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Topical Review

Glaucomas

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Canine and feline glaucomas are commonly presented as ocular emergencies. Glaucoma is a common cause of vision loss and a frustrating disorder in terms of medical and surgical treatment. Increased intraocular pressure (IOP) is a significant risk factor in the disease, leading to damage of the retina and optic nerve head. IOP measurement and gonioscopic and fundic examinations provide the instruments for diagnosis of glaucoma. The primary goal in glaucoma therapy is aimed at vision preservation. Medical treatment provides temporary relief, but alone it fails to control IOP in the long term, and surgical intervention is recommended. Surgical patient selection depends on several factors, from type and stage of glaucoma to the presence of or potential for vision. Available surgical procedures to decrease IOP consist of cyclodestructive techniques to decrease aqueous humor production and filtering techniques to increase its drainage. Even with recent surgical and medical advances, pain and blindness are still common occurrences in the disease: end-stage procedures such as enucleation, evisceration with intrascleral prosthesis, and pharmacologic ablation of ciliary bodies are then recommended to address chronic discomfort for buphthalmic and blind globes.

Introduction

Canine and feline glaucomas represent one of the most common emergencies in veterinary ophthalmology and veterinary practice.

The glaucomas are a group of diseases characterized by the neurodegenerative disorder of retinal ganglion cells and optic nerve, leading to blindness. Despite the increased complexity in the etiopathogenesis of the disease, increase in intraocular pressure (IOP) is still regarded as the main risk factor in the development of glaucoma in veterinary patients. IOP management is the main target of glaucoma treatment; however, medical and surgical therapies vary according to the cause and stage of the disease.

Owing to their common acute presentation and rapid progression, canine and feline glaucomas are one of the true ophthalmic emergencies, and prompt medical and surgical treatment is warranted.

Anatomy and Physiology

Normal IOP in dogs and cats varies according to different studies and testing devices. Currently, mean normal values for IOP in dogs are estimated at 19.2 $\pm$ 5.5 mmHg[1] and 12.9 $\pm$ 2.7 mmHg[2] (by applanation tonometry) and 10.8 $\pm$ 3.1 mmHg[2] and 9.1 $\pm$ 3.4 mmHg[3] (by rebound tonometry). In cats, mean reported values of normal IOP are 18.4 $\pm$ 0.67 mmHg (by applanation tonometry) and 20 $\pm$ 0.48 mmHg[4] (by rebound tonometry).

The value of IOP results from a balance between aqueous humor production and its outflow. Aqueous humor (AH) is secreted by the nonpigmented epithelium of the ciliary bodies through 3 basic mechanisms: diffusion, ultrafiltration, and active secretion. Carbonic anhydrase is the isoenzyme within the nonpigmented epithelial cells responsible for the formation of bicarbonate and its active transport across the ciliary epithelium. It catalyzes the following reaction: $H_2O + CO_2 \leftrightarrow HCO_3^- + H^+$. Na^+ ion is the major constituent actively transported from blood to AH by the enzyme complex Na^+, K^+-ATPase, located along the lateral intedigitations of the nonpigmented epithelium cells. The osmotic gradient of solutes resulting from their active transport across the ciliary epithelium favors the movement of water by diffusion and ultrafiltration, with the formation of AH. Carbonic anhydrase can account for up to 60% of AH production,[5] and even 75% in dogs.[6] This is especially important in the medical management of glaucoma, as carbonic anhydrase inhibitors (CAIs) are among the most effective medications in decreasing IOP.

The AH then flows from the posterior chamber through the pupil, to leave the eye through 2 main pathways, the conventional outflow and the uveoscleral or nonconventional outflow. The anatomic site of the former is the iridocorneal angle (ICA), where slender strands of pigmented tissue (pectinate ligaments) connect the base of the iris to the inner peripheral cornea. The AH flows through the pectinate ligaments and into the trabecular meshwork and from there into the aqueous angular plexus and the episcleral veins.[7] The uveoscleral outflow allows the drainage of AH posteriorly along the supraciliary-suprachoroidal spaces and into the adjacent sclera. This pathway accounts for only 3% of the total AH drainage in cats and 15% in dogs and it is influenced by the contraction of the ciliary body muscle and by the difference in hydrostatic pressure between the anterior chamber and the suprachoroidal spaces.[8] Glaucoma is caused by a pathologic increase in IOP, due to the impairment of aqueous humor outflow. IOP increase is still considered to be the major risk factor in optic nerve damage and subsequent blindness in cats and dogs.

Types of Glaucomas and Etiopathogenesis

Glaucoma is a multifactorial disease with many different phenotypes and etiologies, and consequently its classification is quite complex. According to their etiology, stage of disease, and

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Fig. 1. Severe congenital glaucoma in a young kitten.

ICA morphology, glaucomas can be primary, secondary, or congenital; early noncongestive, acute congestive, and chronic; open-angle, narrow- to closed-angle, and with goniodysgenesis, respectively.

Congenital glaucoma in dogs and cats develops at birth or a few weeks to months later (Fig 1); it is quite rare and usually caused by genetic defects leading to the abnormal development of the anterior chamber and the trabecular outflow pathways.[9] Primary glaucoma is the most common presentation in dogs.[10] In primary glaucoma, the increase in IOP is unrelated to any other concurrent ocular disorder; the disease is bilateral, although it does not usually develop simultaneously in both the eyes, and it has a known hereditary predisposition in several breeds (Table 1). The most common type of primary disorder in dogs is represented by closed angle glaucoma (PCAG), with American Cocker Spaniels and Basset Hounds being overrepresented in North America.[11] The pathogenesis and pathophysiology of PCAG are still largely unknown; however, several predisposing factors have been identified. Aging, gender, and genetics all play a role in the incidence of primary glaucoma. The onset of the disease is more common between 4 and 10 years of age and is usually characterized by abrupt and severe clinical signs; female dogs appear to be twice as likely to develop glaucoma as males.[11,12] Goniodysgenesis is a genetic defect in the development of the pectinate ligaments, which may be malformed and associated with decreased angle width. Several breeds predisposed to glaucoma show a high incidence of pectinate ligament dysplasia. However the incidence of glaucoma does not seem to be directly related to the severity of goniodysgenesis; other factors are clearly at play in the manifestation of the disease.[13] It has been suggested that changes in the ocular anatomy, with bowing of the iris, reverse pupillary block, and age-related increase in lens thickness are responsible for the increase in IOP.[14,15] These changes would result in increased apposition of the peripheral iris and cornea, with collapse of the filtration angle and impairment of outflow. More current theories have focused on the importance of age-related pigment dispersion with tissue remodeling and subtle but relentless inflammation and fibrosis, which are eventually responsible for modification of the trabecular meshwork extracellular matrix and of the outflow pathways.[16] The common final event to all types of glaucoma is the eventual degeneration of the optic nerve head, or glaucoma optic neuropathy (GON). Although the increase in IOP is still considered a main risk factor in dogs, other components are suspected in the progression of GON, from defects in the microcirculation of the optic nerve head and oxidative stress, to liberation of excitotoxic amino acids and mechanical stretching of the lamina cribrosa, with eventual axonal damage to the retinal ganglion cells.

Inherited primary open-angle glaucoma (POAG) is far less common than primary angle closure glaucoma, and is usually described in Beagles and Norwegian Elkhounds. In this disorder, representing the animal model of the most common type of glaucoma in people, the collapse of the ICA is apparent only in the advanced stages of the disease. At the origin of the disorder is a progressive accumulation of basement membrane-like material in the extracellular spaces of the trabecular meshwork, with impairment and eventual suppression of aqueous outflow.[17]

Secondary glaucoma is caused by preexistent intraocular disorders and tends to be unilateral. It is seen in all species, but represents the most common type of glaucoma in cats. Several ocular conditions can cause secondary glaucoma, from inflammatory and degenerative to neoplastic and traumatic (Table 2). The clinical onset is usually more slowly progressive than in primary glaucoma, although it may vary according to the nature of the primary disorder. Anterior and posterior synechiae, formation of preiridal fibrovascular membranes, lens dislocation and pupillary block, and cellular infiltrations (neoplastic or inflammatory) of the trabecular meshwork are all involved in the pathogenesis of secondary glaucoma. Treatment is directed toward the resolution, if possible, of the primary disease affecting the eye, but specific glaucoma therapy is also usually required. An uncommon type of secondary glaucoma is associated with the presence of diabetic, intumescent cataracts in dogs: phacomorphic glaucoma is caused by the anterior displacement of the iris, due to the increased size of the cataractous lens, with narrowing of ICA and anterior chamber (Fig 2). The only possible treatment is phacoemulsification of the lens.

Classification of glaucoma according to its stage is less relevant on the basis of investigative research, but quite pragmatic for its therapeutic approach. Early noncongestive glaucoma characterizes the early stages of both POAG and narrow-angle glaucoma. The clinical signs are insidious and often undetected, with values of IOP approximately at 25-30 mmHg. Sudden and severe spikes in IOP, at times approximating 50-70 mmHg, are typical of acute

Table 1
Canine Breeds Predisposed to Primary Glaucoma

American Cocker Spaniel	Akita
Basset Hound	Jack Russell Terrier
Chow Chow	English Cocker Spaniel
Shar-Pei	Lhasa Apso
Boston Terrier	Bouvier des Flandres
Fox Terrier, Wire	Pekingese
Norwegian Elkhound	Poodle, Toy
Siberian Husky	Beagle
Cairn Terrier	Brittany Spaniel
Poodle, Miniature	Saint Bernard
Samoyed	English Springer Spaniel
Bichon Frise	Poodle, Standard
Shih Tzu	Dalmatian
Australian Cattle Dog	

Table 2
Causes of Secondary Glaucoma in Cats and Dogs

Anterior uveitis
Lens—associated
Cataract
Lens luxation or subluxation
Intraocular neoplasms
Hyphema
Melanocytic (dog)
Aphakic or pseudophakic
Aqueous humor misdirection syndrome (cat)
Malignant