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GLAUCOMA – THE UPDATES

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Glaucoma is a group of conditions that leads to an impairment of aqueous humor outflow that results in an intraocular pressure detrimental to the health of the eye. **Glaucoma is a leading cause of blindness in dogs.** In many instances of glaucoma in dogs the IOP can become very elevated putting vision at immediate risk and making the condition an ocular emergency; urgent referral to a veterinary ophthalmologist is recommended. Unfortunately glaucoma can be difficult to manage successfully. It is a differential diagnosis for a "red eye" or "watery eye".

Investigation of Glaucoma

Measurement of intraocular pressure (IOP) and gonioscopy are important parts of the examination of an eye with glaucoma. Applanation tonometry (Tonopen) or rebound tonometry (Tonovet) are becoming widely available in general practices, making precise measurement of IOP readily available. The techniques require some practice and care should be taken to avoid tight restraint around the animal's neck as this may result in an artificially high IOP reading. The normal range of IOPs in dogs is between approximately 7 and 24 mmHg.

Classification of Glaucoma

Glaucoma is classified as primary (due to a primary abnormality in the aqueous drainage pathways) or secondary (to other ocular disease).

Primary Glaucoma

Primary glaucoma is rare in the cat, but common in the dog. It is divided into those forms where there is a preceding gross abnormality in the development of the iridocorneal angle that can be detected by gonioscopy and those where the opening into the ciliary cleft (iridocorneal angle) appears normal.

- **Narrow angle and goniodysgenesis.** These are the commonest forms of primary glaucoma in dogs. The drainage angle is abnormally formed. In narrow angle there is an abnormally narrow opening into the ciliary cleft. With goniodysgenesis the opening into the ciliary cleft is spanned by an abnormally differentiated pectinate ligament. The malformation of the drainage apparatus is heritable and the more severely affected animals will develop glaucoma. Other, poorly understood factors also play a role because although these abnormalities are present after ocular maturation, glaucoma does not develop until later in age (often middle age). There is a suggestion that further narrowing of the angle may occur with age. Screening dogs of at risk breeds can be performed to identify individuals with the predisposing

anatomical abnormalities. Many breeds of dog are affected including Cocker spaniel, the Basset hound, arctic circle breeds (Samoyed, Siberian husky, Norwegian elkhound), Bouvier, terrier breeds, miniature poodle, Chow chow, Shar pei.
This form of glaucoma typically has a very acute onset and results in very marked increases in IOP (above 60mmHg is common).

- **Open angle glaucoma.** This is the commonest form of glaucoma in man but is uncommon in dogs. It typically has a slow and insidious onset with globe enlargement and accompanying changes (splits in Descemet's membrane of the cornea and lens subluxation) as the first outward sign of the disease. Initially the IOP is only slightly to moderately elevated. As the disease progresses more severe elevations of IOP with accompanying pain may develop.

Secondary Glaucoma
Secondary glaucoma occurs as a complication of, or sequel to, other ocular disease or injury. Any disease process can interfere with aqueous outflow and result in secondary glaucoma. Causes include:
- **Primary lens luxation** Anterior luxation of the lens accompanied by vitreous prolapse leads to pupil block and glaucoma. Typically acute-onset, require emergency medical reduction in IOP followed by lens extraction.
- **Iridocyclitis**. Chronic anterior uveal inflammation can lead to secondary glaucoma by either extensive posterior synechiae (iris to lens adhesions) and pupil block, or impairment of aqueous passage through the drainage apparatus.
- **Trauma**
- **Hyphema**. Bleeding into the anterior chamber (particularly if recurrent) can lead to impairment of aqueous drainage and secondary glaucoma.
- **Retinal detachment**. Can lead to development of a pre-iridal fibrovascular membrane that can obstruct the drainage angle.
- **Neoplasia**. Infiltrating the drainage angle.
- **Ocular melanosis**. This condition is an hereditary condition in cairn terriers. Proliferation of melanocytes within the eye eventually leads to impairment of aqueous drainage. Glaucoma has an insidious onset and leads to globe enlargement prior to eventual loss of vision. It is difficult to control.

CLINICAL SIGNS OF GLAUCOMA
The clinical signs of glaucoma vary considerably depending on the etiology, the duration and the extent of pressure rise.
Acute Glaucoma with Grossly Elevated IOP— typical signs with primary narrow angle or goniodysgenesis glaucoma or secondary to lens luxation include:
- **severe pain** (blepharospasm, photophobia, enophthalmos, elevation of the nictitating membrane, diffuse facial pain--trigeminal neuralgia – winces if head touched), slight epiphora. Altered behavior (hiding, off food). The signs of severe pain reduce a few days after onset.
- **episcleral and conjunctival vascular injection** ("Red-Eye") occurs due to blockage of normal venous drainage and engorgement of anterior ciliary veins.

- **corneal edema** (reversible) due to increased hydrostatic pressure which exceeds the endothelium's capacity to pump fluid out of the stroma
- **pupillary dilation** - Dilation or unresponsiveness to light stimulation doesn't occur until the IOP is about 40 mmHg or higher. Pupil is typically mid-dilated in the acute narrow angle/goniodysgenesis glaucomas. May be constricted in some secondary glaucomas
- **loss of vision** As the pressure rises above the 40s vision is reduced and then lost – in most cases this is initially reversible with very prompt emergency treatment.

Early stages of glaucoma with only moderately elevated IOP – open angle glaucoma and many secondary glaucomas fall into this category. At this stage pain is not an obvious feature and vision loss is not yet significant. There may be a mild episcleral injection. The most obvious sign will be globe enlargement. However this will not be apparent until the IOP has been elevated for some time.

Chronic Glaucoma—Primary open angle glaucoma and some secondary glaucomas may first present when they already have some chronic changes.

- **Buphthalmos** - globe enlargement; occurs more quickly in young animals. If this has developed in an eye with narrow angle glaucoma or goniodysgenesis the eye will probably be blind. Young animals and those with primary open angle glaucoma and glaucoma due to ocular melanosis may still have useful vision at the time when buphthalmos has developed.
- **Corneal changes**. Within a few days of development of severe IOP rises deep corneal vascularization may develop. More chronic changes include: **exposure keratitis** (may occur if the animal is unable to blink over the buphthalmic globe); **corneal** (Haab's) **striae** - gray, linear streaks in the cornea due to breaks in Descemet's membrane.
- **scleral thinning**
- **decreased evidence of pain** - but condition remains painful
- **episcleral vascular engorgement**
- **subluxated/luxated lens** - zonular breakdown, vitreal degeneration with loss of support for lens; subsequent cataract development
- **iris degeneration**
- **ciliary body degeneration** (can eventually result in phthisis bulbi)
- **cataract**
- **funduscopic lesions**
    - **optic atrophy & cupped optic disc** - posterior bowing of the lamina cribrosa
    - **retinal degeneration** - thinning of the retina, increased tapetal reflectivity and retinal vascular attenuation

Management of Glaucoma

In deciding on the best approach to managing a case of glaucoma the cause of the glaucoma must be identified. For example glaucoma secondary to lens luxation requires a different approach to say glaucoma due to an intraocular tumor or a case of primary glaucoma.

Medical Management

Several classes of drug are available to lower intraocular pressure but not all are effective in all species. A combination of several drugs may be used.

Prostaglandin analogues

e.g. latanoprost, travaprost and bimatoprost Cause a profound reduction in the production of aqueous. Use for emergency reduction in IOP in dogs, not effective in cats. For maintenance use q12-q24hr. Cause significant miosis which may exacerbate underlying uveitis – also may increase risk of pupil block.

Osmotic Diuretics

Emergency reduction in IOP - increase the osmotic gradient between plasma and the ocular cavities, dehydrating the vitreous. Less commonly used since prostaglandin analogues became available. E.g. **Mannitol IV 20%** 1-2 g/kg IV over 15-20 minutes. **Glycerol**, 50% oral solution 1 to 2 ml/kg.

Carbonic Anhydrase Inhibitors (CAIs)

Decrease aqueous humor production usually applied topically (e.g. dorzolamide, brinzolamide). Useful for longer-term control.

Beta blockers

Timolol Betaxolol Levobunolol – reduce aqueous formation.

Miotics (applied topically)

Increase aqueous humor outflow. Direct (pilocarpine) and indirect (the cholinesterases such as: demecarium bromide, phospholine) acting parasympathomimetics.

Surgical Management

Cyclodestructive procedures

- Cyclocryotherapy
- Laser therapy (Transcleral diode laser, endolaser)

Alternative drainage pathways

- Aqueous drainage implants

Other surgical options for blind painful eyes

- Enucleation
- Evisceration with intrascleral prosthesis