J Vet Clin 18(4) : 424-428(2001)

Keratoconjunctivitis Sicca (KCS) and Its Surgical Therapy in a Dog

Heung-Myong Woo[1]

Department of Surgical Sciences, School of Veterinary Medicine, University of Wisconsin, Madison, Wisconsin, 53706, USA

Abstract : Keratoconjunctivitis sicca (KCS) is a relatively common eye disease in the dog. Reduction or arrest of aqueous tear production by lacrimal glands results in quantitative alteration of the precorneal tear film (PTF) and KCS may result. A dog with a presumptive diagnosis of KCS referred was used for the study of KCS. Cytological and histopathologic examination is performed, and the various types of medical and surgical treatment are studied, including the operation of parotid duct transposition (PDT). This report describes unilateral KCS in a dog of unknown etiology who responded to PDT.

Key words **:** keratoconjunctivitis sicca, parotid duct transposition, tears, eye, dog

Introduction

Keratoconjunctivitis sicca (KCS) is a relatively common eye disease in the dog. Reduction or arrest of aqueous tear production by lacrimal glands results in quantitative alteration of the precorneal tear film (PTF) and KCS may result. PTF is a composite, trilaminar fluid composed of lipid, aqueous and mucine layers[4,13,19,27]. Alteration of any layer or component of the PTF may result in quantitative or qualitative changes compromising tear function. The major volume of tear is comprised of the middle aqueous layer, produced by the major and accessory lacrimal glands. Reduction or arrest of aqueous tear production by these glands results in quantitative alteration of the PTF and KCS may result[4,13]. Pathologic changes in conjunctival and corneal morphology occur with subsequent clinical signs of KCS[15].

Diagnosis of KCS is based upon characteristic clinical signs and reduction or absence of aqueous tear formation. Tear production is evaluated by the Schirmer Tear Test (I/II)[6,22]. Although the STT presumably lacks reproducibility in man and animals, symptomatic cases of KCS frequently have STT values less than 5 mm/min[1]. Normal tear production in the dog is 21 mm/min[6], (range 14-24 mm/min). Aqueous tear production in this case was repetitively 0 mm/min OS by STT (I), both proceeding and during treatment.

The etiology of KCS is diverse and usually undetermined[11,13]. The majority of cases are bilateral and considered idiopathic, due to presumed immune-mediated dacryoadenitis[11,13]. The lacrimal acinar epithelial cell exhibits intercellular junctions that allow ductules to be immune privileged[26]. Loss of self tolerance in lacrimal or related tissues may trigger auto-immune reactivity to lacrimal components[11,12]. Immune surveillance of lacrimal tissue may be disturbed by alteration of resident lymphocyte subsets and has been shown in murine models of Sjogren's syndrome[9]. Histologic evidence of lymphocyte and plasmacyte infiltration of affected glands[10,11], and circulating auto-antibody to lacrimal, salivary and thyroid tissue[11,12] further supports an immune-mediated pathogenesis. Purported senile atrophy of the lacrimal gland may, in actuality represent an immune mediated process[13]. Non immune-mediated causes of KCS occur and include genetic predisposition[13], infection (canine distemper[17]), neuropathy[1,13], trauma,[1,13] drug toxicosis[24], congenital alacrima[1,13], surgery and chronic blepharitis[4]. Occlusion of lacrimal ducts was possible in this case considering the normal histologic appearance of the nictitans gland.

A dog with a presumptive diagnosis of KCS referred was used for the study of KCS. Cytological and histopathologic examination is performed, and the various types of medical and surgical treatment are studied, including the operation of parotid duct transposition (PDT). This report describes unilateral KCS in a dog of unknown etiology who responded to PDT.

Case

Patient

Canine, German Shorthair Pointer, Female (Spayed), 11 years of age.

History

The dog developed "nattering" of the left eye following a one-week course of trimethoprim-sulfamethoxazole, initiated for the treatment of chronic otitis externa. A presumptive diagnosis of Keratoconjunctivitis sicca was made and the systemic antibiotic discontinued. Symptomatic therapy failed to resolve the clinical signs and the case was referred one week later.

Clinical finding and Diagnosis

Initial clinical finding

Blepharospasm of the left eye was noted with mucopurulent

[1]Corresponding author.
E-mail : woo@surgery.wisc.edu

Fig 1. Initial clinical appearance of the left eye.

ocular exudate (Fig 1). Loosely adherent mucus strands were noted on the corneal surface. No conformational eyelid defects were present and corneal and palpebral reflexes were intact. Biomicroscopy revealed diffusely chemotic and hyperemic palpebral and bulbar conjunctiva. Corneal vascularity, pigmentation and scarring were absent. Schirmer tear test (STT; Schirmer Tear Test Strips, Alcon Lab., Inc., Fort Worth, TX) values were 0 mm/min OS and 18 mm/min OD. Rose bengal (Rose Bengal Ophthalmic Strips, Barnes-Hind, Inc., Sunnyvale, CA) staining revealed faint, stipple-like retention on the left corneal surface. Tear film breakup times were decreased. Sodium fluorescein stain (Fluori-I-Strips, Ayerst Laboratories, Inc., New York, NY) was not retained and traversed the NL system of the left side. Direct and indirect ophthalmoscopy was normal. The right eye was normal. General physical examination was unremarkable.

Differential considerations

Keratoconjunctivitis sicca (KCS) was suspected based upon ocular clinical signs and absence of aqueous tear production. Due to the development of clinical signs following the utilization of a sulfonamide antibiotic, intrinsic toxicity affecting the lacrimal glands was considered. Other etiologies for KCS in the dog include idiopathic diseases (presumed immune or autoimmune mediated), secretory or endocrine disorders, trauma, infection, neuropathy, chronic blepharoconjunctivitis and lacrimal duct occlusion[1,2] . Senile atrophy is a reported but controversial cause of KCS[2]. Congenital alacrima was not considered due to patient age at onset of clinical signs.

Laboratory results

A conjunctival scrapping was performed prior to utilization of vital stain. Cytological examination of modified Wright stained preparations revealed a diffuse, proteinaceous background with abundant segmented neutrophils. Occasional monocytes and non-cornified epithelial cells were noted. Coccoid bacteria were occasionally noted.

Initial treatments

Topical gentamicin betamethasone (Gentocin Durafilm, Sobering Corp., Kenilworth, NJ) TID OS was initiated for its benefit in reduction of conjunctival inflammation and control of bacterial flora. Topical cyclosporine (1%) in corn oil vehicle (Sandimmune Solution 100 mg/m*l*, Sandoz Pharmaceuticals, East Hanover, NJ) BID OS was utilized for its lacrimal anti-inflammatory activity and to reduce corneal inflammation and scarring. Artificial tear solution (Tears Renewed Solution, Akorn, Inc., Abita Springs, LA) qlHr OS, in the waking hours, with artificial tear ointment (Tears Renewed Ointment, Akorn, Inc., Abita Springs, LA) OS, at night, was instituted for aqueous tear replacement and ocular lubrication.

Re-examination

Re-examination followed in one week. Ocular discharge continued. The left nares and planum nasale were crusted and blepharospasm remained. Biomicroscopy revealed a decrease in conjunctival hyperemia and chemosis. The cornea was dull but remained free of lesions. The remaining ocular examination was unchanged. STT (I) values were 0 mm/min OS and 18 mm/minOD. Ocular medications were maintained.

Three weeks following initial examination the owner expressed frustration with the poor response and demanding medical regimen. STT (I) were 0 mm/min OS and 19 mm/min OD. Ophthalmic examination was unchanged. Lingual atropine 1% (Atropine 1% Solution, ProVet, Loves Park, IL) resulted in reflex salivation from parotid papilla bilaterally. Alternative treatments were discussed and parotid duct transposition was elected.

Surgical treatment: parotid duct transposition (PDT)

Under general anesthesia: Induction-Ketamine (Ketaset 100 mg/m*l*, AvecoCo. Inc., Fort Dodge, IA), Diazepam (Diazepam 5 mg/m*l*, Elkins-Sinn, Inc., Cherry Hill, NJ), Maintenance-Isofluorane (Forane, Anaquest, Memphis, TN), the parotid duct was catheterized with 4-0 polypropylene (Prolene 4-0, Ethicon, Inc., Sommerville, NJ). A 5 mm rim of mucosa surrounding the parotid duct papilla was dissected and a 4cm length of the parotid duct freed by blunt dissection from the oral cavity (Fig 2). A horizontal incision (8 cm) was then made over the path of the parotid duct, along the left lateral face. Following subcutaneous dissection, the parotid duct was identified and freed from masseter muscle and fascial attachments. The duct and papilla were then transposed through a subcutaneous tunnel, to the inferotemporal fornix. Papilla and surrounding mucosa were sutured to palpebral conjunctiva with 6-0 polyglactin 910 (Vicryl 6-0, Ethicon, Inc., Sommerville, NJ) (Fig 3). The posterior aspect of the

Fig 2. Oral dissection of left parotid papilla and terminal duct.

Fig 3. Parotid duct transposed to the inferotemporal fornix of the left eye.

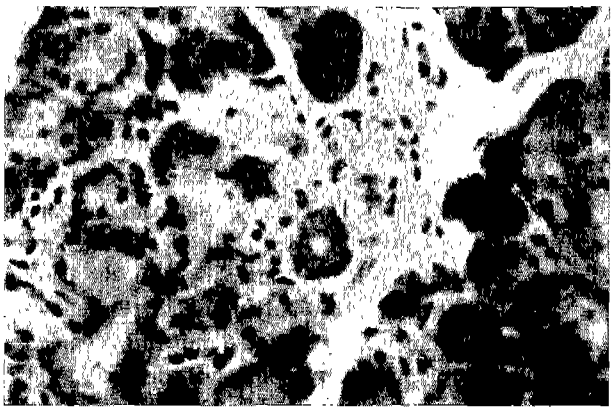

Fig 4. Histopathology of the nictitans gland. Normal acini are noted with occasional lymphocytes or plasma cells.

nictitans gland was exposed by retraction of the nictitans membrane and a 3 nm × 3 rnn wedge of gland removed by sharp dissection. The wound was not closed. The facial incision was closed with 4-0 polypropylene and a temporary lateral tarsorrhaphy of similar material placed to reduce eyelid

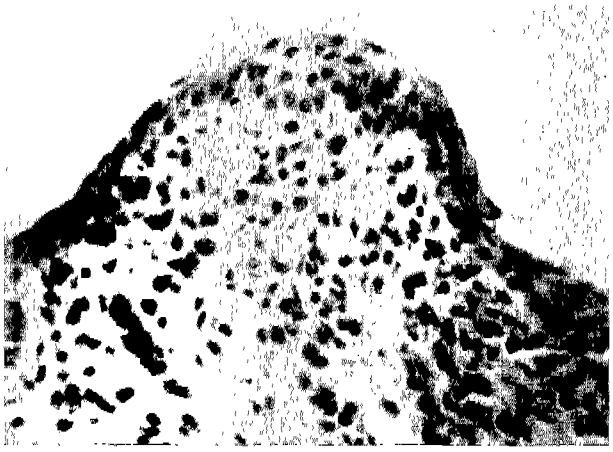

Fig 5. Histopathology of conjunctiva (400X). Lymphocytic plasmacytic interface conjunctivitis evident.

motion pending healing of the transposed papilla. The topical antibiotic corticosteroid BID OS was continued with artificial tear solution BID OS. Amoxicillin (Amoxicillin 500 mg, Rugby Laboratories, Inc., Rockville Centre LI, NY) 15 mg/kg BID PO × 10 days was used for bacterial prophylaxis. Cold compresses QID for 24 hrs, then warm compresses QID were initiated to control postoperative facial swelling. On histopathologic examination the nictitans gland was considered normal with mild fibrosis and mild infiltration of lymphocytes and plasma cells (Fig 4). Lymphocytes and plasma cells are considered normal constituents of the lacrimal interstitium, although only single or small groups of cells are noted. A mild, lymphocytic plasmacytic interface conjunctivitis was noted (Fig 5).

Progress results

Five days postoperatively the dog was re-examined following the gradual onset of left lateral facial swelling and pruritis. A firm but fluctuant swelling extended along the facial incision. A seroma was suspected. The left eye was moist. Biomicroscopy revealed fine, white, granular deposits on the corneal surface. STT (I) values were 13 mm/min OS and 20 nro/min OD. Prednisone (Prednisone 20 mg, Mutual Pharmaceutical Co., Inc., Philadelphia, PA) 0.5 mg/kg q24hrs PO and tapered x 7 days was initiated to reduce facial pruritis and a restraint collar was placed. Vitamin C 100 mg q24hrs PO was begun. Vitamin C (Vitamin C, Roxane Laboratories, Inc., Columbus, OH) has been suggested for its ability to decrease the deposition of precipitates on the cornea following PDF. Other medications were continued.

Ten days postoperatively facial swelling had decreased. Topical antibiotic corticosteroid q24hrs OS was continued. Artificial tears were discontinued and prednisone was tapered and discontinued in one week. Six weeks following surgery the face was poorly re-haired from surgical clipping. Mild

Fig 6. Appearance of the left eye six months following PDT. Epiphora is noted.

epiphora was present from the left eye with reduction in corneal deposits (Fig 6). Serum thyroxine was normal. The topical antibiotic corticosteroid q48hrs OS and Vitamin C 100 mg q24hrs PO were maintained.

Clinical diagnosis and conformation

History, ocular clinical signs and absence of aqueous tear production confirmed a diagnosis of KCS. The etiology was undetermined.

Discussion

The traditional medical management of KCS has involved the use of tear replacement (artificial tears), tear stimulants (pilocarpine), corticosteroids, and antibiotics. Utilization of topical cyclosporine (CsA) has been advocated for its lacrimomimetic effect and control of the inflammatory consequences of KCS[14,20,21,23]. Response to CsA therapy was not noted in this patient. Two to three weeks may be required, following initiation of CsA, for lacrimation to be increased[13]. The prognosis for increased lacrimation in those patients whose STT values are 0 mm/min is poor[13]. For medically unresponsive cases, surgery may be chosen. Reduction in tear film removal or provision for an alternative tear source is the goals of surgery. NL punctal occlusion, canthorrhaphy to reduce the palpebral fissure opening, and transposition of the parotid salivary duct is reported surgical alternatives.

Transposition of the parotid salivary duct may provide a continuous supply of tear substitute[3,5,7,16]. The parotid salivary gland is entirely serous, unlike the other major-paired salivary glands of the dog, with parotid secretion similar to lacrimal secretion in composition and chemistry[16]. The reported success of PDT is 63% to 80% in dogs[5,25]. Reasons for PDT failure include: 1) Lack of glandular activity, 2) Ductal injury from tension, twisting, angulation, or pressure resulting in occlusion or fibrosis, and 3) Infect ion[2,4,5,8,25]. Trauma is suspected as the most frequent cause of failure[2]. Careful surgical technique and postoperative stimulation have been suggested to maximize success. Secretions may be marked and result in epiphora. When problematic, epiphora can be reduced by alternative drainage (conjunctivorhinostomy, conjunctivobucostomy), partial surgical occlusion of the parotid duct, or denervation of the parotid gland[3]. Corneal deposits occur occasionally following PDT and are believed to be precipitates of calcium carbonate, phosphate, oxalate or combinations thereof. Utilization of topical EDTA (0.5%) solution is reported to decrease precipitate deposition[4,5].

This report describes unilateral KCS in a dog of unknown etiology who responded to parotid duct transposition.

References

1. Aguirre GD, Rubin LF, Harvey CE. Keratoconjunctivitis sicca in dogs. JAVMA 1971; 158: 1566-1579.
2. Betts DM, Helper LC. The surgical correction of parotid duct transposition failures. J Am Anim Hosp Assoc 1977; 13: 695-700.
3. Chase RA. Parotid duct transfer for total xerophthalmia. Plast Reconstruct Surg 1966; 27: 201-208.
4. Gelatt KN. Veterinary Ophthalmology, (second ed. Lee and Febiger, Philadelphia. 1991.
5. Gelatt KN. Treatment of canine keratoconjunctivitis sicca by parotid duct transposition. JAVMA 1970; 6: 1-12.
6. Gelatt KN, Peiffer JR, Erickson JL, Gum GG. Evaluation of tear formation in the dog, using a modification of the Schirmer tear test. JAVMA 1975; 166: 368-370.
7. Glen JB, Lawson DD. A modified technique of parotid duct transposition for the treatment of keratoconjunctivitis sicca in the dog. Vet Rec 1971; 210-213.
8. Harvey CE, Koch SA. Surgical complications of parotid duct transposition in the dog. J Am Anim Hosp Assoc 1971; 7: 122-126.
9. Jabs DA, Enger C, Prendergast RA. Murine models of Sjogren's Syndrome. Invest Ophth Vis Sci 1991; 32: 371-380.
10. Kaswan RL, Martin Cl, Chapman Jr., WL. Keratoconjunctivitis sicca: Histopathologic study of nictitating membrane and lacrimal glands from 28 canine cases. Am J Vet Res 1984; 45: 112-118.
11. Kaswan RL, Martin CL, Dawe DL. Keratoconjunctivitis sicca: Immunological evaluation of 62 canine cases. Am J Vet Res 1985; 46: 376-383.
12. Kaswan RL, Martin CL, Dawe DL. Rheumatoid factor determination in 50 dogs with keratoconjunctivitis sicca. JAVMA 1983; 183: 1073-1075.
13. Kaswan RL, Salisbury MA. A New Perspective on Canine

Keratoconjunctivitis Sicca: Treatment with Ophthalmic Cyclosporine. In: The Veterinary Clinics of North America. Vol. 20/Number 3. (Eds: Millichaiip, NJ; Dziezye, J) (Small Animal Ophthalmology.) W.B. Saunders Co., Philadelphia, 1990; 583-613.
14. Kern TJ. Topical cyclosporine therapy for keratoconjunctivitis sicca in dogs. Cornell Vet 1989; 79: 207-209.
15. Kern TJ, Erb HN, Schaedler JM, Dougherty EP. Scanning electron microscopy of experimental keratoconjunctivitis sicca in dogs: cornea and bulbar conjunctiva. Vet Pathol 1988; 25: 468-474.
16. Lavignette AM. Keratoconjunctivitis sicca in a dog treated by transposition of the parotid salivary duct. JAVMA 1966; 148: 778-786.
17. Martin CL, Kaswan L. Distemper-associated keratoconjunctivitis sicca. J Am Anim Hosp Assoc 1985; 21: 355-359.
18. Martin CL, Munnell J, Kaswan RL. Normal ultrastructure and histochemical characteristics of canine lacrimal glands. Ann J Vet Res 1988; 49: 1566-1572.
19. Moore CP. Qualitative Tear Film Disease. In: The Veterinary Clinics of North America. Vol. 20/Number 3. (Eds: Millicharrp. NJ; Dziezye. J) (Small Animal Ophthalmology) W.B. Saunders Co., Philadelphia, 1990; 565-581.
20. Morgan RV, Abrams KL. Topical administration of cyclosporine for treatment of keratoconjunctivitis sicca in dogs. JAVMA 1991; 199: 1043-1046.
21. Olivero DK, Davidson MG, English RV, Naisse MP, Jamieson VE, Gerig TM. Clinical evaluation of 1% cyclosporine for topical treatment of keratoconjunctivitis sicca in dogs. JAVMA. 1991; 199: 1039-1042.
22. Rubin LF, Lynch EK, Stockman BS. Clinical estimation of lacrimal function in dogs. JAVMA 1965; 147: 946-947.
23. Salisbury MA, Kaswan RL, Ward A, Martin CL, Ramsey JM, Fischer CA. Topical application of cyclosporine in the management of keratoconjunctivitis sicca in dogs. J Am Anim Hosp Assoc 1987; 26: 269-274.
24. Sansom J, Barnett KC, Long RD. Keratoconjunctivitis sicca in the dog associated with the administration of salicylazosulphapyridine (sulphasalazine). Vet Rec 1985; 116: 391-393.
25. Schmidt G, Magrane WG, Helper LC. Parotid duct transposition: a follow-up study of 60 eyes. J Am Anim Hosp Assoc 1970; 6: 235-241.
26. Shabo AL. Kenyon KR, Franklin RM. Electron microscopic localization of a blood-tear barrier to tracer proteins in the primate lacrimal gland. Lab Invest 1973; 28: 185-193.
27. Slatter DH. Disorders of the lacrimal system. Part I. Deficiency of precorneal tear film. Camp Cont Educ Pract Vet 1980; 2: 801-807.

개의 건성 각결막염과 수술적 처치

우 흥 명
위스콘신 수의과대학 외과학교실

요 약 : 건성 각결막염(KCS)은 임상에서 많이 접하게 되는 개의 안과 질환이다. 눈물샘으로부터 수성층의 눈물생산이 억제되거나 정지되어 각막 표면 눈물층의 양적변화와 함께 KCS가 유도된다. 본 연구는 KCS로 잠정진단을 받아, 본 대학병원에 의뢰된 개를 대상으로 조직 병리학적 변화를 검사하고, KCS의 진단 및 내과적 치료 방법들을 검토하였으며, 내과적 치료에 반응하지 않은 예를 외과적 처치인 parotid duct transposition(PDT)으로 치료하는 방법을 서술하고자 한다.