Cataracts in dog and actually trends in opacified lens removal

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Abstract: Cataract is an opacity of the lens resulting from pathologic changes in lens protein composition or disruption of lens fiber arrangement. Cataracts or opacities of the lens of the eye are a leading cause of blindness or an increasing cause of vision loss in dogs. Cataract removal or lens extraction is one of the most common surgeries performed by veterinary ophthalmologists. A lot of techniques for lens removal have been described, including intracapsular (removal of the lens capsule, cortex and nucleus together) and extracapsular (removal of the lens cortex and nucleus, and leaving most of the lens capsule in situ) procedures. Phacoemulsification with aspiration of the lens material, a type of extracapsular cataract removal, is the most common technique for removal of cataracts in veterinary patients.

Surgical 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. Lens proteins are immunologically sequestered (large amounts of lens material which gain access to the anterior chamber is capable of inciting a destructive immunologic reaction). Anything that alters the metabolism or structure of the lens is capable of producing a cataract (6, 7).

Lens congenital anomalies

Lens congenital defects include aphakia, microphakia, lenticous, lentiglobus, coloboma, vascular anomalies (persistent pupillary membranes, persistent hyaloid artery, and persistent hyperplastic primary vitreous (PHPV)). Multiple ocular abnormalities associated with lens changes are seen in 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); Labrador retriever (congenital cataracts associated with retinal detachment and dysplasia) (2, 4).

Cataracts

Cataract is defined as any opacification of the lens, regardless of cause, size or location (1, 2, 4, 10). 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. Cortex changes include fiber swelling. These are the earliest indicators of cataract formation, usually seen in the peripheral (subcapsular) cortex. As lens proteins degenerate, liquefaction of lens fibers is seen. Liquefied lens material may or may not leak out of the lens capsule. Mineralization is seen in extremely advanced cataracts. Epithelial changes include posterior migration of epithelium, fibrous pseudometaplasia (lens epithelial cells can undergo fibrous metaplasia to function as fibroblasts) and subcapsular fibroplasia. Size and shape changes are intumescence (lens swelling) and lens resorption (hypermature cataract). Resorption is associated with flattening of the normal lens curvature as the lens becomes smaller. Only in very young dogs (<2 years) can the lens resorb completely. One of the most common sequelae to resorbing lens material is phacolytic uveitis. If significant resorption occurs, the capsule becomes wrinkled. The term “after cataract” refers to a diverse group of lens changes that occur after surgical lens extraction. In the most common method used to remove lenses from animals (phacoemulsification), most of the lens capsule remains in the eye. In the event the lens epithelium is still viable at the time of surgery (young animals and immature cataracts), surgical stimulation invariably stimulates the epithelium to replicate and secrete lens material. Fibrous metaplasia of lens epithelium also occurs with chronicity to cause capsule fibrosis and wrinkling. The collagen secreted by the metaplastic lens epithelium has a characteristic tendency to line the lens capsule. As it matures and contracts, the lens capsule becomes wrinkled(4,8,9).

Lens Capsule Rupture

Phacoclastic uveitis may occur if the lens capsule of an animal is ruptured as a result of a penetrating injury, because massive amounts of highly antigenic lens material can gain access to the immune system. The subsequent immune response is characterized histologically by the combination of lens capsule rupture, intra-lenticular neutrophils, and perilenticular mononuclear cells. 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(4,10).

Classification of Cataracts

Cataracts may be classified by age of onset: congenital (present at birth), juvenile (developmental, less than eight years of age), and senile (generally over eight years of age). Cataracts are also described in terms of location as determined by biomicroscopy: capsular, subcapsular, cortical, nuclear, axial and polar. However, their degree of maturation is the most important feature relative to lens extraction: incipient (earliest changes, 10% tapetal reflex obstructed); immature; mature (completely solid, no tapetal or fundus reflex visible, lens capsule smooth and regular); and hypermature (lens material, particularly cortical, may undergo liquefaction, may be “complete” cataract or may see a partial fundus reflex). Hypermature is recognized by rough or irregular anterior lens capsule and signs of lens-induced uveitis. If enough cortex liquefies, the nucleus will settle to the bottom of the lens and is termed a Morgagnian cataract(2,4,8,11,12).
Causes of cataracts

Genetic defects are the most common cause of cataracts in dogs. Many breeds are affected. For example, recessive cataracts are suspected in the Miniature Schnauzer—congenital, American Cocker Spaniel—congenital to juvenile, Afghan—juvenile, Standard Poodle—juvenile, Old English Sheepdog—congenital, Miniature Poodle—adult, Terrier breeds and many brachycephalic breeds. Dominant cataracts are suspected in the Labrador Retriever—adult, Beagle—congenital, and Golden Retriever—congenital to juvenile (some disagree and list this as unknown inheritance). Cataracts whose inheritance pattern is unknown are seen in the 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, old English Sheepdog)(4). 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. A large majority of diabetic dogs eventually develops cataracts. Puppies and kittens may develop cataracts from nutrient deficiency (amino acid). This usually does not occur unless puppies are orphaned within the first two weeks of life and fed exclusively milk replacer. Cataracts may or may not regress. Toxic cataracts are seen after exposure to some drugs (uncommon), or secondary to PRA (cataracts occur secondary to by-products of retinal degeneration (dialdehydes) which diffuse through the vitreous to the lens). Cataracts may also occur secondary to inflammation (anterior uveitis), persistent vascular remnants (PPMs, hyaloid remnants), and trauma (usually requires lens capsule penetration)(4,8,12).

Actually surgical technique

Cataract surgery is generally indicated in any patient with significant vision impairment, or when significant vision impairment is impending. The current standard for canine lens extraction is phacoemulsification. A conventional lens extraction follows the following sequence: 1) patient positioning; 2) neuromuscular blockage to achieve neutral globe position; 3) 2/3 depth, 7–8 mm corneal groove; 4) 3 mm corneal stab wound; 5) instillation of viscoelastic material; 6) anterior capsulotomy; 7) nuclear sculpting with phacoemulsification (ultrasonic lens fragmentation at 30,000-50,000 cycles per second); 8) cortical material cleaning by irrigation/aspiration; 10) intraocular lens implantation; and 10) wound closure with continuous sutures of 8-0 or 9-0 Vicryl or Monocryl. Many different approaches are taken to control postoperative complications. The protocol consists of one week of topical antiinflammatory, antibiotics and tropicamide(3,5).

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.

BIBLIOGRAPHY