II- DRY EYE DISEASE AND PIGMENTARY KERATITIS IN BRACHYCEPHALIC DOGS - A BRIEF REVIEW

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INTRODUCTION

A number of ocular disorders have been described in the brachycephalic breeds, and some are related to the anatomical peculiarities of these breeds. Common ocular pathologies include entropion, distichiasis, trichiasis, kerato-conjunctivitis sicca (KCS), corneal pigmentation (CP), corneal erosion, corneal ulceration, corneal perforation and corneal vascularization (CV) (Williams 2008; Labelle *et al*. 2013)

In a retrospective study, Krecny *et al*. (2015) correlated the ophthalmologic findings with age, gender, presenting signs and time of onset of disease. In total of 130 pugs examined, ocular abnormalities identified included keratoconjunctivitis sicca (KCS) (39), macroblepharon (258), entropion (258), distichiasis (56), ectopic cilia (8), conjunctivitis (88), corneal pigmentation (CP) (101), opacity (63), ulceration (46), vascularization (35), iris–iris persistent pupillary membranes (PPM) (21) and cataract (18). KCS was significantly associated with the presence of corneal pigmentation. However corneal pigmentation was also identified in pugs (61) without KCS.

Labelle *et al*. (2013), in a study, examined 295 pugs and detected corneal pigmentation (CP) in at least one eye of 243 of the 295 (82.4%) pugs.

Dry eye disease and pigmentary keratitis

Dry eye and pigmentary keratitis are multifactorial disorders of the tears and ocular surface, associated with discomfort and visual disturbance. Clinical features of dry eye include an unstable tear film, ocular surface inflammation and epitheliopathy like pigmentary keratitis and ulceration. Drug therapy is not always effective. Hence, new treatment alternatives are needed.

The main causes of dry eye are, de-

creased tear production, increased tear evaporation or instability of the corneal tear film. Dry eye caused by decreased tear production could be due to Sjogren Syndrome and more commonly due to the use of certain medications such as anti-cholinergic agents or antihistamines. Increased tear evaporation could be caused by lagophthalmos. Meibomian gland dysfunction is a very common condition which prevents secretion of the superficial lipid layer. Loss of goblet cells due to autoimmune or inflammatory diseases such as Steven Johnson Syndrome or Ocular Cicatricial Pemphigoid is another culprit. (Behrens *et al*., 2006)

Pigmentary keratitis or non- ulcerative keratitis is the inflammation of cornea with deposition of melanin pigments. It is most commonly associated with conditions like chronic superficial keratitis, KCS, distichiasis, trichiasis and chronic ulcerative keratitis (Gelatt,2014)

Pathogenesis

Brachycephalic breeds are more prone for dry eye and pigmentary keratitis due to their characteristic short nose, excessive nasal folds and protruded eyeball. Apart from this, their tears may not get spread completely across the cornea, resulting in evaporative tear loss, particularly at centre of cornea, and resultant exposure keratitis. Such prolonged exposure of cornea results in dry eye - particularly at central cornea, which leads to scarring and formation of black pigment layer (pigmentary keratitis) leading to loss of vision (Gunderson.2013)

A reduction in the tear film may result in compensatory conjunctival cell hyperplasia and increased mucin production. Additionally, at least in the acute phase, the tear film becomes more hypertonic, leading to dehydration of the ocular surface epithelium (corneal and conjunctival), in turn resulting in oedema, vacuolar degeneration, and generalized thinning of the corneal and conjunctival epithelium. Corneal epithelial cells are more readily exfoliated by the greater friction associated with blinking over a roughened, keratinized conjunctival epithelium. Epithelial erosion or corneal ulceration may then occur, leading to substantial ocular pain due to exposure of the trigeminal nerve endings in the cornea. The corneal epithelium thickens and keratinises and inflammatory cells and blood vessels infiltrate the anterior corneal stroma and pigment, lipid, and calcium may be secondarily deposited in the cornea. Loss of antimicrobial substances normally suspended in the aqueous portion of the tear film (IgA, lysozyme) predisposes the dry eye to secondary bacterial and sometimes fungal infections. (Miller, 2013)

Corneal pigmentation is a kind of non-specific response of cornea to inflammation of varying degrees (Slatter, 2003). Melanosis of corneal stroma occurs as a result of migration of melanocytes from the limbal and perilimbal tissues. Melanin pigments are carried by macrophages and fibroblasts and this will be accompanied by corneal vascularization, migration of inflammatory cells to corneal stroma and formation of granulation tissue. These pigments then get deposited in the corneal epithelial cell and stromal tissue (Gelatt, 2014). With exposure to irritation caused by medial canthal entropion or nasal fold trichiasis or distichiasis or absence of precorneal tear film, the cornea may become pigmented. (Slatter, 2003). Removal of the stimulus for inflammation often prevents the progression of pigmentation (Gunderson, 2013)

Diagnosis

In order to establish the correct diagnosis, the following diagnostic tests should be performed: STT (Schirmer tear test), TBUT (Tear break up time), Rose Bengal test, biomicroscopic examination with a slit lamp, altogether in combination with the clinical picture (Van–Bijsterveld, 1990; Nelson, 1994; Maggs, 2008). The most recognizable clinical sign of KCS is the presence of a mucopurulent–like eye discharge (Martin, 2010).Testing of palpebral light reflex can be useful in the diagnosis of lagophthalmous. Similarly, menace, dazzle and pupillary light reflex give an idea about the animal vision. Also the affected eye should be carefully observed for the presence of distichiasis, trichiasis, entropion, ectropion, or any kind of abnormal eyelid conformation. Corneal cytology and biopsy reveal any infectious origin for the condition, and culture and ABST helps in choosing the right antimicrobial agent for the condition (Gunderson, 2013).

Treatment

Barnett and Sansom (1987) described the treatment of KCS in dog. They explained various types of medical treatment, including replacement therapy with artificial tears, ocular inserts, other preparations; lacrimogenics, other forms of medical treatment including antibiotics, corticosteroids, mucolytics and hormones. Surgical treatment included punctal occlusion, temporary tarsorrhaphy, conjunctival flaps, contact lenses, superficial keratectomy as well as parotid duct transposition.

Recently, transplantation of oral mucosa as an autograft into the conjunctival fornix to treat severe dry eye syndrome has shown satisfactory results in experiments in humans and dogs. The secretions from the minor salivary glands of the oral muco-

sal transplant will serve as an alternative for tears, lubricating the ocular surface and thus treating dry eye syndrome. The lower punctal occlusion prevents the drainage of secretions of the mucosal autograft and tears through the nasolacrimal duct and thus helps to restore moistness on the eye. Cornea, in most cases of dry eye disease would either be melanotic or ulcerated and vision will get impaired. Almost all the treatments for pigmentary keratitis are directed to control the progression of pigmentation by removing the cause of irritation (Gunderson, 2013). Medical management includes application of cyclosporine or tacrolimus (immunosuppressant drugs), judicial application of steroids and anti-inflammatory agents. Beta -irradiation, carbon dioxide laser ablation, lamellar keratectomy and nasal canthoplasty are the most commonly reported surgical corrective measures for pigmentay keratitis. Another technique is to subject the melanocytes to very low temperature, the technique is termed as cryotherapy. As melanocytes are highly sensitive to cryotherapy the technique is being used by veterinary ophthalmologists for the treatments of corneal pigmentation and conjuctival or scleral melanoma (Moreau *et al*., 1971).

Facial fold resection and medial canthoplasty helps in correcting the medial canthal entropion, nasal fold trichiasis and reduction in the palpebral fissure and thereby reducing the constant stimulas of irritation to eyes (Gunderson, 2013).

Superficial keratectomy is a surgical removal of the pigmented corneal layer. Pigmented epithelium, basement membrane and part of the underlying pigmented epithelium are surgically removed in this technique. But the main complication reported is scar formation and repigmentation (Anoop *et al*., 2015). After the superficial keratectomy, lamellar keratoplasty is practiced for corneal remodelling. It is the surgical replacement of the entire cornea with collagen based biomaterials.

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