

Glaucoma

Glaucoma is a leading cause of vision loss in dogs. It is one of the most often missed and under diagnosed vision threatening diseases of dogs, cats, and horses. Unfortunately, glaucoma in its early stages resembles standard conjunctivitis, uveitis and corneal epithelial ulcers. As a result, when owners initial present a dog with glaucoma, the disorder is often misdiagnosed as one of these other problems.

Pathophysiology of glaucoma. Intraocular fluid is the eye's internal blood. It produces aqueous humor as a filtration of blood and uses this fluid to nourish intraocular structures, namely the lens and cornea and inner retina, as well as maintain the shape of the globe which is critical for normal optics and vision.

Aqueous humor is produced by the epithelial cells that line the inside of the ciliary body at the equator of the eye. The fluid is generated as an ultrafiltration of blood (driven by blood pressure) and by active secretion. Similar to fluid movement in the kidneys, the active secretion of fluid by the ciliary epithelial cells into the eye requires the enzyme carbonic anhydrase. Blocking this enzyme is one the main stays of medical therapy.

Aqueous humor drains from the eye via the iridocorneal angle. This is the junction of the base of the iris with the cornea/sclera. The fluid drains through a series of canals and ducts to the venous system.

Aqueous humor is constantly produced and drained form the eye at a relatively steady rate. This results in maintenance of intraocular pressure with a range of 8 to 22 mmHg in normal animals. Glaucoma or increased intraocular pressure occurs when the outflow of aqueous humor is decreased. Unfortunately, production does not decrease resulting in the accumulation of fluid within the eye and elevation of intraocular pressure.

There are two general types of primary glaucoma, open angle and closed angle. In open angle glaucoma the anatomical structure of the iridocorneal angle is near normal, yet the rate of fluid egress is diminished. The cause of the decreased rate of egress is unclear. In closed angle, the iridocorneal angle is physically closed. However, this physical change alone is likely not the only cause of decreased fluid drainage. Clinically, open angle glaucoma is characterized by slow progressive increases in pressure and a slow loss of vision. This is the primary form of glaucoma in humans. Dogs typically have closed angle glaucoma. Changes in iris position (such as dilation of the pupil at night) can cause a sudden and severe decrease in aqueous humor outflow and rapid severe rise in intraocular pressure. Pressures can rise from 15 mmHg to 60 mmHg in 30 to 60 minutes. Such rapid and high elevations in pressure cause extreme pain as well as rapid retinal damage and permanent blindness.

Glaucoma causes two main problems: Pain and loss of vision. Our goal in treating the disease is to prevent and resolve both of these problems.

Signalment, History, and presenting clinical changes. Primary glaucoma is by far the most common form and is an inherited disease in many dog species.

Signalment. Any breed of dog can develop primary glaucoma. However, the disease is much more prevalent in certain breeds. The following breeds represent the most common presented to our hospital with primary glaucoma.

Breed	Age of onset	Rate of progression to vision loss	Other factors
American Cocker spaniel	3 to 8 years of age	Rapid - days. Often blind by the time buphthalmia occurs	OS often first eye and females more common
Basset Hounds	Any age	Rapid, but mildly tolerant of buphthalmia and can be buphthalmic and visual	Marked intraocular flare and sometimes miosis - look like uveitis dogs. Third eyelid elevation earliest sign
Siberian Huskies/ Malamutes	Often young - 0.5 to 6 years	Rapid - days to weeks	Change in iris color in blue eyed dogs
Labrador Retrievers	often older 8 to 12 years of age	Rapid - days	Often extremely painful
Poodles	often older 8 to 12 years of age	Moderate - weeks often	Glaucoma secondary to cataracts or age related lens luxation more common than primary
Cockapoos	Same as cockers		
Shar peis	3 to 8 years of age	Slow - often lose vision over months. Will maintain vision even with significant buphthalmia.	Rarely present for pain, just enlarged blue eye
Chow Chows	3 to 8 years of age	Slow - often lose vision over months. Will maintain vision even with significant buphthalmia.	Rarely present for pain, just enlarged blue eye
Beagles	3 to 8 years of age	Slow. Have a form of open angle glaucoma. Lose vision over many months	Present with bilateral buphthalmia and still have vision
Dalmatians	3 to 8 years of age	Rapid - vision loss in days	Very painful

Shih Tzus/Lhaso Apsos	often older 8 to 12 years of age	Slow - often lose vision over months. Moderately responsive to medications	Minimal pain, present for dilated pupil or red eye
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German Shepherds	3 to 8 years	Moderate. Lose vision over months	May present with a lens luxation secondary to globe enlargement
Terriers - especially Jack Russel terriers	3 to 8 years	Rapid.	May present with lens luxation although glaucoma is primary disease
Flat Coat retrievers	Any age	Rapid	
Rhodesian Ridgeback	Older dogs - 8 yo 12 years	Rapid	Often quite painful
Greyhounds			
Norwegian Elkhounds	Any age	Moderate - lose vision over weeks to months	Can retain vision while buphthalmic

Age. Glaucoma is more common as a rule in middle aged to older dogs. However, Siberian huskies (and other Arctic circle breed dogs), Bassett hounds, and flat coat retrievers can develop glaucoma early in life, within the first 6 months.

History. The early signs of glaucoma are often not recognized by owners or veterinarians as serious. Unfortunately, the best hope for preserving vision is to recognize the early signs of glaucoma, before the onset of the congestive or persistently high pressure stage.

Owners often not the following changes: **Keep in mind that most pressure elevations occur in the evening and at night so owners may observe these changes transiently in the evening or first thing in the morning.**

- Elevated third eyelid. Transient elevations of the third eyelid secondary to the animal pulling its eye back into the orbit due to discomfort is often an early warning sign that the pressure is elevated.
- Lethargy or decreased appetite. The eye hurts, they have a headache
- Pacing or the inability to settle down at night, or frequently getting up at night. the eye hurts
- Red eye. Non- specific and NOT ALL DOGS WITH ELEVATED PRESSURE HAVE A RED EYE.

- Mydriasis. Dilated pupil is often an early sign of increased pressure, especially in cats. Beware, dogs with significant blood aqueous barrier breakdown with their glaucoma such as Bassett hound and Huskies may actually have miotic pupils.

- Corneal edema/Cloudiness. Often occurs in the congestive stage.

- Decreased vision - RARELY ever noted unless the first eye is blind and the second eye is developing glaucoma.

- Globe enlargement. This can come and go initially.

- Rubbing at the eye - not a common sign

- Vomiting - nausea from a severe headache.

Exam Findings. There are 4 main stages of glaucoma. The clinical exam findings vary with each stage.

Acute Non-congestive glaucoma. This is the stage we want to detect glaucoma, before severe damage is present. This stage is characterized by mild and intermittent elevations in intraocular pressure.

- Pain: Intermittent pain when pressure is elevated. Often signs are present at night or first thing in the morning. Blepharospasm, elevated third eyelid and enophthalmia, lethargy. Restless at night, can't sleep.

- Mydriasis. Often marked mydriasis - NOTE: some dogs have

- Red Eye. Very nonspecific and not always present. But all red eyes need an IOP check. Some dogs, especially Bassett hounds and Huskies may present with significant flare and signs of uveitis due to break down of the blood aqueous barrier during pressure increases.

- Corneal Edema - intermittent edema , often above IOP of 35 mmHg

- Buphthalmia - globe NOT enlarged in this stage

- Vision. Vision normal except during times of elevated pressure when vision is reduced or absent. returns when pressure decreases.

- Posterior chamber and posterior segment changes. Often few to mild optic nerve head pallor and cupping. Lens subluxation with slowly progressive forms of glaucoma.

Acute Congestive glaucoma. In this stage the intraocular pressure increases and stays increased. It is critical in this stage to lower pressure immediately. Vision loss occurs rapidly at this stage (days).

- Pain: Marked pain when pressure is elevated. Often signs are worse at night or first thing in the morning but persist. Blepharospasm, elevated third eyelid and enophthalmia, lethargy. Restless at night, can't sleep.
- Mydriasis. pupil dilation common. HOWEVER, miosis due to secondary uveitis especially in Bassett hounds and Huskies.
- Red Eye. Common - may have marked chemosis or conjunctival swelling. nonspecific and not always present. But all red eyes need an IOP check.
- Corneal Edema -persistent edema that varies in severity with level of pressure - steamy eye
- Buphthalmia - globe NOT enlarged to early buphthalmia.
- Vision. Vision often absent. HOWEVER, rapid decrease in pressure can result in return of vision. Some permanent loss of vision will be present.
- Posterior chamber and posterior segment changes. Marked decreased blood flow to optic nerve head, peripapillary retinal edema, mild to moderate optic nerve head pallor and cupping. Lens subluxation with slowly progressive forms of glaucoma.

Chronic Congestive glaucoma. In this stage the intraocular pressure has been elevated for days to weeks to months. The globe is enlarged, vision is almost always lost, and the pain is less, but still present.

- Pain: Mild to moderate pain. But, less clinical signs. rarely is there squinting or third eyelid elevation. But lethargy and headache are common. Restless at night, can't sleep.
- Mydriasis. pupil dilation common.
- Red Eye. Variable. Scleral venule dilation less prominent and redness less than in acute congestive stage
- Corneal Edema - Edema is often less that with acute congestive form.
- Buphthalmia - globe obviously enlarged in this stage
- Vision. Blind. With the exception of a few breeds such as Shar peis, chows, beagles, and shih tzus, there is end stage optic nerve and retinal degeneration.
- Posterior chamber and posterior segment changes. Peripapillary to complete retinal degeneration. often segmental in shape. Significant optic nerve pallor and cupping. Lens subluxation present due to marked buphthalmia.

End Stage glaucoma. In this stage the intraocular pressure often has decreased due to ciliary body atrophy from hypoxia.

- Pain: Mild to no pain. Pain is directly related to pressure and as the pressure decreases so does the pain.
- Mydriasis. pupil dilation common due to complete retinal degeneration and iris sphincter muscle atrophy.
- Red Eye. Often not red
- Corneal Edema - Edema is often present due to endothelial degeneration. Lowering the pressure does not resolve the edema
- Buphthalmia - variable. Initially enlarged but phthisis bulbi can eventually occur.
- Vision. Blind.
- Posterior chamber and posterior segment changes. Peripapillary to complete retinal degeneration. often segmental in shape. Significant optic nerve pallor and cupping. Lens subluxation present due to marked buphthalmia.

Treatment. There are two goals in treating glaucoma: Preserve Vision and eliminate pain. Both objectives require lowering the intraocular pressure. The urgency and aggressiveness of therapy is determined by the stage of the glaucoma. Acute non-congestive and acute congestive require immediate therapy as vision maybe saved. Chronic and end stage glaucoma also require therapy, but less aggressive therapy is acceptable as vision has been lost.

REMEMBER: GLAUCOMA IS A MEDICAL EMERGENCY BUT A SURGICAL DISEASE.

Pain Management. Acute glaucoma is extremely painful. Can even cause a patient to present with vomiting due to nausea form pain. Sedate with narcotic (NOT Alpha 2 antagonists like metadomidine). This decreases pain, makes patient easier to treat, and can lower IOP by lowering blood pressure.

Acutely lower intraocular pressure. When an animal presents with acute glaucoma, the following initial therapy is recommended.

- Xalatan OR Travatan - topical PGF2 alfa - 1 drop in the affected eye. Lowers pressure in 1 to 2 hours.
- Azopt, Trusopt, or Cosopt - Azopt and trusopt are topical carbonic anhydrase inhibitors. Cosopt is trusopt plus timolol. 1 drop every 15 minutes for 8 treatments or until IOP is less than 25 mmHg.
- Methazolamide (oral carbonic anhydrase inhibitor) 1 mg/lb. DO NOT USE DIAMOX (Acetylzolamide) as it often causes provide acidosis.

- Recheck IOP in 2 hours (15 minutes after last drop) - If IOP < 30 mmHg go to maintenance medications. If still elevated and not coming down - go to paracentesis
- Intravenous Mannitol - 0.25 to 0.5 grams/lb slow IV. If the above eye drops do not lower IOP in 90 to 120 minutes, then Mannitol can be helpful. BUT IF TOPICAL XALATAN OR TRAVATAN DID NOT WORK IT IS RARE FOR MANNITOL OR GLYCERIN TO WORK
- Aqueous paracentesis - removal of fluid from the anterior chamber. ONLY PERFORM IF YOU ARE COMFORTABLE WITH THIS PROCEDURE. Severe intraocular damage can occur if the iris or lens are cut. Use 27 to 30 ga needle. Let the hub fill then remove.

Long Term control of intraocular pressure in eyes with the potential vision. To help maintain a lower pressure in a visual eye.

- Laser cycloablation surgery. Medical therapy is helpful to lower intraocular pressure and provide short term control of glaucoma (days to weeks, occasionally months). However, medical therapy rarely controls glaucoma long term. Many owners elect medical therapy then decide weeks later to pursue laser surgery. However, they are often disappointed that by then their pet is irreversibly blind.

Laser surgery is the best therapy for long term control of glaucoma in eyes with the potential for vision. Pressure reduction requires 4 to 6 weeks and control of intraocular pressure the first few weeks post laser surgery can be difficult. The process involves destruction of ciliary epithelial cells using a diode or YAG laser. Success rate of 90%. Risks include severe intraocular hemorrhage, posterior synechia, and cataract formation. But these complications are mute considering blindness will occur with out long term control of the glaucoma.

- Filtering device. Implantation of various tubes to allow alternative drainage of aqueous humor to peri-orbital tissue. Very effective in the short term, but all eventually fail (weeks to months). However, helpful in controlling intraocular pressure after laser surgery.
- Xalatan - topical PGF2 alfa - 1 drop in the affected eye once a day in the evening or BID. Much more effective when used in the evening.
- Azopt, Trusopt or Cosopt - carbonic anhydrase inhibitors - 1 drop TID. Can be topically irritating in some patients. Can cause nightmares in people (dogs?).
- Methazolamide - carbonic anhydrous inhibitor - 1 mg/lb BID to TID. Quite effective in cats who do not tolerate some topical medications.
- Timolol - Beta blocker - 1 drop B-TID. Well tolerated by most pets. Synergistic with other meds and in mild cases of glaucoma can lower the pressure enough on its own. Inexpensive.

Long Term control of intraocular pressure in blind eyes. To help maintain a lower pressure in a visual eye.

- Laser cycloablation surgery. While the main benefit of laser surgery is the ability to preserve vision, it can give a cosmetically near normal eye. The down side is that most patients will continue to require medical therapy after surgery.

- Enucleation with an intraorbital prosthesis. Cost effective, straight forward, low risk, immediately reduction in pain. Drawback is the loss of the cosmetic appearance of an eye.

- Evisceration with an intrascleral prosthesis. Highly successful (99%) and permanently resolves glaucoma. Drawbacks include variability in appearance. Some globes very dark and near normal in appearance, some nearly white. Most a medium grey color. Failure due to corneal ulceration or intraocular infection can occur necessitating enucleation.

- Intravitreal gentocin. Gentocin is neuroepithelial toxic and destroy ciliary epithelial cells. It also kills all retinal cells and lens epithelial cells. As a result, it will immediately blind an eye. **DO NOT USE IN A VISUAL EYE.** The main drawback is that ciliary epithelial cells can regrow and glaucoma recurs months later in 30% of patients. Furthermore, approximately 50% of patients experience marked phthisis bulbi and owners dislike the cosmetic outcome.

Pain control. Decreasing pain is best accomplished by lowering intraocular pressure. However, the use of pain medications are helpful. Systemic narcotics such as butorphanol are very effective. Systemic NSAIDs are also helpful, but must be used with caution. NSAID therapy can raise intraocular pressure. TOPICAL NSAID therapy is contraindicated in glaucoma therapy. **NEVER GIVE ATROPINE TO ANY PATIENTS WITH GLAUCOMA OR SUSPECTED OF HAVING GLAUCOMA, OR ANY RED EYE WHERE THE INTRAOCULAR PRESSURE IS UNKNOWN.** Atropine will raise intraocular pressure and greatly increase the level of pain.