Diseases of the Lens and Cataract Surgery

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Mark P. Nasisse, DVM, Diplomate, ACVO

Carolina Veterinary Specialists

501 Nicholas Rd., Greensboro, NC 27409

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Embryology

The lens is a unique internal structure in that it is derived entirely from surface ectoderm. By gestation day 17 surface ectoderm thickens to form the lens placode. The lens placode subsequently invaginates to form the lens vesicle which becomes sequestered within the developing optic vesicle. Epithelial cells that line the posterior portion of the lens vesicle elongate to form the embryonic lens nucleus. The lens is nourished during embryologic life by the tunica vasculosa lentis, which posteriorly is comprised of the hyaloid artery (a part of the primary vitreous), and anteriorly by the embryologic pupillary membrane. Errors in normal lens embryogenesis may result in colobomas, microphakia, persistent hyperplastic primary vitreous, lenticonus and cataract formation.

Anatomy And Physiology

The lens capsule is the basal lamina of the lens epithelium and is thickest anteriorly. The epithelium lines the lens capsule anteriorly and at the equator and produces the basal lamina (capsule). The lens fibers are produced at the equator and compressed toward the nucleus as new cells are formed thus contributing to leticular sclerosis. Lens metabolism is most active at the equator and mediated predominantly by anaerobic glycolysis and to a lesser extent by the hexose monophosphate shunt, sorbitol pathway and the krebs cycle. Anything, which alters the metabolism or structure of the lens, is capable of producing a cataract.

Congenital Defects

Congenital lens defects include aphakia, microphakia, lenticonus, lentiglobus, coloboma, vascular anomalies (persistent pupillary membranes, persistent hyaloid artery, Mittendorf's dot, Bergmeister's papillae, and persistent hyperplastic primary vitreous (Figure 1). Although rare, multiple ocular abnormalities associated with lens changes have been reported in many breeds, including the Australian shepherd (congenital cataracts associated with microphthalmia, microcornea, equatorial staphylomas and retinal detachments), St. Bernard (microphakia and aphakia associated with microphthalmia, retinal detachment and retinal dysplasia), and Bedlington terrier, Sealyham terrier and Labrador retriever (congenital cataracts associated with retinal detachment and dysplasia).

Lens Pathology

Cataract is defined as any opacification of the lens, regardless of cause, size or location. Most cataracts in dogs are inherited, although they may be caused by congenital defects, nutritional deficiencies, toxic substances, uveal adhesions, and diabetes mellitus. The basic abnormality in cataract formation is degeneration of the normal protein structure of the lens fibers. As such, cataract formation affects predominantly the lens cortex. The earliest histologic feature of cortical protein degeneration is fiber swelling, referred to as bladder cells and Morgagnian globules. Bladder cells are swollen,

nucleated lens cells, and Morgagnian globules are spherical clumps of degenerating lens protein (Figure 2). These changes are first seen in the peripheral, subcapsular cortex. As lens proteins degenerate they liquefy, and such cataracts are referred to as hypermature. Liquefied lens material may or may not leak out of the lens capsule. Leakage of lens material through an intact capsule typically results in a lymphocytic-plasmacytic inflammatory response called phacolytic uveitis. Mineralization is seen in extremely advanced cataracts, particularly in the lens capsule. Epithelial changes include posterior migration of epithelium, fibrous pseudometaplasia (lens epithelial cells can undergo fibrous metaplasia to function as fibroblasts) and subcapsular fibroplasia. Rapidly developing cataracts, particularly in diabetic patients, will absorb water and swell (intumescence). Intumescent cataracts are recognized clinically by the presence of a shallow anterior chamber. Resorption of lens proteins is recognized clinically by wrinkling or flattening of the anterior lens capsule. In only very young dogs (< 2 years) can the lens proteins resorb completely.

The term after cataract refers to a diverse group of lens changes that occur after surgical lens extraction. In the most common surgical procedure used to remove lenses from animals (phacoemulsification), most of the lens capsule remains in the eye (Figure 3). In the event the lens epithelium is still viable at the time of surgery (young animals and immature cataracts), the surgical stimulation stimulates the epithelium to replicate and secrete lens material. The characteristic pathologic features of after cataracts are regeneration of new cortex, Elschnig's pearls, (large, globular, malformed lens cells) and Soemmering's ring (a ring shaped donut of abnormal secondary cortex in the periphery of the capsular bag). Fibrous metaplasia of lens epithelium also occurs with chronicity and causes capsule fibrosis and wrinkling. As newly secreted collagen matures and contracts, the lens capsule becomes wrinkled.

Lens Capsule Rupture

The term phacoclastic uveitis describes the potential inflammatory response that occurs if the lens capsule of an animal is ruptured as a result of a penetrating injury (e.g. cat scratch). The immune response is characterized histologically by lens capsule rupture, intra-lenticular neutrophils, and perilenticular mononuclear cells (Figure 4). With chronicity, a dense zone of fibroplasia will form around the ruptured lens. This form of uveitis, unless treated early by lens extraction, almost always results in loss of the eye due to secondary glaucoma. Spontaneous lens capsule rupture is occasionally seen secondary to chronic uveitis.

Classification Of Cataracts

Cataracts may be classified by age of onset (congenital, juvenile or developmental - less than 8 years of age, and senile - generally over 8 years of age), or by their location within the lens as determined by slit lamp biomicroscopy. Classifying cataracts by their degree of maturation, however, provides the most useful information relative to the prognosis for vision. Very minute opacities are referred to as incipient cataracts, and generally obstruct less than 10% of the tapetal reflection when the lens is retroilluminated. Cataracts are referred to as immature until they obstruct the entire tapetal reflection, after which they are considered mature cataracts. Cataracts are considered hypermature if lens material has become liquefied, and are recognized by a rough or irregular anterior lens capsule, a deep anterior chamber, or clinical signs of uveitis (aqueous flare and low intraocular pressure) (Figure 5). If enough cortex liquefies the nucleus will settle to the bottom of the lens capsule, the cataract is termed Morgagnian.

Causes Of Cataracts

Genetic defects are the most common cause of cataracts in dogs. A large number of breeds are affected and the time of onset varies markedly. For example, recessive cataracts are suspected in the Miniature schnauzer (congenital), American cocker spaniel (congenital to juvenile) Afghan hound (juvenile), standard poodle (juvenile), old English sheepdog (congenital), miniature poodle (adult or senile), terrier breeds, and many brachycephalic breeds. Dominant cataracts are suspected in the Labrador retriever (adult), beagle (congenital), and golden retriever (congenital to juvenile). Cataracts whose inheritance pattern is unknown are seen in the Chesapeake Bay retriever, Labrador retriever, and red cocker spaniel. Associated ocular diseases include progressive retinal atrophy (PRA) (miniature poodle, cocker spaniel, and miniature schnauzer), central PRA (Labrador retriever) retinal dysplasia (Labrador retriever), and multiple ocular defects (red cocker spaniel, beagle, and old English sheepdog). Senility accounts for spontaneous cataract formation in aged dogs of all breeds.

The most common metabolic cataract is caused by diabetes mellitus. As glucose levels increase in the eye, hexokinase, the regulatory enzyme, becomes saturated. Glucose accumulates in the lens and begins to be metabolized through the sorbitol pathway. The sugar alcohols sorbitol and fructose accumulate within the cells of the lens since they penetrate cell membranes (including the lens capsule) poorly. The result is an intracellular accumulation of solutes and hypertonicity, which results in an accumulation of water within the lens fibers. Swelling of the lens fibers progresses and the fibers rupture forming vacuoles in the lens cortices. This continues until the entire lens becomes cataractous. The majority of diabetic dogs eventually develop cataracts.

Puppies and kittens may develop cataracts as a consequence of nutrient (presumed amino acid) deficiency. Nutritional cataracts rarely occur unless puppies are orphaned within the first 2 weeks of life and fed milk replacer exclusively. Cataracts may or may not regress. Rarely cataracts may be seen after exposure to certain drugs or electric shock. Dogs in the terminal stages of sensory retinal atrophy commonly develop cataracts secondary to the toxic by-products of retinal degeneration. Cataracts may also occur secondary to inflammation (anterior uveitis), persistent vascular remnants (persistent pupillary membranes and hyaloid artery remnants), or trauma (lens capsule penetration).

Spontaneous Cataract Resorption

All hypermature cataracts are associated with some degree of lens protein liquefaction, depending on the age of the animal. In very young animals, presumably because their lens proteins are still highly soluble, resorption may occur sufficiently to restore vision. In most animals, however, lens resorption is more likely to result in complications such as lens-induced uveitis and retinal detachment (Figure 6).

Patient Selection For Cataract Surgery

Cataract surgery is generally indicated in any patient with significant vision impairment, or when significant vision impairment is impending. The prognosis for return of vision is optimum if the cataract is removed prior to the cataract becoming hypermature. Retinal disease is ruled out by electroretinogram. The presence of a pupillary light response does not necessarily indicate absence of retinal disease as pupillary light responses are preserved until late in the course of the degenerative process. Ultrasonography is indicated in animals with hypermature cataracts to rule out retinal detachment. Gonioscopy is advisable in breeds predisposed to primary glaucoma. Lens induced uveitis, if present, should be suppressed with preoperative corticosteroid therapy.

Contemporary Surgical Technique

The current standard technique for lens extraction in dogs is phacoemulsification (Figure 7-9). A conventional lens extraction follows the following sequence: 1) patient positioning, 2) ventilator controlled respiration, 3) neuromuscular blockage to achieve neutral globe position (atracurium or pancuronium), 4) 2/3 depth, 7-8 mm corneal groove, 5) 3mm corneal stab wound, 6) instillation of viscoelastic material, 7) anterior capsulectomy, 8) nuclear sculpting with phacoemulsification (ultrasonic lens fragmentation at 30,000-50,000 cycles per second), 9) cortical material cleanup by irrigation-aspiration, 10) intraocular lens insertion, and 11) wound closure with continuous sutures of 8-0 or 9-0 vicryl. Many different approaches are taken to control postoperative complications. Our protocol consists of 1 week of QID Pred Forte, TID tropicamide, and TID 0.3% tobramycin. Thereafter topical TID Pred Forte is used for up to 3 months.

Prognosis

Refinements in phacoemulsification technique have improved the short-term success to 95%. The most common immediate complications are uveitis, glaucoma and endophthalmitis. Other complications such as retinal detachment, hyphema, hypopyon, IOL luxation, and posterior capsule tears are largely the consequence of poor surgical technique. Long term success decreases to approximately 70%. Chronic postoperative uveitis is a major risk factor for development of secondary glaucoma.

Subluxation And Luxation Of The Lens

Lens displacement occurs secondary to damage to or spontaneous degeneration of the ciliary zonules. The specific causes are 1) trauma (uncommon), 2) secondary to glaucoma, 3) secondary to chronic uveitis (especially in cats), 4) secondary to hypermature cataracts, and 5) senile zonular degeneration. Most treatable lens luxations are primary (familial) and are seen in wirehaired fox terriers, Sealyham terriers, Manchester terriers, Welsh terriers, poodles, Shar peis, and Jack Russell terriers. The clinical signs are aphakic crescent, iridodonesis - quivering of iris with eye movement, deep anterior chamber, shallow anterior chamber and vitreous in the anterior chamber. Complications of lens luxation are corneal endothelial damage, corneal edema, secondary glaucoma, anterior uveitis, and retinal detachment.

Lens subluxations are treated by intracapsular lens extraction with suture fixation of an intraocular lens. The prognosis is best if the lens is removed prior to the onset of glaucoma.

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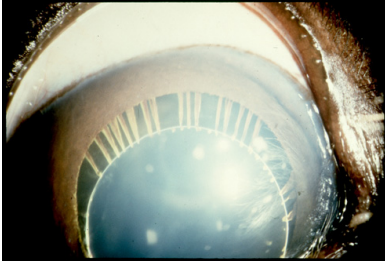
Figure Legends:

Figure 1. Clinical appearance of microphakia in a young dog. Note the elongated ciliary zonules
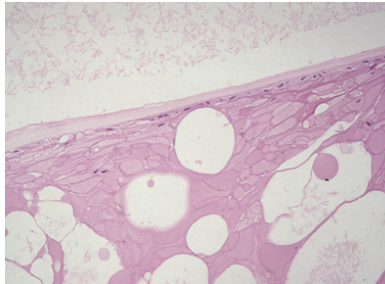

Figure 2. Photomicrograph revealing typical features of cataract formation. Markedly swollen lens cells are interspersed with large vacuoles created by lens

Figure 3. After-cataract in a dog that had previously undergone lens extraction. The pupillary opacification is caused by capsule wrinkling and the production of opaque lens material.
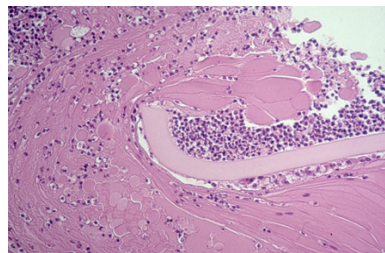

Figure 4. Photomicrograph of a lens that had been ruptured by a perforating injury. Enucleation was required because the inflammation caused secondary glaucoma. Swollen and degenerate lens fibers are infiltrated with numerous neutrophils. (H&E, X400).

Figure 5. Appearance of a hypermature cataract, The anterior lens capsule is wrinkled as the result of lens protein liquefaction.

Figure 6. Hypermature cataract and chronic lens induced uveitis. The dyscoria is caused by numerous posterior synechia
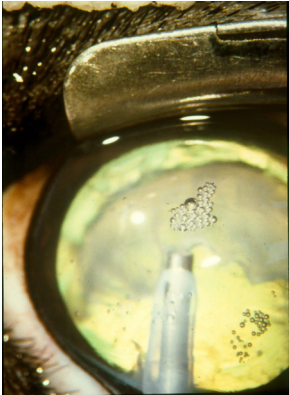

Figure 7. Operating microscope photograph depicting nuclear fragmentation during phacoemulsification.

Figure 8. Operating microscope photograph depicting aspiration of loose lens cortical material following fragmentation of the lens nucleus.
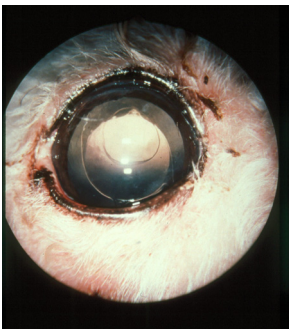

Figure 9. Appearance of a canine eye containing a prosthetic lens implant 1 year after phacoemulsification