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Ophthalmology

Ocular Emergencies in Small Animals
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TRAUMATIC ORBITAL DISEASES

Proptosis is forward displacement of the eye from the orbit. It is seen commonly with retrobulbar hemorrhage and edema following trauma. Often, there is sufficient trauma to cause stretching and/or tearing of extraocular muscles. Proptosis occurs fairly commonly in dogs (especially brachycephalics). Proptosed eyes should be evaluated for hyphema (blood inside the eye), pupil size, pupillary light responses, extraocular muscle damage, and duration of proptosis. All proptosed eyes should be fluorescein stained to evaluate for corneal ulceration. If there are favorable prognostic indicators (absence of hyphema, short duration, muscles intact, miotic pupil) the eye should be replaced. Most proptosed eyes do not regain vision unfortunately.

Keep the eye moist pre-surgically; this is something the owner can do on the way to the emergency clinic. Gently remove debris with copious sterile saline flushes. If the animal is stable enough to handle a short general anesthetic episode, replace the eye, performing a lateral canthotomy if necessary. A complete temporary tarsorrhaphy is performed and the sutures are kept in place 1-3 weeks (until lid tension is minimal). Replace the suture if lagophthalmos is present. Traumatic strabismus (usually esotropia or lateral deviation) may be corrected if it still present after 6-8 weeks. Maintain medical treatment with systemic antibiotics, topical antibiotic ointment and atropine. Systemic anti-inflammatory drugs may be used to help decrease severe swelling of the periocular tissue.

ANTERIOR UVEITIS

Infectious uveitis is common to cats (FIP, FIV, FELV, herpes, toxoplasmosis, cryptococcus), can be secondary to corneal or scleral disease, and can be immune-mediated as in lens-induced uveitis (LIU). It may be caused by systemic diseases and trauma. Clinical signs include enophthalmia, prolapsed nictitans, hyperemia of conjunctiva, corneal edema, keratic precipitates, aqueous flare in anterior chamber, hyphema or hypopyon, iris is swollen. Miosis, iris color change and rubeosis iris and synechiae with hypotony. Treatment of anterior uveitis includes topical atropine, steroids, NSAIDs and antibiotics. Complications of uveitis include persistence, leukomas, iris bombe, endothelial degeneration, cataracts, phthisis and glaucoma.

HYPHEMA is blood in anterior chamber. THIS INDICATES UVEITIS!!!
CATARACTS

Strictly defined, a cataract is any opacity of the lens or its capsule. It may be congenital, inherited, or caused by disease, toxicity, trauma, or age. Many purebred dogs are predisposed to developing cataracts. Cataracts and nuclear sclerosis are both associated with advancing age. Nuclear sclerosis is a normal lenticular alteration in most dogs over 6 years. Nuclear sclerosis is not a true cataract, and there is no pathologic alteration of the lens fiber pattern.

Another common cause of cataracts in dogs is diabetes mellitus. Initially, diabetic cataracts begin as equatorial cortical vacuoles but rapidly (weeks to months) progress to form complete or resorbing cataracts. Advanced cataracts produce vision loss. In comparison, focal incomplete cataracts have varying degrees of blindness.

If the cataracts are mature, clues such as careful assessment of the history, the pupillary light reflex and dazzle response, in addition to ocular ultrasonography and ERG, may be required to rule out concurrent retinal disease.

Terms frequently used to describe cataract severity include: immature or incomplete, mature or complete, and hypermature or resorbing cataracts. By convention, the degree of completeness of a cataract is related to the amount or percentage of tapetal reflection that it blocks. Hypermature cataracts are often associated with a deep anterior chamber, wrinkled anterior lens capsule, and signs of uveitis. An incipient cataract is synonymous with an early cataract. An intumescent cataract describes a lens that has "swollen" and enlarged due to an imbibition of fluid. The lens can actually swell enough to alter aqueous outflow dynamics and increase intraocular pressure (IOP).

DIABETES MELLITUS

There is a high incidence of cataracts in diabetic dogs, with many cataracts apparently developing rapidly over days to weeks. Most diabetic dogs form cataracts within 2.5 years after diagnosis. KCS, slow corneal epithelial healing, Horner’s, stromal abscesses and canine fungal keratitis are also found in diabetic dogs. Early cataractous changes appear as vacuoles in the subepithelial equatorial cortex which progress to mature, intumescent cataractous lenses with prominent Y-suture clefting. Cataracts occur much less frequently in diabetic cats as the diabetic cat lens contains less aldose reductase activity. Anterior uveitis is also found in some canine diabetics. Diabetic retinopathy is slow to develop in diabetic dogs and cats. Treatment can be divided into the acute management of diabetic ketoacidosis and the stabilisation of the uncomplicated diabetic. Surgery is necessary to treat the cataracts.

LENS LUXATION AND SUBLUXATION

Luxation is when the lens is totally free of zonular attachments. Subluxation is when the lens is only partially freed from its zonular attachments and remains in the patellar fossa of the vitreous face. This is common in terriers. Management of lens luxation depends on whether the animal is painful in the affected eye or not, and whether the animal has the ability to see out of this eye or not.

CANINE AND FELINE GLAUCOMAS

Aqueous humor is produced in the ciliary body by active secretion and ultrafiltration of plasma. The enzyme carbonic anhydrase participates in the energy-dependent secretory phase of aqueous production. Most of the aqueous humor flows from the posterior chamber, through the pupil, to the anterior chamber, and exits at the iridocorneal angle into the intrascleral venous plexus. A small percentage of the
outflow in dogs and cats (uveosclerol or nonconventional) also exits through the iris,
ciliary body, choroid, and sclera. The balance between formation and drainage of
aqueous humor maintains intraocular pressure (IOP) within a normal range of
approximately 15 to 25 mm Hg.

By definition, glaucoma is increased IOP with associated visual deficits. In most
cases in dogs and cats, glaucoma is caused by obstruction or stenosis of the aqueous
humor outflow pathways. It remains a challenge to the veterinarian to detect the
early subtle disturbances of glaucoma and to effectively treat this condition. Delayed
or inadequate therapy can lead to irreversible blindness and a painful, cosmetically
unacceptable eye.

All ocular tissues are eventually affected by the elevated IOP. The presence,
individually or as a group, of a "red eye," corneal edema, mydriasis, blepharospasm,
blindness, and buphthalmos can be explained by the increased IOP. If the IOP
cannot be reduced, an overall increase in the size of the globe may result
(buphthalmos). This change may occur more rapidly in young dogs and cats.
Ruptures of the cornea's inner limiting (Descemet's) membrane may accompany the
elevated corneal tension and buphthalmos to produce multiple, linear corneal striae.
Persistent corneal endothelial damage can result in corneal edema. Buphthalmos
causes increased tension on the lens zonules. Zonular disinsertion results in lens
subluxation or luxation.

Pupillary light reflexes may be normal, slow, or absent in early glaucoma, depending
on the functional status of the iris sphincter muscle, retina, and optic nerve. Acute
elevation of IOP (greater than 45 mm Hg) causes paralysis of the iris sphincter and
dilator muscles. Prolonged or recurrent elevations of IOP lead to degeneration of the
retina and optic nerve, with excavation or cupping of the optic nerve head.

Primary glaucoma in dogs is a breed-related, hereditary condition. Predisposition to
primary open-angle glaucoma in the Persian and Siamese cat breeds has also been
noted, but in the author's experience, domestic short-hairs are more often affected.
In both dogs and cats, affected animals may present with only one eye involved, but
the risk is very high for development of glaucoma in the other eye.

Secondary glaucoma is more commonly encountered than primary glaucoma in dogs
and cats. The elevated IOP results from other disease processes within the eye. The
glaucoma may be open or closed angle, and in some instances is associated with
pupillary block. The condition tends to be unilateral without an inherited basis.

The presentation of a patient with a painful, red eye requires that glaucoma be ruled
out among the possible diagnoses of conjunctivitis, uveitis, or keratitis. Pain
manifested as depression, anorexia, rubbing at the eye, and squinting is common.
Congestion of episcleral vessels, diffuse corneal edema, a fixed and dilated pupil, and
blindness will occur as the IOP increases. The onset of clinical signs in cats is often
insidious, as cats are less likely to demonstrate the acute intense corneal edema and
episcleral congestion exhibited in dogs. Signs of chronic glaucoma are dramatic.
They include combinations of the early signs with buphthalmos, lagophthalmos,
exposure keratitis, luxated lens, corneal striae, optic nerve atrophy with cupping,
and retinal atrophy.

IOP must be accurately measured to diagnose glaucoma. The normal canine and
feline IOP is 15 to 25 mm Hg. An IOP greater than 30 mm Hg is considered
pathologic and diagnostic for this condition. It is possible to crudely evaluate IOP digitally if the IOP is very high or low, but this is not satisfactory to evaluate clinical response to therapy. The Schiotz’s indentation tonometer allows the practitioner to diagnose and evaluate treatment in small animals with glaucoma. The human Schiotz table is accurate for the dog. The Tonopen applanation tonometer has made it much easier to diagnose and treat the animal glaucoma.

Medical therapy is the treatment of choice in animals with a history of acute primary or secondary glaucoma. Treatment should be instituted to reduce the IOP as soon as possible to alleviate pain and preserve vision. Animals presented with a history and clinical signs of chronic glaucoma should be considered for medical and surgical therapy. The iridocorneal angle gradually closes in most types of glaucoma and the initially effective treatment becomes inadequate. Surgery is the only option available when vision continues to diminish in spite of maximum medical therapy.

Multiple drug therapy to decrease IOP by reducing production of aqueous humor and diminishing the resistance to aqueous humor outflow is the most effective approach. Treatment of the ocularly normotensive eye in a purebred dog with apparently unilateral glaucoma can delay the onset of overt ocular hypertension in the second eye a median of 30 months. Betaxolol and demecarium were each effective at delaying onset of glaucoma in dogs when administered topically. Carbonic-anhydrase inhibitors reduce ciliary-body production of aqueous humor independent of diuresis. These drugs can cause metabolic acidosis, and the dosage should be carefully adjusted to minimize side effects, which include panting, nausea, and vomiting. Non-carbonic anhydrase-inhibiting diuretics do not significantly reduce IOP! Topical parasympathomimetic drugs act primarily to cause ciliary muscle contraction, increasing the outflow of aqueous humor. This action is independent of their effect on the iris sphincter muscle. Parasympathomimetics are contraindicated in glaucoma associated with anterior uveitis. They should be used with caution in glaucoma associated with anterior lens luxations. Sympathomimetic drugs reduce IOP by increasing production of aqueous humor and increasing outflow. These drugs are most effective in reducing IOP when combined with parasympathomimetics. β-adrenergic antagonists decrease production of aqueous humor, but the specific mechanism of action is not known. The ocular hypotensive effects are additive to those of carbonic-anhydrase inhibitors and parasympathomimetics.

Oral and intravenous hyperosmotic agents lower IOP rapidly by osmotically reducing the volume of the vitreous. They are used in the emergency treatment of acute glaucoma but are ineffective or impractical for long-term or maintenance therapy.

Surgical procedures are divided into those that increase aqueous humor outflow and those that decrease aqueous humor production. Surgery should be considered when the IOP cannot be controlled medically, especially when vision is still present. Anteriorly luxated lenses should be removed in functioning eyes to relieve pupillary block and prevent corneal damage due to the lens touching the corneal endothelium. Cyclocryotherapy has been found to be effective in decreasing production of aqueous humor by the transcleral freezing of the ciliary body with nitrous oxide. This may require repeated applications for optimal IOP control. The YAG laser is preferred over nitrous oxide by the author to cause ciliary body necrosis (cyclophotocoagulation). The eye is less irritated postoperatively and the IOP stays low for longer periods of time. Only 6% of dogs with cyclophotocoagulation will still be visual at one year after installation. Gonioimplants are available to passively shunt aqueous humor to the subconjunctival space. They tend to fail by fibrosing shut in dogs. Only 18% of
dogs with gonioimplants will still be visual at one year after installation. Enucleation or evisceration with prosthetic silicone implants is indicated when vision is lost in uncontrolled glaucoma. The source of pain is removed, and no further medication is necessary. The cosmetic appearance of the prosthetic implant is sometimes preferred to that of enucleation. Prosthetic implants should not be used when glaucoma is or may be associated with intraocular infection or neoplasia. The use of intraocular silicone prosthesis (ISP) implants have been used successfully in cats with buphthalmos and absolute glaucoma.

HEMORRHAGIC RETINOPATHY is seen associated with anemia, and systemic hypertension (Treatment: Salt restricting diets; amlodipine 0.625 mg/10 lbs BW PO SID), coagulopathies, and systemic infections. The cat has an apparently unique predisposition to the development of small multifocal areas of intra and preretinal hemorrhage associated with severe anemia due to a variety of causes. In one study, retinal hemorrhages were observed in 20 of 26 cats with anemia (hemoglobin values less than 5 gm per 100 ml). Retinal hemorrhages were bilateral and occurred in both the tapetal and nontapetal zones. Other causes include lympho-sarcoma, reticuloendotheliosis with thrombocytopenia, chronic bleeding from a duodenal ulcer, FIP, and hemobartonellosis. Functional vision is not usually affected and treatment is normally directed against the primary cause of anemia.

RETINAL DETACHMENTS are rhegmatogenous (due to holes in retina which allow vitreous to "flow" into the subretinal space) or non-rhegmatogenous (no retinal holes). Most are due to "holes" or tears of the retina in dogs and humans. Retinal detachments associated with systemic hypertension due to chronic renal failure are common in older cats. They often respond to amlodipine and dietary adjustment. (amlodipine 0.625 mg/10 lbs BW PO SID).

OPTIC NEURITIS
Sudden blindness, with pupils fixed and dilated can be caused by optic neuritis. Causes include multiple viral infections (distemper) systemic mycosis (blastomyces, cryptococcus) neoplasia, CNS reticulosis or GME, trauma, and idiopathic.

REFERENCE