

OCULAR Outlook

A QUARTERLY PUBLICATION FOR THE VETERINARY COMMUNITY FROM EYE CARE FOR ANIMALS

OCULAR MANIFESTATIONS OF DIABETES MELLITUS



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Diabetes mellitus (DM) may develop in many species; the majority of affected dogs will develop ocular manifestations. Although cats also develop DM, the ocular manifestations are not as severe. Fifty percent of dogs develop cataracts within 170 days of being diagnosed with DM. Seventy-five and eighty percent of diabetic dogs develop cataracts by 370 and 470 days, respectively; this complication is the most obvious as it interferes with the patient's visual function and the owner notices the visual disturbance (VO 1999;2:169-172).

Cataract formation occurs when normal metabolism by the anaerobic hexokinase pathway is saturated by increased concentrations of glucose, and aldose reductase, an enzyme in an alternate energy pathway, is activated to metabolize the excess sugar associated with DM. Aldose reductase metabolizes glucose to sorbitol. Sorbitol does not diffuse well across the lens capsule, so water (from the aqueous humor) is imbibed into the lens resulting in lens opacification. Cats are not affected in the same way because they do not have high levels of aldose reductase and thus do not usually develop "diabetic cataracts" (Veterinary Ophthalmology, 4th Ed., Gelatt, ed. 2007).

The immune system of the eye responds to any change in lens structure and organization with inflammation. As a cataract develops and the altered lens proteins result in lens opacity and stimulation of the ocular immune system, uveitis develops and should be addressed with non-steroidal anti-inflammatories (NSAID) in diabetic dogs. In cases of severe LIU, steroids may be utilized, but can interfere with diabetic regulation. Usually NSAIDS's are used



Mature Diabetic Cataract

on a long-term basis to control lens-induced uveitis. Chronic inflammation may lead to the sequelae of glaucoma, lens luxation, retinal detachment, etc. Managing diabetic cataracts with an anti-inflammatory prevents pain and irritation for the patient and preserves the eye as a candidate for cataract surgery, if the owner elects that procedure. The majority of practicing veterinary ophthalmologists feel that this is the appropriate treatment for cataractous lenses.

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OCULAR Outlook

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Sunset, Utah
Leesburg, Virginia
Pewaukee, Wisconsin

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The cataractous lenses of diabetic dogs were significantly increased in axial thickness (8.4 +/- 0.9 mm) compared to normal lenses (6.7 +/- 1.0 mm), immature cataractous lenses (6.4 +/- 0.8 mm), and mature cataractous (7.4 +/- 0.9 mm) lenses. The anterior chamber depth was significantly decreased in diabetic cataracts vs. immature and mature cataracts of non-diabetic dogs. The lenses of non-diabetic cataract patients also had a trend to increased axial thickness.

Immature cataracts of non-diabetic dogs had a trend to decreased axial thickness. No change in axial globe length was noted in any dogs (VO 2004;7:91-95).

Another complication of diabetic cataract formation is capsular rupture. There is a good prognosis for vision if surgery is performed soon after rupture, prior to development of glaucoma and/or retinal detachment and dysfunction (VO 2006; 9: 328-334). Cataract surgery is generally shorter with a faster healing time if the lens is operated when the cataract is still immature. This finding substantiates the recommendation to perform phacoemulsification on immature diabetic cataracts.

Although cataract formation is the most obvious complication of diabetes mellitus, other ocular health issues also develop. Conjunctival epithelial

dysplasia, squamous metaplasia, and decreased goblet cell density has been documented on conjunctival biopsies in diabetic patients. A 2005 study (VO 2005;8:215-224) found that diabetic dogs had altered pre-corneal tear film and decreased aqueous tear production. 3 groups of dogs were assessed: 1. diabetic dogs with cataracts, 2. non-diabetic dogs with cataracts, 3. non-diabetic dogs without cataracts. Schirmer tear test values were lower in diabetic dogs. Tear film break up time (TFBUT) was also lower in diabetic dogs. Tear glucose concentrations were higher in diabetic patients, not surprisingly. In summary, altered tear film function with qualitative and quantitative tear film changes and mucin abnormalities were noted. Often the only sign was mild conjunctival hyperemia.



KCS and Tear Film Deficiency

Supplementation with a mucinomimetic and/or lacrimomimetic such as cyclosporine may be indicated. In a publication in Diabetes/Metabolism Research and Reviews (2001;17:189-212) it was concluded that vitamin C and E supplementation may decrease nitrate levels

and improve STT, TFBUT, and goblet cell density in human patients

Corneal sensitivity was lower in diabetic dogs with cataracts, and also in non-diabetic dogs with cataracts, but the trend was stronger in diabetic patients with cataracts.

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OCULAR MANIFESTATIONS OF DIABETES MELLITUS
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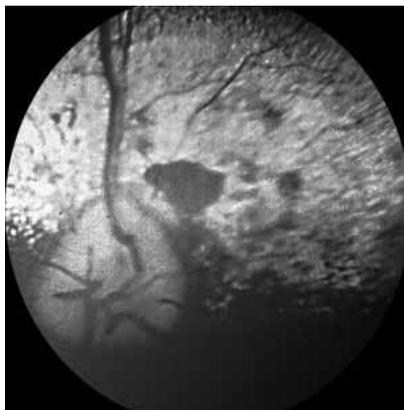
The finding of decreased corneal sensitivity may be associated with a greater predisposition to corneal ulceration in diabetics. In people, this is considered a manifestation of diabetic neuropathy (the polyol metabolic pathway may be disrupted; in man, treatment with aldose reductase inhibitors has increased nerve conduction velocity). The degree of glycemic control does not significantly affect these abnormalities; (Proceedings ACVO 2003, Coeur D'Alene, ID). Decreased corneal sensitivity in diabetic dogs was also noted in an AJVR study (AJVR 2003;64:7-11).

Corneal endothelial cell loss (Current Eye Research 1985;4:759-766) and corneal endothelial cell pleomorphism and polymegathism (IOVS 1990;31:2201-2204) were noted in two publications. Lynch et. al found corneal edema to be a short-term complication of phacofragmentation in cataracts in all patients (VO 2006;9:303-310).

However, mean central corneal thickness was significantly increased in the diabetic group (764 um) compared to the non-diabetic group (629 um) at all time points. The change was greatest from pre-operatively to 1 day post-operatively, and by more than 2 months post-operatively, the affected corneas compensated for the initial sharper rise in thickness by returning to below baseline. This observation was exacerbated in those patients experiencing post-operative ocular hypertension (VO 2006;9:303-310).

Ultrasound biomicroscopy of the anterior segment in dogs with diabetic

or non-diabetic cataract formation was performed in another study. 87 eyes of 47 dogs were examined. The diabetic group had greater corneal thickness measurements and shallower anterior chambers (Proceedings ACVO 2010, San Diego, CA).



Diabetic Retinopathy

Lastly, the retina may be affected by diabetes mellitus. Retinal vascular damage, e.g., formation of microaneurysms and diabetic retinopathy has been noted in both human and canine diabetic patients (Archives of Ophthalmology 1990;108:1301-1309).

Based on the above studies several conclusions and recommendations are noted:

1. Monitor STT, tear film quality and cataract formation in diabetic dogs.
2. Initiate topical tear replacement and lacrimomimetics as necessary.
3. Start topical anti-inflammatories when cataract formation is noted.
4. Counsel the owner/client to have phacoemulsification performed as soon as possible if that treatment is considered so that fewer possible complications occur.

Spotlight on NEW LOCATIONS

Torrance

4240 Artesia Boulevard Suite #101
Torrance, CA 90504
Phone: (310) 533-EYES (3937)
Monday - Friday 8:00 a.m. - 5:00 p.m.

Wichita

727 South Washington Avenue
Wichita, KS 67211
Phone: (913) 381-EYES (3937)
Select Fridays each month
9:00 a.m. - 4:00 p.m.
By appointment only - please call first

Lexington

1591 Winchester Road
Lexington, KY 40505
Phone: (502) 272-4340
Fax: (502) 272-4341
Select Tuesdays each month
9:00 a.m. - 4:00 p.m.
By appointment only - please call first

Upcoming EVENTS

March 22, 2011 — Ontario, CA
*Ocular Emergencies & Topical
Therapy in Dermatology*
Sara Calvarese, DVM, DACVO
Jennifer Aniya, DVM, DACVD

April 2, 2011 — Tempe, AZ
Day with the Specialists

April 30-May 1, 2011- Newport Beach, CA
Weekend with the Specialists

May 6, 2011 — Washington D.C.
CVC Wet lab: Ophthalmic surgery
Jennifer Hyman, DVM, DACVO
Mark Bobofchak, DVM, DACVO
Reuben Merideth, DVM, DACVO

August 20-21, 2011- Albuquerque, NM
Weekend with the Specialists

August 31, 2011- Gilbert, AZ
Ophthalmology CE

For more information please
contact Julie Gamarano at
jgamarano@eyecareforanimals.com

DIAGNOSTIC CHALLENGE

Superficial Keratectomy and Adjunctive Cryotherapy for Canine Corneal Squamous Cell Carcinoma



Lynsey Smith, DVM
Diplomate, American College of
Veterinary Ophthalmologists

Signalment: 12-year-old male neutered English Bulldog.

History: The patient presented for a 1-year history of a slowly progressive, non-painful, corneal mass and 6-month history of progressive vision loss OS. Keratoconjunctivitis sicca was diagnosed seven years prior to presentation by the rDVM. Previous medications included BNP ointment with dexamethasone and Tacrolimus.

Initial Exam Findings and

Diagnostics: Evaluation revealed severe conjunctival hyperemia and mild mucoid discharge OS; no blepharospasm was noted. Euryblepharon and prominent globes were present OU. Slit-lamp biomicroscopy OS revealed a raised, verrucous mass with distinct margins emanating from the ventral periaxial cornea. Circumferential arborizing neovascularization as well as diffuse, moderate corneal edema were noted. (*Figure 1*) Schirmer Tear Test (STT) values were WNL at 25 mm/min OU. Corneal fluorescein stain uptake was negative OU. Intraocular pressures were WNL OU.

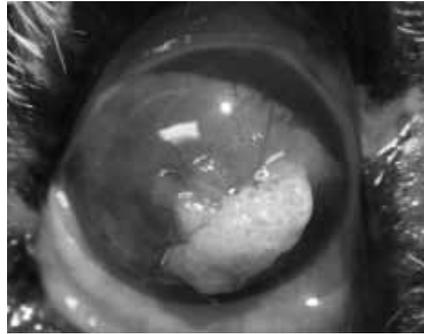


Figure 1: A pink, raised, multi-lobulated, verrucous mass with distinct margins

Differential Considerations:

Neoplasia; corneal granulation tissue (secondary to trauma, foreign body, neurotrophic keratitis, neurogenic KCS, neuroparalytic keratitis, precorneal tear film deficiency – qualitative or quantitative, spontaneous chronic corneal epithelial defects); chronic superficial keratitis, epithelial dysplasia/hyperplasia, and corneal epithelial inclusion cyst.

The clinical appearance of the mass was most consistent with corneal neoplasia (SCC and papilloma being most likely) or corneal granulation tissue secondary to trauma.

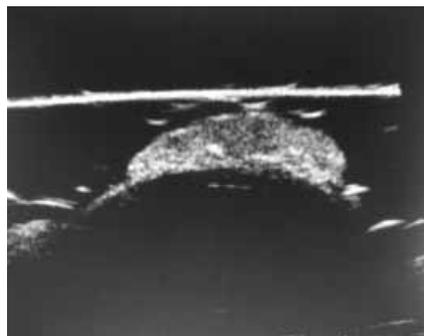


Figure 2: B-scan ultrasound image OS using a 35 MHz probe

Laboratory and Ancillary Diagnostic Tests: CBC and chemistry panels were WNL. A cytology was obtained via cytobrush under topical anesthesia. This was followed by a 1

mm snip biopsy of the mass in order to obtain a histopathologic diagnosis. B-scan ultrasound using a 35 MHz probe was performed to evaluate the depth of the mass in the cornea and aid in surgical planning. The mass did not appear to extend past the normal corneal epithelium. (*Figure 2*) Three-view thoracic radiographs as well as regional lymph node aspirates were recommended and declined by the owner. All peripheral lymph nodes palpated WNL.

Diagnosis: Corneal Squamous Cell Carcinoma

Treatment: Cytology and histopathology imparted the diagnosis of SCC and a superficial keratectomy followed by cryotherapy was recommended. The corneal lesion was delineated with an ovoid, 20 percent depth, circumferential corneal incision. The mass was undermined via corneal lamellar dissection. (*Figure 3*) The keratectomy site was frozen with liquid nitrogen in a double freeze thaw cycle. Postoperative medications included: tobramycin, cephalexin, carprofen, tramadol, and an elizabethan collar.



Figure 3: Intra-operative photograph of the superficial keratectomy

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Follow-up: The histopathology report confirmed the diagnosis of corneal squamous cell carcinoma with clean margins.

Two weeks post operatively, fluorescein staining was negative OU. Topical tobramycin was stopped and neomycin-polymixin-dexamethasone drops were instituted OS TID to decrease scarring and neovascularization.

Three weeks postoperatively, conjunctival hyperemia had resolved and the OS was open and comfortable. The surface of the cornea was smooth and the corneal edema had decreased resulting in a faint diffuse corneal nebula OS. All

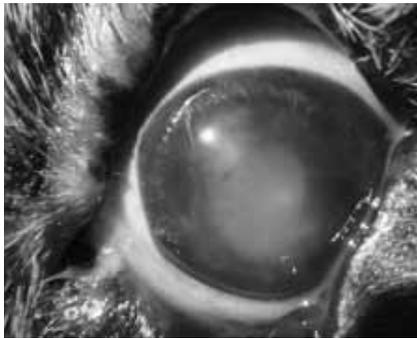


Figure 4: Three weeks post-operatively

medications were stopped at that time. (Figure 4)

Summary: SCC follows a progressive course from precancerous dysplastic lesions, to papillomas, followed by carcinoma in situ, and finally SCC. Predisposing factors reported for the development of SCC include exposure to UV radiation, previous trauma,

papilloma virus infection, age, and chronic irritation.

Cryosurgery with a double freeze technique has been used with a 97% success rate in cattle SCC. Reports of its use in canine patients are limited to case reports with disease free intervals of over 1 year.

Corneal SCCs are typically locally aggressive but may demonstrate local regional and distant metastasis. No visible evidence of recurrence was present 8 months postoperatively. This reinforces previous reports of successful treatment of corneal SCC with keratectomy and cryotherapy.

Eye Care for Animals Would Like to Welcome



Katherine Cutter, DVM
Diplomate, American College of
Veterinary Ophthalmologists

Dr. Cutter completed her BS in Animal Science and DVM at North Carolina State University in 1996 and 2000, respectively. In 2000, Dr. Cutter moved on to Cornell University where she completed her rotating internship in small animal medicine and surgery. She remained at Cornell to complete her ophthalmology residency (2001-2004). Dr. Cutter became board-certified in 2004 and, following her residency training, Dr. Cutter accepted an adjunct teaching appointment at Massey University in NZ, where she continues to lecture. From 2004-2006, Dr. Cutter worked as a staff ophthalmologist at Canada West Veterinary Specialists in Vancouver, BC. In 2007, Dr. Cutter accepted a position at Animal Eye Care in Athens, GA. In 2011, Dr. Cutter relocated to Santa Fe, NM with her husband and two children. She has joined ECFA and will be seeing appointments in our Santa Fe, NM location. Her ophthalmic interests include tear film deficiencies, corneal repair, glaucoma, and neuro-ophthalmology. Dr. Cutter also relishes her time spent primarily caring for her two young boys, Zachary (2 1/2 years) and Alexander (4 1/2 years). She practices in Santa Fe, NM.



Dan Lavach, DVM
Diplomate, American College of
Veterinary Ophthalmologists

Dr. Lavach obtained his veterinary degree from the College of Veterinary Medicine & Biomedical Sciences at Colorado State University. After several years in general veterinary practice he returned to CSU to complete a Masters of Science degree and a comparative ophthalmology residency. Dr. Lavach has a broad background in veterinary medicine and comparative ophthalmology. His experiences include private practice, academic position appointments, and consulting for ophthalmic instrument manufacturers and pharmaceutical companies. He was a member of the Board of Regents of the American College of Veterinary Ophthalmologists for several years and served as its President. Dr. Lavach has written ophthalmology textbooks, numerous book chapters and scientific papers, and he presents continuing education seminars for veterinarians, veterinary technicians, and pet clubs. He practices in Reno, Nevada.

HOW NOT TO IRRITATE YOUR CLIENTS

Employees don't have to work very hard to make your clients feel uncomfortable, irritated, or even unhappy. The basic examples below are what you never want to see any of your team members doing or saying, as they are sure fire ways to turn your clients off and maybe even chase clients away!

Don't forget to acknowledge clients as soon as they enter your reception area.

If possible, help clients with the door when they are entering with their pet. If on the telephone, make eye contact with clients entering and hold up a finger to acknowledge them. **Don't call your patient by the wrong name.** Ensure that employees have the patient chart out in advance, watch for the patient around its arrival time, and call the patient by the correct name. **Don't call your patient by the wrong gender.** Consider using color coded folders or stickers to differentiate between male and female on the

patient's medical record. **Don't consistently run behind on appointments.** For the most part, clients understand when emergencies present themselves and there may be an occasional time when they have to wait longer for their pet to be seen. Please be sure your client's pets are not consistently waiting at each visit. Always apologize for the wait and explain that there was an emergency, if applicable. **Don't ignore bad smells or pet messes.** Be sure to clean up all pet messes right away and ensure there are no foul smells lingering. It's easy for clients to associate bad smells with the hospital not being clean! **Don't forget to project a professional image.** Never drink or eat food in front of clients. Never wear wrinkled or unkept attire and make every effort to project a clean, polished image. **Don't send clients away without reviewing "go home" instructions.** Clients expect some direction after their pet is seen. Always go over instructions



Karen Webster, MBA
President & CEO
Eye Care for Animals

with the client, even if the patient is in for a routine or follow-up exam. **Don't forget to show appreciation to EVERY client!** Take the time to show appreciation to every client and patient walking through the door. Clients have choices and you want them to know how much you appreciate them. Give your surgical patient an inexpensive toy to keep in the run and take home after surgery, and give your exam patients a pet treat when they leave.

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