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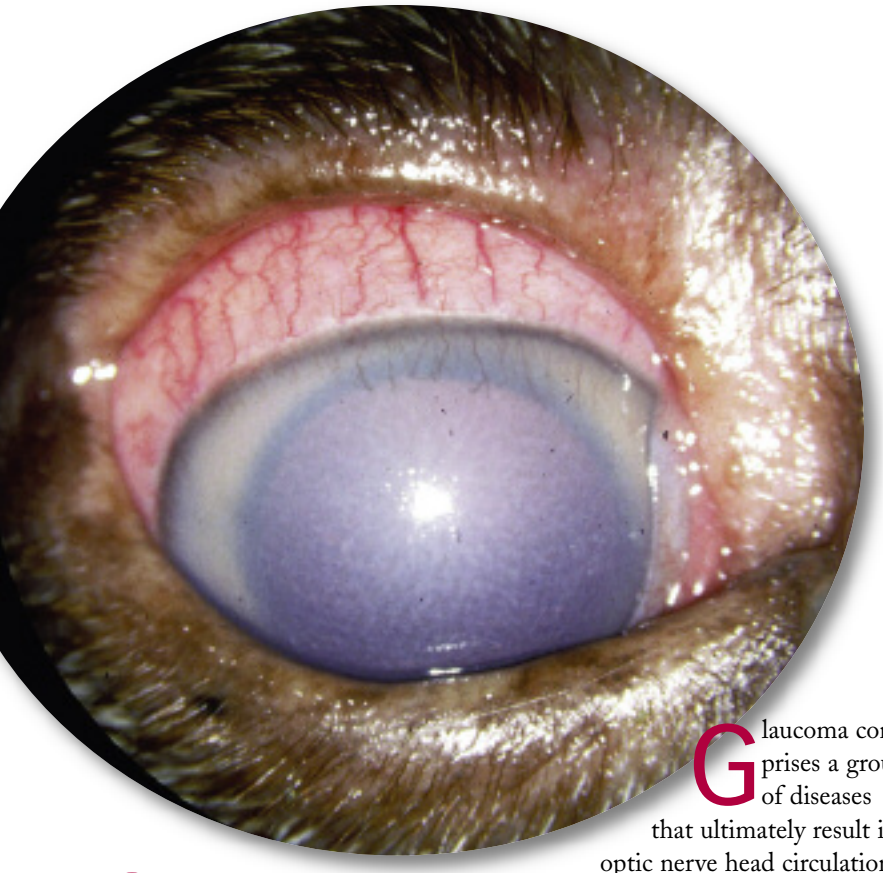
Canine Glaucoma



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An eye with acute congestive glaucoma. There is diffuse corneal edema, scleral injection, mydriasis, epiphora, corneal vascularization, and conjunctival hyperemia of the nictitans.

Glaucoma comprises a group of diseases that ultimately result in optic nerve head circulation damage, retinal ganglion cell death, and irreversible blindness.[1-3] It is a common and painful cause of blindness in dogs, affecting nearly 0.9% of purebreds in North America.[4]

LEVELS OF CARE

The goal of the practitioner should be to determine:

- Whether glaucoma is the correct diagnosis
- Whether glaucoma is primary or secondary
- Whether it is acute or chronic.

Once these determinations are made, a treatment plan can be developed.

Diagnosis

A glaucoma diagnosis is established by measuring intraocular pressure (IOP) in all red eyes. IOP varies with species, age, breed, time of day, method of restraint, tonometrist, and tonometer. Younger dogs have a higher normal IOP than older ones. The normal IOP range should be established by the tonometrist; in our practice, using a Tono-Pen (reichert.com), we consider normal IOP to be between 8 and 20 mm Hg. Routine screening of dog breeds predisposed to primary glaucoma makes purchase and use of a tonometer cost-effective.

Primary or Secondary

Primary glaucoma is caused by abnormal anatomy of the iridocorneal angle and usually occurs between 3 and 9 years of age, rarely in mixed breed dogs. Glaucoma can also be secondary to other eye diseases and conditions, including anterior lens luxation (inherited in at least 45 breeds, including most types of terriers, basset hounds, beagles, and Arctic circle breeds[2]), chronic anterior uveitis, chronic long-standing cataracts, prior cataract surgery, retinal detachment, hyphema, and intraocular neoplasia. It is not always obvious whether glaucoma is primary or secondary, but effective treatment varies depending on underlying cause.

Acute or Chronic

Clinical signs of **acute** congestive glaucoma **(Figure 1)** include diffuse corneal edema, scleral injection, mydriasis, and diminished or loss of vision. Diffuse corneal edema often makes fundic examination difficult. The eye will likely be blind if IOP is greater than 40 mm Hg and may have an inconsistent dazzle response and consensual pupil light reflex (PLR). Generally, if IOP has been greater than 50 mm Hg for more than 3 days, the potential for vision is negligible. Because it is usually unknown how long IOP has been elevated, always try to immediately decrease it.

Clinical signs of **chronic** glaucoma **(Figure 2)** include buphthalmia, Haab's striae, lens subluxation or luxation, fundic changes, including optic nerve cupping, retinal vascular attenuation, and tapetal hyperreflectivity **(Figure 3)**. Chronically affected eyes do not regain sight.

WHEN TO CONSIDER REFERRING

Time is imperative when managing and/or referring a glaucoma case. If you do not have a tonometer or are unsure of diagnosis, cause (primary or secondary), or chronicity, call a veterinary ophthalmologist for advice and/or referral. Evaluation through digital palpation of the globe alone is not acceptable. If there is good comfort level with your diagnosis and appropriate medications are accessible, then initiate treatment immediately, even prior to referral. The client must understand that surgical intervention combined with medications may be necessary to effectively manage glaucoma and that irreversible blindness may ensue, even when all appropriate interventions are employed.

THE REFERRAL PROCESS

When calling an ophthalmologist for advice or referral, start the conversation with the dog's signalment. Describe both eyes (some subtle signs may indicate a problem in the seemingly unaffected eye) and provide medications used and dose information. Inform the client of the approximate cost of an initial referral examination, which will be provided by the veterinary ophthalmologist. Typically, a referral letter is generated following the evaluation. A complete ophthalmic examination includes:

- Direct and consensual PLRs
- Schirmer's tear test
- IOP measurement
- Fluorescein staining
- Evaluation of the extraocular/intraocular structures, including slit lamp evaluation and binocular indirect ophthalmoscopy.

If medical therapy is unsuccessful, surgical options for animals that have vision or the potential for vision include traditional or endolaser diode laser cycloablation, gonioimplants or glaucoma valves, or a combination of both.

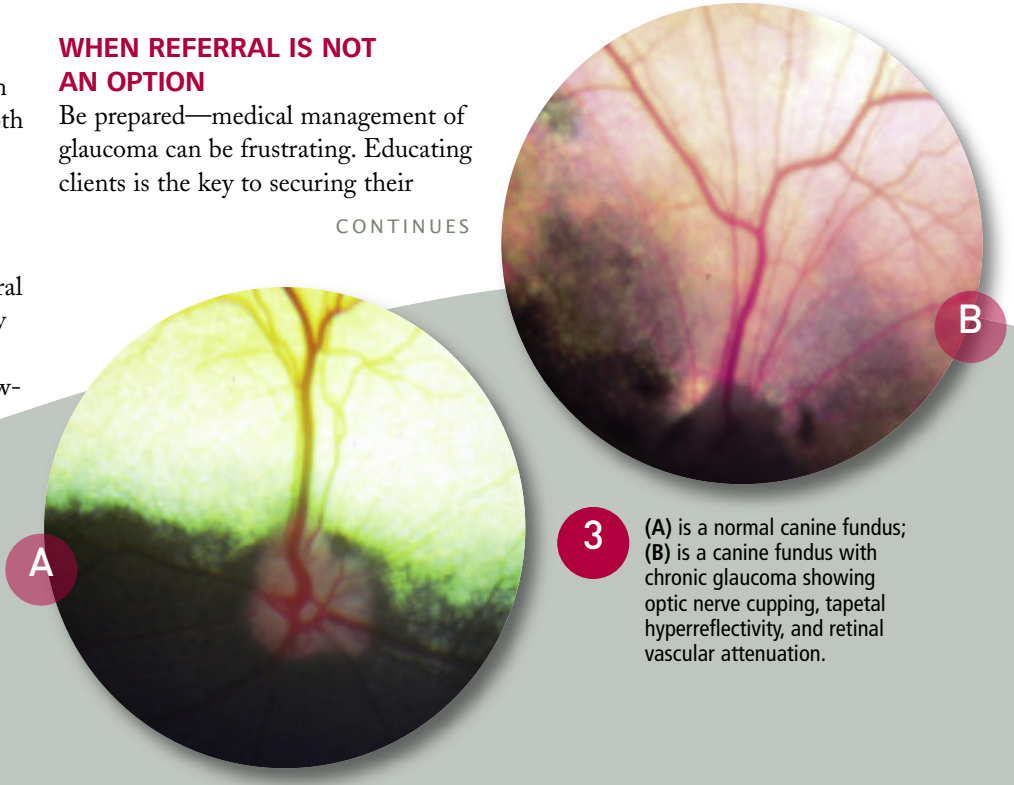
2

A husky with bilateral chronic glaucoma. Both eyes are buphthalmic, have extreme mydriasis, and the lenses are posteriorly luxated and located in the ventral vitreous.

WHEN REFERRAL IS NOT AN OPTION

Be prepared—medical management of glaucoma can be frustrating. Educating clients is the key to securing their

CONTINUES



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(A) is a normal canine fundus; **(B)** is a canine fundus with chronic glaucoma showing optic nerve cupping, tapetal hyperreflectivity, and retinal vascular attenuation.

IOP = intraocular pressure; PLR = pupil light reflex

tolerance and patience. Even after appropriate and aggressive medical management, glaucoma can progress. It can become recalcitrant to therapy if the patient is not periodically reevaluated with medications adjusted to maintain IOP in the safe range. For my well-managed glaucoma patients, a safe IOP is below 20 mm Hg. If IOP rises into the low 20s or higher and medications are at a maximum, call a veterinary ophthalmologist. Intraocular pressure in dogs does not always slowly increase over time, but pressure spikes frequently occur that can be blinding. Treating and/or preventing these spikes is of utmost importance. Monitor IOP once or twice a week for the first month to ensure that therapy is adequate.

Treatment options by stage are discussed in **Table 1**. Once IOP is consistently below 20 mm Hg for a 12- to 24-hour period, medical management **(Table 2)** is continued and rechecked periodically. If IOP does not drop significantly or is still above 30 mm Hg, it is unlikely that continued medication will decrease the IOP. If the animal is blind and IOP is above 30 mm Hg, pain is likely and a fair suggestion to the client is enucleation or ciliary/chemical ablation to alleviate discomfort. Another palliative surgical procedure is evisceration with intrascleral prosthesis placement; this procedure is usually only available through a veterinary ophthalmologist.

PAIN MANAGEMENT

Glaucoma pain results from elevated IOP and is proportional to its magnitude. *The only way to control the pain is to decrease IOP*. Nonsteroidal antiinflammatory medications are contraindicated with primary glaucoma; they can elevate IOP in canine patients and will not diminish pain. Humans with glaucoma have migraine-like headaches, nausea, vomiting, and profuse sweating. Animals with glaucoma are also in significant pain; however, their stoic natures often hide their discomfort. Tramadol may be used to help with pain; however, it has not been evaluated for this purpose in glaucoma.

See Aids & Resources, back page, for references and suggested reaading.

Table 1. Clinical Signs and Treatment of Glaucoma

Stage	Clinical Signs	Treatment Options
Early noncongestive glaucoma	IOP higher than contralateral eye by ≥ 20% (IOP between 20 and 35 mm Hg); subtle to negligible clinical signs, mild corneal edema, anisocoria with affected eye being more mydriatic	Topical and/or oral carbonic anhydrase inhibitor; suggest referral. Treat contralateral eye as well. Recheck in 2–3 days; adjust if IOP is not below 20 mm Hg.
Acute congestive glaucoma	IOP > 35 mm Hg, diffuse corneal edema, blepharospasm, (±) elevated nictitans, mydriasis, scleral injection, diminished or loss of vision	Topical prostaglandin analog,* topical and oral carbonic anhydrase inhibitor, IV prednisolone sodium succinate (Solu-Delta-Cortef, pfizerah.com) then mannitol; refer immediately, if possible.
Chronic end-stage glaucoma	Buphthalmia, Haab's striae keratitis, lens subluxation/luxation, optic nerve cupping, retinal degeneration, variable IOP, blindness, mydriasis	Enucleation, intrascleral prosthesis, or intravitreal gentocin injection. Suggest referral for surgery, if necessary.

*Contraindicated if lens is in anterior chamber or glaucoma is secondary to anterior uveitis

Table 2. Antiglaucoma Medications, Doses, & Mechanisms of Action

Medication	Dose	Mechanism of action
Methazolamide	2–5 mg/kg Q 8–12 H	Carbonic anhydrase inhibitor (CAI)
Dorzolamide, brinzolamide	1 drop Q 8 H	Carbonic anhydrase inhibitor (CAI)
Timolol maleate (do not use in cardiac patients)	1 drop Q 8–12 H	Nonspecific beta-blocker
Dorzolamide and timolol combination	1 drop Q 8 H	Combination of CAI and nonspecific beta-blocker
Latanoprost, travaprost, unoprostone isopropyl	1 drop Q 12 H	Prostaglandin analog
Prednisolone sodium succinate	15 mg/kg IV	Diminishes reperfusion injury
Mannitol	0.5 to 1.5 g/kg IV over 15–20 minutes	Dehydrates the vitreous

Some antioxidants, including coenzyme Q10 (ubiquinone), epigallocatechin gallate (green tea extract), and grapeseed extract, have been shown to support cells damaged by glaucoma (ie, trabecular meshwork, retinal ganglion cells).