptomatic anti-hypertension treatment consists of oral amlodipine. Hypertension patients should be closely monitored, preferably by an internist.

Lens luxation
The lens can dislocate or luxate by rupture of the zonular fibers. This disorder occurs much more frequently in the dog than in the cat (about 9:1). In the cat, lens (sub) luxation is seen predominantly in elderly cats suffering from a chronic anterior uveitis (often subclinical). Secondary glaucoma occurs usually less acutely, less rapidly, and less often in the cat than in the dog.

The earliest recognizable sign of lens luxation is the presence of vitreous in the anterior chamber, observable as very thin white treads or clouds. If the lens is displaced, an "aphakic crescent" will be visible between the pupil margin and the lens equator. If the lens is fully luxated, the anterior chamber is deeper (in posterior luxation). The loss of support for the iris can also result in an iridodonesis. In lens luxation towards anterior the lens is completely visible in the anterior chamber (slit lamp!), directly behind the cornea.

Medical (miotic) and surgical therapies are available. The surgical therapy consists of complete (hence intracapsular) removal of the lens. Because secondary glaucoma occurs less rapidly in the cat, removal of the lens is less urgent in most cases.

Hereditary photoreceptor degenerations
The term "PRA" is used, especially in breeders' circles, for a large group of hereditary primary retinal atrophies with the collective clinical denomination "PRA". PRA is always hereditary, bilateral and symmetrical. The most important types of PRA are < gekenmerkt > by progressive, irreversible photoreceptor disease with secondary degeneration of the other retinal components.

PRA can be divided into many types, most of which start with degeneration of the rods and, hence, with night blindness. This is mainly of importance in connection with the age at which the signs of night-blindness first appear. For the cat, two main types are of

importance. If the rods and/or cones are abnormally developed (dysplastic), they will also degenerate early in life. One form of PRA of this type is rod cone dysplasia which occurs in Abyssinian and Somali (Rdy). The mutation is a single base pair deletion in a different gene *(CRX)*, which results in a defective protein that is critical for ophthalmic development. Cats carrying one copy of this mutation have a retarded development and subsequent degeneration of photoreceptor cells, which leads to *early-onset* blindness by 7 weeks of age. The Rdy mutation is inherited as a dominant trait. This mutation is rare.

In the late onset forms, the rods are normally developed and the degeneration starts later in life. In Abyssinian, Somali and some Ocicat breeds, an inherited *late-onset* PRA has been identified. This disease has been designated rdAc. Cats affected with this form of blindness have a normal vision at birth, with electroretinographic (ERG) demonstrable signs of degeneration at about seven months of age. Vision loss is progressive, with most cats becoming blind by 3-6 years of age.

The diagnosis of retinal atrophy is based on the signs and the findings in ophthalmic examination (fundoscopy!), and can be confirmed by an ERG. In differential diagnosis the disease could be confused with non-inherited, bilateral, diffuse, progressive retinal degenerations, such as taurine deficiency or quinolone-associated retinal atrophy in the cat.

There is no available therapy. The prognosis for vision is hopeless. Blind cats can, however, manage quite well in their familiar environment.

Prevention: Animals with signs of PRA should be considered as having the disease until proven otherwise. It is also of great importance that the diagnosis is confirmed by DNA testing (if available).

Drug-associated photoreceptor degeneration
Quinolone-associated retinal atrophy was first diagnosed in cats in 1997. This photoreceptor degeneration progresses very fast, and may occur after one dosage.

Predispositions: elderly cats, outgoing, summer/Sunny wheter, and a (too) high dose vrijloop, zomer/zonnig weer, (te) hoge dosering. The dosage should not exceed 2.5 mg/kg (once every 12 hours max), and should be administered as shortly as possible.

Taurine deficiency
Taurine is an acid amine that - although not a building stone for protein - is still sometimes described as an amino acid. In most animal species it is not an essential nutritional element but cats cannot produce sufficient taurine themselves. High concentrations of taurine are found in the retina, brain, liver, and heart. Considerable taurine is also present in other animal "products" such as milk, meat, fish, and shellfish.

Taurine is especially important in neurotransmission and for the cell membranes and photoreceptors, but it also has a function as a bile acid conjugator and in energy transport.

Taurine deficiency can be expected in cats that are fed an exclusively vegetarian diet or only dog food (often containing much less meat than the label suggests!).

Symptoms: Disturbances in the ERG occur only after 5 weeks on a taurine-free diet. After about 20 weeks, granulation develops in the central area, which is the area with the highest concentration of cones. Hyperreflection in this area then occurs and spreads out dorsally along the papilla, in a more or less discus or stripe form (Plate 14.20.). Finally there is generalized hyperreflection and vessel atrophy, usually after more than a year of deficient taurine intake, and the cat is completely blind.

Diagnosis: The diagnosis can be made on the basis of the history and can eventually be confirmed by measuring the concentration of taurine in plasma. The reference values are of the order of 15-150 µmol/l. The costly measurement is seldom performed. Blood from healthy reference animals should thus also be included. In differential diagnosis consideration must be given to such abnormalities as PRA, FCRD (see 14.16) and, in the end stage, all abnormalities associated with bilateral generalized, diffuse retinal atrophy.

Therapy: Treatment consists of changing the food and if necessary adding meat, fish, shellfish, or taurine in powder form. The degeneration already present is irreversible but the process will, however, be stopped.

To prevent the disease, owners should be made to realize that cats are emphatically carnivores and should be fed a diet that is appropriate to this.