

**EVALUATION AND MANAGEMENT OF EYE
AFFECTIONS IN CHINESE PUGS**

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KERALA, INDIA
2012**

EVALUATION AND MANAGEMENT OF EYE AFFECTIONS IN CHINESE PUGS

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(2010-13-134)

**Thesis submitted in partial fulfillment of the requirement for the
degree of**

MASTER OF VETERINARY SCIENCE IN VETERINARY SURGERY AND RADIOLOGY

Faculty of Veterinary and Animal Sciences

Kerala Veterinary and Animal Sciences, Pookode, Wayanad

2012

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KERALA, INDIA

DECLARATION

I hereby declare that this thesis, entitled “**EVALUATION AND MANAGEMENT OF EYE AFFECTIONS IN CHINESE PUGS**” is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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CERTIFICATE

Certified that this thesis, entitled **“EVALUATION AND MANAGEMENT OF EYE AFFECTIONS IN CHINESE PUGS”** is a record of research work done independently by **SHERIN B. SARANGOM, (2010-13-134)** under my guidance and supervision and it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to him.

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EXTERNAL EXAMINER

ACKNOWLEDGEMENTS

*I was indeed fortunate to have **Dr. Syam. K. Venugopal**, Associate Professor and Head, Department of Veterinary Surgery and Radiology, as my major advisor. I attribute the level of my Masters degree to his encouragement and effort and without him this thesis, too, would not have been completed or written. One simply could not wish for a better or friendlier supervisor. I surmise it a rare privilege to work under his counsel and indomitable spirit. His sense of dedication to duty, personal discipline and novel approach has never failed to inspire me as a scholar.*

*With profound gratitude and respect, I would like to depict my deep sense of obligation and heartfelt indebtedness to **Dr. John Martin K. D.**, Associate Professor and Head, Department of Veterinary Surgery and Radiology. I am grateful to him for the guidance, whole-hearted support, and affectionate encouragement rendered to me throughout my research work.*

*I would like to place on record my acknowledgment to **Dr. M. K. Narayanan**, Assistant Professor Department of Veterinary Surgery and Radiology for the constant encouragement and interest in my work.*

*With exquisite pleasure I express my deep thanks to **Dr M. Mini**, Professor and Head, Department of Veterinary Microbiology and member of the advisory committee for her valuable guidance, care, encouragement and timely help.*

*I am obliged to **Dr. S. Anoop**, Assistant Professor, Department of Veterinary Surgery and Radiology for his services offered during my course of study amidst his hectic schedule of academic period.*

*My deepest affection to **Dr. Sarada Amma**, Department of Veterinary Surgery and Radiology who until her day of retirement had kind concern and consideration regarding my academic requirements.*

*My sincere gratitude to **Dr. C. B. Devanand** and **Dr. K. Rajankutty**, Department of Veterinary Surgery and Radiology for their valuable contributions rendered during my course of study.*

*I am indeed overwhelmed and grateful to **Dr. B. Bibin Becha** and **Dr. Hiron M. Harshan**, Assistant Professors of Department of Animal Reproduction Gynaecology & Obstetrics, for their valuable guidance, encouragement and cooperation.*

*I am extremely thankful for the lively association and affection showered by my seniors **Dr Shanthi** , **Dr. Firdous** and **Dr. Chinhu**.*

*Many thanks go in particular to **Nithina K. Baburaj** and **Basheer Ahmed Khan** for being wonderful colleagues in the department.*

*I warmly remember the help and support given to me by my junior colleagues, **Drs. Ashay, Bini Sharanya** , **Susannah, Anaihitha, Manju** and **Edison***

*Nothing will be sufficient to express my deep sense of gratitude to **Dr. Vamshi Krishna, Dr. Abhijeet** and **Dr. Dhanush** for sparing their valuable time and helping me in the completion of my thesis.*

*No words can ever express my heartfelt thanks to my dear friends **Mittu, Vinu, Dhanesh, Vimal, Johnson** for their help in different forms.*

*I remember with gratitude the help and support rendered by **Dr. Ammu** and **Dr. Ambili**.*

*My special thanks to **2007 batch** students for their wonderful association in the clinics.*

*I am also thankful to the **nonteaching staff** of the department.*

*I owe a special word of thanks to **Dr. Divya mol Thomas** and **Dr. Priya**, M. V. Sc students of Madras Veterinary College for their valuable help and support for collecting research articles.*

*I warmly remember the company of my juniors and active blue cross members **Aswin, Basil, Vishnu, Karthika, Aiswarya and Varsha**.*

*Special thanks for **Dr. Terrence B. Remady** who has been my inspiration during my internship period, whose association has instilled in me the confidence to pursue my ambitions in life.*

*My deepest gratitude goes to my family for their unflogging love and support throughout my life. I wouldn't have come this long way if not for **my parents**, who has been my motivation to overcome many a hurdles during the study. I would like to express my love to **younger sister** who make our home lively.*

*Last but not the least, one above all of us, the omnipresent God, for answering my prayers for giving me the strength to plod on despite my constitution wanting to give up and throw in the towel, thank you so much **Dear Lord**.*

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Dedicated to my Family
& Teachers

Introduction

1. INTRODUCTION

The population of Chinese pugs has increased tremendously in Kerala during the past few years with the popularity of this toy breed among pet lovers. This has led to considerable rise in hospital population. Recently, a noticeable number of pugs are presented with ocular diseases which are often severe and threaten the integrity of the globe. The conditions noticed in Chinese pugs include glaucoma, pigmentary keratitis, ulcerative keratitis and subsequent complications like descemetocoele, staphyloma and iris prolapse. Immediate and appropriate medical and/or surgical treatment may be required in most of the cases, which may otherwise end up in loss of vision.

Even though ocular emergencies are primarily due to trauma or infections, breed predisposition and anatomical peculiarities exaggerate the conditions in Chinese pugs. The over-presentation of this breed with ophthalmic disease conditions may be attributed to these significant peculiarities.

A number of anatomical features combine to result in potentially blinding disease in Chinese pugs. These brachycephalic breeds possess a pronounced globe position, shallow orbits and large palpebral fissures (Bedford, 1987 and Tolar *et al.*, 2006). Also, the inherited corneal insufficiency, poor corneal reflex and lack of protective eye consciousness predispose this breed to neurotrophic ulcers especially on central cornea (Startup, 1984). In addition, the poor corneal sensitivity in them compared to mesocephalic individuals further decrease their tendency to blink (Blocker and Woerdt, 2001) and lead to exposure keratopathy (Hartley, 2010). These factors are often exacerbated with the presence of lid and adnexal abnormalities like medial lower lid entropion, hairy caruncle at medial canthus, prominent nasal folds which may cause trichiasis, concurrent keratoconjunctivitis sicca (Whitley *et al.*, 1991), immunosuppressive diseases such as demodicosis, dermatophytosis and renal failure (Prado *et al.*, 2005) and bacterial involvement (Sweeny and Irby, 1996).

Generally, the clinical signs of ocular diseases in brachycephalic breeds include epiphora, corneal opacity, corneal neovascularisation and corneal pigmentation (Renwick, 2007). Such patients may be subjected to thorough general, clinical and specific ophthalmic examination for assessing the visual function, anatomical and physiological derangements and to rule out the presence of any systemic diseases (Wilkie and Whittaker, 1997; Felchle and Urbanz, 2001 and Mandell and Holt, 2005).

Ideally, the most important step in the treatment of ophthalmic affections is to determine and eliminate the cause, followed by attempts to create an ideal environment for the repair of the lesions. In addition to the topical and systemic antibiotics, anti-inflammatory agents, tear substitutes, cycloplegics, collagenase inhibitors and vitamins are included in the medical therapy (Wilkie and Whittaker, 1997). Most often, superficial corneal lesions resolve in five to seven days with medical care, while the deep and full thickness defects take longer time to heal and may end up in loss of vision. In such cases, a timely surgical intervention provides better results and maintains visual function. The surgical treatment includes debridement and various keratectomy and transposition techniques. The different bandaging procedures include use of contact lenses, collagen shields, cyanoacrylate tissue adhesives, temporary tarsorrhaphy, third eyelid flap, and conjunctival flaps (Moore, 2003). In full thickness corneal lacerations, direct suturing can be practiced (Herring, 2003).

There can be functional diseases like increased intraocular pressure resulting in glaucoma in brachycephalic breeds. Various categories of antiglaucoma drugs are available for managing such conditions. For refractory cases of glaucoma, several surgical techniques are also described in literatures (Miller, 2003 and Palmer *et al.*, 2008).

The brachycephalic breeds of dogs with prominent eye ball may be prone to corneal drying on account of over exposure and result in tear film deficiency diseases. Such a condition may be managed with medical therapy as well as with surgical techniques to reduce the corneal exposure. Similarly adnexal

abnormalities may also be rectified surgically (Renwick, 2007 and Townsend, 2007).

The treatment of ocular diseases in Chinese pugs usually requires either single or a combination of these relatively straightforward techniques to achieve long term visual function and patient comfort. Also, the knowledge on breed predispositions and heritability of ocular diseases in Chinese pugs are important for accurate diagnosis, prognosis and to offer genetic counseling.

Hence a comprehensive study is undertaken with the following objectives.

1. To study the occurrence of different conditions of eye in Chinese pugs.
2. To study the clinical signs associated with each condition and the effectiveness of treatments adopted.

Review of Literature

2. REVIEW OF LITERATURE

2.1 AFFECTIONS OF EYE IN CHINESE PUGS

2.1.1 Incidence

Krawitz (1963) observed the higher incidence of ocular problems in short headed dogs.

Bedford (1987) pointed out the importance of knowledge of breed related incidence of various ocular diseases in diagnosing them.

In a study to evaluate the biomicroscopy of tear film in brachycephalic dogs, marked exophthalmos and a variable degree of medial pigmentary keratitis was noticed in all the dogs (Carrington *et al.*, 1989).

Whitley *et al.* (1991) reported that breeds like Chinese pugs, Bull dogs, Lhasa apso, Terriers, Shih Tzu and Pekingese were overrepresented with eye affections due to keratoconjunctivitis sicca (KCS).

In a study on prevalence of breed related glaucoma in North America, Gelatt and Mackay (2004a) reported the prevalence of glaucoma in Chinese pugs to be 0.42% during the year 1974-'83 and 0.97% during the year 1984-'93.

In a retrospective study on bacterial keratitis in 85 dogs, 66% were brachycephalic breeds. 8% of the total dogs comprised Chinese pugs and all the dogs with tear production less than 15 mm/min were brachycephalic breeds (Tolar *et al.*, 2006)

Resmi (2008) reported rise in intraocular pressure in most of the brachycephalic breeds like Chinese pugs presented with corneal affections.

Wang *et al.* (2008) reported the increased incidence of *Pseudomonas*

aeruginosa associated keratitis in brachycephalic dogs of China (70%).

Williams (2008) reported that KCS due to central area of tear film deficiency or increased tear loss through evaporation as a result of tear film lipid deficiency were noticed in brachycephalic dogs with lagophthalmos like Chinese pugs.

In a retrospective study of ulcerative keratitis in dogs of Korea, out of the 32 dogs that were presented, 75% were brachycephalic breeds (Kim *et al.*, 2009).

Ledbetter *et al.* (2009) reported increased incidence of *Pseudomonas aeruginosa* infected ocular affections in brachycephalic dogs (60%).

Chandler *et al.* (2010) reported the increased incidence of refractory corneal ulcers in brachycephalic dogs. Out of the 89 dogs presented, 32 (35.9%) were brachycephalic breeds.

Chinchu (2010) reported that incidence of corneal ulceration was more common in brachycephalic breeds like Chinese pugs.

Bharathi *et al.* (2011) reported increased incidence of eye affections in Chinese pugs. Among the 110 dogs with ocular affections studied, 10.93% were Chinese pugs. The occurrence of ocular diseases like glaucoma in Chinese pugs comprised 10.9%.

Venugopal (2011) reported rise in intraocular pressure in all the Chinese pugs presented with corneal affections.

2.1.1.1 Breed predisposition

Bedford (1982) observed predisposition of corneal ulceration in Pekingese and keratitis pigmentosa in Chinese pugs.

According to Startup (1984), breeds with large corneal surfaces like Pugs and Pekingese had inherited corneal insufficiency, poor corneal reflex and a lack

of protective eye consciousness. These breeds were predisposed to nerve deficiencies especially on central corneal areas, leading to neurotrophic ulcers.

The combination of shallow orbit and a large palpebral fissure predisposes brachycephalic breeds to complete prolapse of the globe through the palpebral fissure on orbital trauma (Bedford, 1987).

According to Carrington *et al.* (1989), brachycephalic dogs were predisposed to chronic corneal damage, pigmentary keratitis and episodes of central corneal ulceration which might progress because of bacterial involvement.

Whitley *et al.* (1991) reported that Chinese pugs were at greater risk of developing KCS.

Wolfer and Grahn (1994) stated that ulcerative keratitis was common among the brachycephalic breeds, as they were predisposed to corneal trauma due to globe prominence, lagophthalmos, and decreased corneal sensitivity. This might account for the frequency with which melting corneal ulcers were seen in these breeds.

Kaswan *et al.* (1995) reported that Chinese pugs were predisposed to KCS.

Whitley *et al.* (1995) stated the importance of knowledge on breed predispositions and heritability of ocular diseases for accurate diagnosis and prognosis. He reported that pure bred breeds like Chinese pugs were predisposed to aberrant dermis, caruncular trichiasis, corneal dystrophy, distichiasis, entropion, exposure keratopathy, lagophthalmos or exophthalmos or euryblepharon, nasal fold trichiasis, pigmentary keratitis, progressive retinal atrophy, traumatic proptosis, trichiasis and ulcerative keratitis.

According to Herrera (2005), the brachycephalic breeds were predisposed to prolapse of membrane nictitans gland.

The pronounced globe position of brachycephalic dogs predisposed the eye to ocular traumatic proptosis and exophthalmia, secondary to the shallow orbit (Tolar *et al.*, 2006).

Bouhanna *et al.* (2008) opined that ulcerative keratitis in brachycephalic dogs was usually recurrent and located in a central or paracentral corneal position.

2.1.1.2 Age

According to Magrane (1977), some Pekingese and pug blood lines showed congenital predisposition to corneal pigment deposition at an early age.

Prolapse of the third eyelid gland occurs more commonly in puppies than in adults (Christmas, 1992).

Bussieres *et al.* (2004) reported corneal perforation with anterior synechia in a Chinese pug aged 2 months.

Out of the total number of Chinese pugs presented with glaucoma during the period 1984-'93 in North America, the percentage of dogs belonging to the age groups 2 to 6 months, 6 to 12 months, 1 to 2 years, 2 to 4 years, 4 to 7 years, 7 to 10 years, 10 to 15 years and above 15 years were 13.04%, 13.04%, 17.4%, 8.7%, 8.7%, 8.7%, 26.08% and 4.34% respectively (Gelatt and Mackay, 2004).

SCCEDs in dogs were typically found in middle-aged dogs of all breeds (Bentley, 2005).

Kato *et al.* (2006) reported the early onset of glaucoma in Chinese pugs, compared to other breeds of dogs.

Raji (2006) reported the higher incidence of corneal ulcer in Chinese pugs between 3.5 months to 24 months of age with an average age of 14.4 months.

Crispin *et al.* (2008) observed that age at which glaucoma developed

varied with breeds of dogs, but the disease was mostly of middle aged dogs.

In a study of *Pseudomonas aeruginosa* associated keratitis in dogs of China which included 70% brachycephalic breeds, the age of the dogs ranged from 28 to 108 months (Wang *et al.*, 2008).

Williams (2008) pointed out that brachycephalic breeds like Chinese pugs, bull dog and Pekingese with lagophthalmos were at increased risk of tear deficiency with old age.

According to Chandler *et al.* (2010), indolent ulcers were usually seen in middle aged to older dogs of all breeds.

Chinchu (2010) reported that the incidence of corneal lesions was more in puppies and juveniles of brachycephalic breeds like Chinese pugs.

2.1.1.3 Sex

As loss of sex hormones play a major role in the pathogenesis of KCS, neutered, older dogs of both sexes were predisposed to KCS (Gionfriddo, 1995b).

Slater and Erb (1986) reported that the female dogs were at twice the risk of male dogs in development of glaucoma.

Bentley *et al.* (2001) described that the clinical findings showed SCCEDs in dogs had no clear sex predilection.

In a study on prevalence of breed related glaucoma in North America, Gelatt and Mackay (2004) reported the prevalence of glaucoma in Chinese pugs by gender to be 0.71% in males and 1.2% in females with a male to female ratio of 1:0.59 during the year 1984-'93.

Bath and Dua (2006a) observed no sex predisposition to glaucoma in dogs.

Janssens (2007) stated that indolent ulcers had no sex predisposition in dogs.

Chinchu (2010) observed no sex predisposition in Chinese pugs presented with ulcerative keratitis.

2.1.2 Etiology

According to Magrane (1977), as the dog does not possess complete bony orbit, the globe is more prone to physical trauma.

According to Startup (1984), the avascularity of cornea will reduce the surface temperature by 0.5-1°C and breeds with large corneal surface are likely to show less resistance to corneal infection and ulceration because of lowered temperature and sensitivity.

Carrington *et al.* (1989) opined that excessive globe prominence, decreased corneal sensitivity, lagophthalmos, low rate of complete blinks and physical abrasion by nasal folds and distichiasis were the major factors in the pathogenesis of keratitis and corneal ulcerations in brachycephalic dogs.

Kaswan *et al.* (1995) opined that in brachycephalic dogs, especially Chinese pugs that could not blink effectively, excessive corneal exposure greatly exaggerated the severity of corneal lesions and even resulted in dense pigmentation and corneal ulcers.

Berger and King (1998) opined that KCS typically affected smaller dogs since they have inherently lower basal tear production.

Blocker and Woerdts (2001) pointed out that brachycephalic dog breeds have diminished corneal sensitivity compared to mesaticephalic and dolichocephalic breeds.

According to Moore (2003), lagophthalmos, abnormal eyelid conformation or cranial nerve deficit could lead to exposure keratitis and

SCCEDs.

Mandell and Holt (2005) pointed out that although corneal ulcers were traumatic in origin, eyelid confirmation or an underlying disease process might predispose an animal to corneal ulcer. They enlisted the causes as trauma, foreign body, infection, KCS, topical irritants, exposure keratitis and entropion.

Tolar *et al.* (2006) opined that brachycephalic breeds have decreased corneal sensitivity and lacked many of the protective mechanisms compared to mesocephalic or dolicocephalic breeds making them vulnerable to ocular trauma. Brachycephalic confirmation, tear film deficiency and use of corticosteroid increased the risk of ocular trauma and decreased healing, enhancing bacterial infection in them.

Renwick (2007) pointed out that brachycephalic breeds possess shallow orbits and large palpebral fissures. Their relative exophthalmos in addition to poor corneal sensitivity might result in poor blinking. The lid abnormalities like entropion and nasal fold trichiasis along with exposure due to lagophthalmos, poor tear film distribution and presence of foreign materials resulted in corneal pigmentation, scarring, neovascularisation and ulceration in a ventro-medial position which might be further worsened by concurrent KCS.

Resmi (2008) associated the rise in intraocular pressure to the development of corneal lesions in brachycephalic breeds like Chinese pugs.

Williams (2008) pointed out that in brachycephalic dogs, lagophthalmos lead to central area of tear film deficiency or deficiency in tear film lipids lead to increased tear loss through evaporation resulting in keratoconjunctivitis sicca, which might further result in corneal vascularisation, pigmentation and frank ulceration.

Chinchu (2010) opined that the overrepresentation of brachycephalic breeds like Chinese pugs with corneal affections was associated with the rise in intraocular pressure and lagophthalmos.

Hartley (2010) opined that exposure keratopathy might occur because of the inability to complete a blink reflex due to lagophthalmos. Also, the palpebral reflex might be absent due to a neurological deficit as in trigeminal or facial nerve paralysis or due to mechanical restriction of the eyelids.

2.2 OCULAR AFFECTIONS IN CHINESE PUGS

2.2.1 Keratitis

Wilkie and Whittaker (1997) opined that corneal diseases were common in dogs and might result in opacification, vascularization, pain, ulceration, pigmentation or perforation.

Ollivier (2003) stated that bacterial keratitis in dogs and cats were usually presented as ulcerative keratitis.

According to Bath and Dua (2006b), being exposed to environment, cornea was vulnerable to diseases. Keratitis comprised an important proportion of ocular complaints in dogs.

2.2.1.1 *Ulcerative keratitis*

According to Startup (1984), any break in the continuity of the intact corneal epithelium allowed contamination by microorganisms resulting in ulceration.

The glycogen on the corneal epithelium act as the main source of energy under stressful conditions like trauma or surgical wounds and therefore, if the glycogen stores get depleted, normal healing of epithelium and cellular locomotion over the surface would be inhibited (Gum, 1991).

Wilcock (1993) observed that corneal injury in dogs was resulted from physical or chemical trauma, microbial agents and increased intraocular pressure and rarely from inborn errors of metabolism.

Brooks and Ollivier (2004) considered corneal ulceration as a disorder of proteinase homeostasis i.e., over expression of certain destructive proteinases and reduction in antiprotease activity leading to rapid degradation of collagen and other components of the corneal extracellular matrix.

Pardo *et al.* (2005) opined that immunosuppressive diseases such as demodicosis, dermatophytosis and renal failure increased the bacterial load in eyes with corneal ulcer.

Mandell and Holt (2005) described corneal ulcers as ocular emergencies that involves the loss of corneal stroma in addition to loss of epithelium and accompanied a variable degree of reflex uveitis.

Ledbetter *et al.* (2006) opined that morphologic and neurological abnormalities of the eyelids, aberrant eyelashes or facial hair, quantitative or qualitative tear film abnormalities, deficiency of corneal innervations, foreign bodies and microbial infection contributed to the occurrence of corneal ulcers.

Kim *et al.* (2009) described ulcerative keratitis as one of the most common ocular problems leading to pain and vision loss in man and dogs.

2.2.1.1.1 Classification

According to Renwick (1996) ulcerative disease may be subdivided into erosions which involved only the corneal epithelium and ulcers which additionally involved varying depths of the underlying corneal stroma.

Whitley (2000) classified corneal ulcers by the depth of corneal involvement as superficial, deep stromal, and descemetocoele. Superficial corneal ulcers were further classified as uncomplicated, progressive, or refractory. Deep stromal ulcers were divided into progressive and non-progressive types.

Moore (2003) classified corneal ulcers based on ease of healing as complicated, uncomplicated, refractory, and progressive.

Gilger *et al.* (2008) classified ulcerative keratitis by the depth of corneal involvement as superficial corneal ulcer, deep stromal corneal ulcer, descemetocoele and corneal perforation and by the underlying causes as bacterial, fungal, traumatic, immune mediated, indolent ulcers etc.

2.2.1.1.1.1 *Superficial ulcers*

Wilcock (1993) opined that the loss of corneal epithelium stimulated tear imbibitions and that caused superficial stromal oedema, followed by immigration of neutrophils from the tear film and the limbus collagenases, proteases and stimulatory cytokines released by neutrophils caused progression of conjunctival reactions like hyperemia, cellular exudation and lymphofollicular hyperplasia.

Wilkie and Whittaker (1997) opined that superficial ulcers were more painful than deep ulcerations as the anterior stroma and corneal epithelium were innervated by long ciliary nerves that arise from the ophthalmic branch of the trigeminal nerve.

Murphy *et al.* (2001) opined that SCEEDs in dogs were a reflection of alteration in corneal innervation and substance P.

Moore (2003) opined that superficial ulceration involved epithelium and basement membrane, with minimal or no involvement of corneal stroma.

Uncomplicated superficial ulcers were usually not infected and healed rapidly within a few days with minimal scar formation, but persistent ulcers healed slowly and showed tendency to recur (Ollivier, 2003).

According to Mandell and Holt (2005), superficial corneal defects were relatively clear in the cornea and some were visible only with fluorescein dye. Uncomplicated ulcer should heal by re-epithelisation within three to five days.

2.2.1.1.1.2 *Indolent ulcers*

Miller (1996) stated that persistent corneal erosions were characterized by

prolonged and often incomplete healing, resulting from a failure of the epithelial cells to form firm attachments to the underlying basement membrane and ultimately to the corneal stroma.

Whitley (2000) reported that refractory ulcers took weeks to months to heal and recurrence was common.

According to Moore (2003), disorders which interfere with epithelialisation, basement membrane formation, or adherence between epithelial cells, basement membrane and stroma were reasons for indolent corneal ulcers. These ulcers were diagnosed by their classic appearance of a persistent, superficial ulcerations with a non-adherent epithelial border (epithelial lip) without stromal involvement.

Bentley and Murphy (2004) opined that damage to the higher sensory innervations of cornea resulted in altered wound healing, persistent corneal defects or ulcers.

Bentley (2005) described the clinical appearance of an indolent ulcer as superficial, non-infected erosion, surrounded by a sheet of non-adherent or loose epithelium.

Carter (2009) described that Recurrent epithelial erosions (REEs) or indolent ulcers were characterized by a lack of adherence of the migrating corneal epithelial cells to the underlying stroma.

2.2.1.1.1.3 Deep stromal ulcers

According to Wilkie and Whittaker (1997), superficial ulcers might turn into deep stromal ulcers because of infections; tear film and eyelid abnormalities, inappropriate use of corticosteroids or topical agents etc.

Whitley (2000) pointed out that progressive deep stromal ulcers in dogs were potentially a threat to globe and vision. Hence, a detailed investigation

should be carried out.

Deep ulcerations extend to one-half the stromal depth or greater, required corneal vascularisation for healing. The healing time was about three weeks. It was observed that these ulcers might lead to infected stromal ulcers and descemetocoeles (Moore, 2003).

Ollivier (2003) stated that the complicated deep ulcers might lead to impaired vision because of corneal scarring or anterior synechia, or lead to loss of the eye because of endophthalmitis, glaucoma, phthisis bulbi, or a combination of these.

According to Mandell and Holt (2005), deep stromal ulcers required vascularisation for healing, if they involved more than one third of corneal thickness and took three weeks to heal.

2.2.1.1.1.4 *Melting ulcers*

Startup (1984) pointed out that melting ulcers or *Pseudomonas* infected ulcers caused rapid destruction of corneal tissue due to the production of collagenase and subsequent dissolution of the corneal stroma. Large areas of cornea might be badly affected, but had little tendency to perforate the cornea as collagenase did not affect the Descemet's membrane.

Melting ulcers in dogs might result from the action of proteolytic substances on the corneal stroma. These ulcers, often of traumatic origin, then infected by opportunistic bacteria, could melt in very rapidly (Wolfer and Grahn, 1994).

Whitley (2000) described melting ulcers as collagenase and protease associated ulcers. Proteases and collagenase aid in the removal of devitalized cells and debris from the cornea in the corneal healing. Corneal epithelial cells, fibroblasts, polymorphonuclear leukocytes, some bacteria (*Pseudomonas* spp.) and some fungi produced protease and collagenase. These enzymes contributed to

the progressive breakdown and rapid melting of the corneal stroma in some corneal ulcers.

Miller (2001) opined that deep stromal ulcers that were enlarging, deepening or not healing appropriately were considered to be progressive.

Melting ulcers were characterized by progressive stromal dissolution secondary to proteolytic activity due to the excessive levels of proteases resulting from imbalances between proteases and protease inhibitor levels (Vanore *et al.*, 2007).

2.2.1.1.1.5 *Descemetocele*

Startup (1984) described descemetocele as a complication of ulcerative keratitis with exposed Descemet's membrane impending perforation of the globe. Being elastic in nature, Descemet's membrane usually projects forward as descemetocele recognized by lack of fluorescein dye retention.

Wilkie and Whittaker (1997) defined descemetocele as deep corneal ulcers which failed to retain fluorescein stain in the central portion.

According to Whitely (2000), descemetocele were ocular emergencies and required immediate surgical intervention.

Miller (2001) stated that Descemet's membrane was a significant barrier to perforation from deep corneal ulcers and resistant to enzymes that denatured overlying stroma.

Descemet's membrane is always under some tension and might get herniated through the surrounding corneal stroma to bulge anteriorly to form descemetocele. Fluorescein retention occurs in the surrounding exposed stroma only. Restraint or any pressure on the neck or jugular vein might cause increased intraocular pressure and corneal rupture (Mandell and Holt, 2005).

2.2.1.1.1.6 *Staphyloma/corneal perforation*

In corneal perforation, the extrusion of iris through the corneal wound will seal the wound, but provided a potent tract to intraocular infection. Such wounds should be repaired as early as possible so that the risk of staphyloma formation and risk of infection could be reduced (Bedford, 1987).

Wilkie and Whittaker (1997) reported that corneal perforation occurred as a result of a complicated corneal ulcer. They also pointed out that the temporary seal formed by the prolapsed iris and the aqueous clot should not be disturbed until the animal was anaesthetized and the cornea was about to repair.

Sansom (2000) opined that penetration of anterior chamber caused loss of aqueous humor and acute pain and later the fibrin clot sealed the wound. In iris prolapse, a distorted pupil in the direction of corneal wound could be seen and such wounds resulted in shallowing of anterior chamber, uveitis, miosis, aqueous flare and hyphaema.

Miller (2001) pointed out enophthalmos was the serious threat after corneal perforation.

Mandell and Holt (2005) opined that infected stromal ulcers could progress quickly to corneal perforation and become a surgical emergency. Restraint or any pressure on the neck or jugular vein might cause increased intraocular pressure and corneal rupture. Seidel test could be performed to check for leakage of aqueous humor.

2.2.1.1.2 Clinical signs

Corneal ulceration might be accompanied by corneal oedema, vascularization, pain and photophobia. Epithelial abrasions were extremely painful and affected animal showed blepharospasm, lacrimation and photophobia (Startup, 1984).

Bedford (1987) opined that epiphora and blepharospasm indicated trigeminal irritation and pain associated with keratitis.

Sansom (1988) opined that purulent ocular discharge indicated a bacterial infection. Serous discharge might be due to viral or non-pyogenic bacterial infection. If there was an allergic etiology, the ocular discharge would be mucoid.

Wolfer and Grahn (1994) reported the clinical signs like pain and purulent discharge in a brachycephalic dog with melting ulcer.

Murphy *et al.* (2001) described that SCCEDs in dogs were characterized by the presence of epithelial erosion surrounded by a circumferential sheet of loosely adherent or non-adherent epithelial cells (“epithelial lip”).

Ollivier (2003) pointed out that ulcers with highly active proteases had a greyish, gelatinous appearance, which must be distinguished from corneal oedema.

Mandell and Holt (2005) mentioned the clinical signs associated with corneal perforation and it included signs like pain, lacrimation, blepharospasm, corneal oedema, a misshapen cornea, and a pink or red tissue. There might be change in the depth of anterior chamber and dyscoria. The iris might adhere to the rent in the cornea or prolapse as a brown or black mass.

2.2.1.1.3 Neovascularisation of cornea

Magrane (1977) observed vascularisation of cornea in response to different pathologic processes and in stromal healing.

Gionfriddo (1995a) pointed out that extensive corneal vascularisation might be presented as red eye and it often indicated chronic, active keratitis.

The incidence of deep corneal vascularisation depended upon the duration of ulcer and developed after three to five days (Wilkie and Whittaker, 1997).

According to Miller (2001), when the stromal architecture was disrupted, limbal blood vessels might progress towards the wound on the cornea and remained although they might not carry blood after wound healing.

Corneal blood vessel pattern varied with the type of keratitis present. Long branching vessels were consistent with a superficial ulcerative or non-ulcerative keratitis whereas, deep fine non-branching vessels were associated with deep keratitis. Deep corneal vessels forming a 360° perlimbal pattern were seen with intraocular disease like uveitis and glaucoma (Moore, 2001).

Featherstone *et al.* (2001) pointed that corneal neovascularisation was beneficial in the early healing stages of ulcerative keratitis and an excessive degree caused ocular discomfort and corneal opacity.

Uncomplicated corneal ulcers healed without vascularisation (Slatter and Dieterich, 2003).

Bussiere's *et al.* (2004) observed neovascularisation of cornea following implantation of small intestine submucosa (SIS). They opined that neovascularisation might be due to surgery, initial traumatic event and corneal repair rather than an immune rejection.

Bentley (2005) reported that central corneal lesions commonly existed weeks to months without any vascular response, whereas peripheral lesions resulted in more vascularisation of cornea.

Brunott *et al.* (2007) observed complete withdrawal of blood vessels once cornea was healed.

After a study on repair of corneal defect using SIS, Vanor *et al.* (2007) noticed corneal neovascularisation. They opined that the neovascularisation that was nearly present at the time of ulceration might be induced later by surgery and got amplified during SIS integration into the corneal stroma because of the stimulation by growth factors present within both the SIS and the cornea.

2. 2.1.1.4 Corneal opacity

According to Bedford (1982), corneal opacity was related to

inflammation, oedema and dystrophy or scar formation

Startup (1984) defined the scar that followed ulceration as nebula, if there was a faint white cloudiness; as macula, when there was a definite grey opacity and as leucoma, if there was a dense white opacity.

Corneal oedema might occur due to an acute elevation in intraocular pressure resulting from compression of stromal lamellae forcing water into epithelium. Chronically elevated intraocular pressure and megaophthalmia often damage endothelial cells and Descemet's membrane with focal striae and associated corneal oedema (Peiffer *et al.*, 1987).

Wilkie and Whittaker (1997) reported that the disruption of the regular lamellar arrangement of stroma or changes in the collagen type appeared as opacity and interfered with transparency. Corneal oedema resulted from a break in the dehydrated state of stroma maintained by corneal epithelium and endothelium. Break in the former resulted in diffuse oedema and the latter resulted in focal oedema.

According to Miller (2001), when the stroma was damaged, keratocytes were altered to form fibroblasts. Collagen manufactured by these fibroblasts gets deposited in a random manner producing scar.

Moore (2001) opined that corneal opacities were associated with corneal scarring or corneal oedema.

Herring (2003) stated that the transparency of cornea had been maintained by the high regular arrangement of collagen in combination with the smooth non-keratinized squamous epithelium and tear film, lack of blood vessels and relative dehydrated nature of cornea.

Morreale (2003) opined that the loss of clarity of cornea as the primary sign of corneal disease, which might be due to vascularisation, pigmentation, fibrosis, accumulation of cellular and non cellular infiltrate or oedema.

Rodrigues *et al.* (2006) stated that disturbance in endothelial function, which maintains a constant thickness, hydration and transparency of cornea, resulted in corneal oedema and partial or complete loss of transparency.

Corneal oedema has resulted from imbibitions of fluid by the epithelium or stroma and failure of extrusion of electrolytes by the endothelium. It was regarded as the increased water content that resulted in the increased thickness and scattering of light and reduced transparency (Gilger *et al.*, 2008).

2.2.1.2 Pigmentation of cornea/Pigmentary keratitis

Roberts (1954) reported corneal pigmentation in breeds with large and prominent eyes. He observed that pigmentation started at the corneal side of limbus and at times the pigment, usually melanin, covered the entire cornea.

Magrane (1977) observed that uveal pigment migrated through nerve and vessel opening and deposited in the corneal stroma, following the development of anterior synechiae in corneal perforation and iris prolapse.

In dogs, pigmentation of cornea was an indication of chronic inflammation or mechanical irritation such as entropion or facial fold trichiasis (Peiffer *et al.*, 1987).

Gionfriddo (1995b) stated that chronic KCS might result in pigmentary keratitis.

Kaswan *et al.* (1995) opined that in exophthalmic breeds and in breeds with periocular pigmentation such as Chinese pug, Schnauzer and Dachshund, pigmentary keratitis could be a primary problem or a devastating consequence of KCS. Free pigment granules and melanocytes could be deposited beneath the corneal endothelium. Also, in Chinese pugs, pigmentary keratitis might be so dense and might result in blindness.

Slatter and Dietrich (2003) stated that in chronic exposure, corneal

epithelium might get reverted to skin pattern with keratinisation and pigmentation.

According to Gilger *et al.* (2008), corneal pigmentation was usually associated with chronic inflammation and irritation. Corneal pigmentation resulted from migration of melanocytes from the limbal and perilimbal tissues which were deposited in the basal epithelial cells and anterior stromal tissue and usually accompanied with corneal vascularisation, stromal inflammatory cell infiltration, and granulation tissue formation.

2.2.1.3 Exposure keratitis

According to Startup (1984), ulceration might follow in conditions where the cornea was not kept moistened and protected by eyelids. Breeds with protruding eyes in addition to the poorly developed corneal reflex and lack of corneal sensitivity were susceptible to corneal drying, particularly in the central cornea, where adequate spreading of precorneal tear film might not occur.

Carrington *et al.* (1989) described tear film instability particularly over the central cornea in brachycephalic breeds with marked exophthalmic configuration. Lagophthalmos or impaired elimination of ocular mucus and debris might lead to abnormalities of the tear film lipid layer and enhanced evaporation rates over the central cornea affecting the normal healing.

In brachycephalic dogs like Chinese pugs that cannot effectively blink, excessive corneal exposure greatly exaggerates the severity of corneal lesions. Inflammation of the cornea causes the overlying epithelium to become keratinized and hypertrophic, resulting in irregular corneal surface which in turn affect the uniform redistribution and spread of tear film (Kaswan *et al.*, 1995)

Renwick (2007) pointed out that the deleterious effects of globe exposure and poor blink function might be exacerbated by the medial lower eyelid entropion and might lead to corneal pigmentation, scarring, neovascularisation and ulceration.

According to Hartley (2010), exposure of the cornea might occur due to lagophthalmos. The absence of palpebral reflex due to a neurological defect (trigeminal or facial nerve paralysis) or due to mechanical restriction of the eyelids might lead to exposure keratopathy. Such patients might benefit from temporary tarsorrhaphy placement.

2.2.1.3.1 Clinical signs

In dogs, pigmentation of cornea was an indication of chronic exposure (Peiffer *et al*, 1987).

The clinical signs associated with exposure of globe included epiphora, medial corneal pigmentation, axial grey corneal scarring, corneal neovascularisation and rapidly progressive corneal ulcers which were often axial or ventromedial in position (Renwick, 2007).

Williams (2008) reported ocular pathologic findings associated with ocular surface tear deficiency due to tear evaporation and it included corneal vascularisation, pigmentation and in severe cases, frank ulceration.

2.2.2 Uveitis

According to Startup (1984), any ulcerative condition might allow toxins to penetrate Descemet's membrane and produced anterior uveitis.

Based on anatomical grounds, uveitis could be classified as anterior uveitis (iritis, cyclitis, iridocyclitis), posterior uveitis (choroiditis, chorioretinitis) and panuveitis (the whole uveal tract). According to the characteristics of the inflammation, the uveitis was termed as granulomatous or non-granulomatous (Crispin, 1988).

Gionfriddo (1995d) mentioned that the complications and clinical signs of uveitis were due to the breakdown of blood aqueous barrier and exudation of blood proteins into the eye, which appeared as aqueous flare. This might progress

to development of hyphema, hypopyon and keratic precipitates i.e., aggregation of proteins inside cornea, which were diagnostic for uveitis. The inflammatory debris in the anterior chamber might lead to corneal oedema, cataracts, synechia, blockage of aqueous outflow and subsequently glaucoma.

Sansom (2000) reported that in addition to the infectious etiology, uveitis might occur subsequent to common ocular causes such as corneal ulceration and penetrating trauma.

Massa *et al.* (2002) described uveitis as one of the most common causes of blindness in dogs. The most common infectious organisms associated with uveitis in his study were *Ehrlichia canis* and *Blastomyces dermatitidis*. Other identified causes included protothecosis, Rocky Mountain spotted fever, *Dirofilaria immitis*, primary bacterial and fungal endophthalmitis.

Giuliano (2004) opined that uveitis was a common sequel to many ocular diseases and described the inflammatory mediators that caused uveitis in companion animals. Uveitis secondary to ulcerative keratitis was due to trigeminal nerve stimulation of the cornea and secondary axonal stimulation of inflammatory mediators like substance P, leading to increased vascular permeability, chemotaxis of neutrophils and miosis.

2.2.2.1 Clinical signs

Gionfriddo (1995) described uveitis as a component of many disease processes and mentioned the clinical signs associated with anterior and posterior uveitis. The clinical signs of anterior uveitis were miosis, aqueous flare, conjunctival and episcleral congestion, hyperemia or darkening of the iris, blepharospasm, keratic precipitates, hypopyon and hyphema, while the signs of posterior uveitis included complete or partial blindness, subretinal exudates, subretinal or intraretinal hemorrhages, retinal detachment and optic neuritis.

In ulcerative keratitis, stimulation of corneal nerves might potentiate an axonal reflex, clinically manifested as anterior uveitis with a reflex miosis, ocular

hyperemia and aqueous flare (Wilkie and Whittaker, 1997).

Massa *et al.* (2002) observed the clinical signs associated with acute and chronic uveitis. They were corneal oedema, conjunctival hyperemia, scleral blood vessel congestion, aqueous flare, hypopyon, hyphema, miosis, vitreous cellularity, chorioretinitis and hypotony. The signs in chronic uveitis included corneal oedema and neovascularisation, hypopyon, posterior synechia, keratic precipitates, hyphema, cataract formation, vitreous and retinal degeneration, retinal detachment and phthisis bulbi.

Mandell and Holt (2005) observed that the animals with anterior uveitis were presented with a painful red eye, with or without loss of vision

2.2.3 Glaucoma

Glaucoma is an elevation of intraocular pressure sufficient to damage the optic nerve and produce temporary or permanent blindness (Gionfriddo, 1995c).

According to Brooks *et al.* (1999), glaucoma is the final common pathway of a group of diseases with decreased retinal ganglion cell (RGC) sensitivity and function, RGC death and optic nerve head cup enlargement and incremental reduction in visual fields and blindness.

2.2.3.1 Classification

Glaucoma could be classified as primary or secondary, acute or chronic and open angle or closed angle (Smedes and Dubielzig, 1994).

According to Cook (1997), sub classification of primary glaucoma generally referred to the anatomic configuration of the iridocorneal angle as viewed by gonioscopy: open, narrow or congenital goniodysgenesis.

Deehr and Dubielzig (1998) classified glaucoma based on etiology as primary, which was a heritable and breed related disease with bilateral potential and secondary glaucoma, which was due to a pre-existing or concomitant ocular or systemic disease that appeared to be responsible for an alteration in aqueous humor dynamics.

Crispin *et al.* (2008) classified primary glaucoma into primary open angle glaucoma or primary angle closure glaucoma based on the gonioscopic appearance of iridocorneal angle.

2.2.3.1.1 Primary glaucoma

Ekesten and Narfstrom (1991) observed that the intraocular pressure was significantly higher in eyes with closed iridocorneal angles than in eyes with any other width of the angle.

Gionfriddo and Powel (2001) pointed out that most common cause of elevated IOP was a primary or secondary iridocorneal abnormality and the primary risk factor of optic nerve damage was elevated IOP.

According to Kallberg *et al.* (2007), etiology of primary glaucoma could be multifactorial of which mechanical, vascular and other factors may influence individual susceptibility to optic nerve damage.

Mangan *et al.* (2007) found disruptions of retinal pigment epithelium, increased permeability of vascular endothelium, accumulation of inflammatory cells and retinal swelling or thinning in canine primary glaucoma.

2.2.3.1.2 Secondary glaucoma

In inflammatory condition of eyes, the inflammatory cells migrated into anterior chamber and their presence in iridocorneal angle physically obstructed the outflow pathways leading to an elevated IOP (Peiffer and Gelatt 1980).

Gelatt (1981) opined that intumescent cataracts produced glaucoma with

enlargement of lens capsule which displaced the iris or ciliary body forward causing narrowing of iridocorneal angle.

Johnson and Miller (1990) reported that fungal diseases could cause uveitis via haematogenous route and secondary glaucoma was a frequent complication noticed in dogs.

Gelatt and Mackay (2004b) pointed out that secondary glaucomas could also occur with drugs, diseases, corneal and scleral inflammation, uveitis and trauma following ocular surgeries.

2.2.3.2 Clinical signs

Gelatt (1981) opined that the effects of elevated IOP in dogs varied with age of animal, duration and levels of IOP. The young dogs rapidly developed buphthalmia, which was reversible. He also noticed that corneal changes associated with glaucoma were influenced by the rapidity, duration and extent of elevated IOP. The corneal changes included pigmentation and ruptures in Descemet's membrane with focal irregular areas of oedema. Corneal oedema associated with glaucoma would disappear within hours after IOP normalization.

Primary glaucoma in advanced stages was often accompanied by opacities of lens considered as secondary forms of glaucoma (Barnett, 1985).

According to Helper (1989), the vortex veins within sclera were affected by initial increase in pressure and thus anterior ciliary vein would take over their work and become distended in the process resulting in episcleral vascularisation. Cupping of optic disc was not an early finding, but could be readily seen in blind clear glaucomatous eye.

In case of chronic glaucoma, the corneal endothelium may stretch, breaking Descemet's membrane and leading to striae, characterized by white streaks in the cornea (Gionfriddo, 1995c).

Cook (1997) opined that secondary lens luxation associated with glaucoma was usually posterior and incomplete resulting in an aphakic crescent within a fixed and dilated pupil.

Gelatt (1997) reported that buphthalmos developed in dogs with intraocular pressure of 40 mm of Hg or higher or over a few years when IOP was about 30mm of Hg.

Hasegawa *et al.* (2001) found retinal vessel attenuation, hyper reflective tapetum and pale optic disc in a bilateral glaucoma.

According to Woerdt (2001), the signs of acute glaucoma included decreased menace response, conjunctival and episcleral hyperemia, corneal oedema and a mydriatic and non responsive pupil. In chronic cases, buphthalmos was present and corneal oedema was less pronounced.

Mughannam *et al.* (2004) opined that mild elevations in IOP caused no observable clinical signs.

Karlberg *et al.* (2007) enlisted the clinical signs of glaucoma which included episcleral congestion, mydriatic pupil, optic nerve cup enlargement, neuroretinal rim narrowing, optic cup deepening and retinal degeneration.

The clinical signs of glaucoma were photophobia, blepharospasm, episcleral and conjunctival hyperemia, diffuse corneal oedema, serous to mucoid ocular discharge and diffuse facial pain (Palmer *et al.*, 2008).

Storm *et al.* (2011) stated the clinical signs associated with glaucoma which included episcleral congestion, corneal oedema, mydriasis, increased IOP and variable degrees of buphthalmos and optic nerve cupping along with associated signs of underlying problem.

2.2.4 Keratoconjunctivitis sicca (KCS)

Startup (1984) opined that KCS might result in corneal ulceration

subsequent to corneal drying due to lack of corneal lubrication.

Kaswan *et al.* (1995) stated that immune mediated destruction of lacrimal glandular tissue represented the largest proportion of KCS cases. Also, the dogs in which the third eyelid gland was removed to correct cherry eye had an increased incidence of KCS.

Davidson and Kuonen (2004) opined that KCS resulting from a deficiency in the aqueous component of tear film might predispose the ocular surface to infection. The normal flora of dogs suffering from KCS included many pathogenic organisms like coagulase positive *Staphylococcus* spp., β -haemolytic *Streptococcus* spp. and *Pseudomonas* spp.

According to Mandell and Holt (2005), KCS was a common cause of conjunctivitis in dogs and might lead to secondary bacterial conjunctivitis.

Hartley *et al.* (2006) opined that reduction in tear production resulted in increased corneal inflammation and mentioned the causes of KCS which included congenital tear deficiency, breed predisposition, drug induced lacrimal failure, irradiation, neurogenic diseases, iatrogenic tear deficiency, canine distemper, metabolic diseases, trauma, chronic blepharo-conjunctivitis and immune mediated lacrimal inflammation.

Williams (2008) divided KCS into two types: one in which tear production was deficient and the other with ocular surface tear deficiency due to tear evaporation, commonly seen in brachycephalic dogs with lagophthalmos.

Kim *et al.* (2009) reported KCS as the predominant cause of ulcerative keratitis in dogs.

2.2.4.1 Clinical signs

Bedford (1982) opined that a thick and tenacious ocular discharge along with a 'dry eye' indicated KCS.

Sansom and Barnett (1985) described the clinical signs in dogs presented with KCS. It included profuse, tacky, opaque, mucopurulent and unpleasant discharge in the conjunctival fornices, conjunctival hyperaemia, blepharospasm and a dull and lusterless cornea with ulceration and irregularities. In chronic cases, secondary vascular changes and pigmentary keratitis might occur.

Gionfriddo (1995b) stated that chronic KCS might be presented as a mild conjunctivitis with minimal corneal changes or as severe conjunctivitis and dense pigmentary keratitis with ulcerations or corneal scarring.

Kaswan *et al.* (1995) stated that in KCS affected patients, ropy mucus produced by conjunctiva was not dispersed by the aqueous tears and got accumulated in and around the eye. The hallmark of KCS was the presence of mucoid or mucopurulent ocular discharge.

Davidson and Kuonen (2004) stated that KCS in dogs were characterized by a mucoid to mucopurulent conjunctivitis, keratitis and corneal ulcers.

Williams (2008) stated that the ocular pathologic findings associated with ocular surface tear deficiency due to tear evaporation included corneal vascularisation, pigmentation and in severe cases, frank ulceration.

2.2.5 Prolapse of membrane nictitans gland ('Cherry eye')

Cherry eye resulted from hereditary weakness in the connective tissue surrounding the gland (Schoofs, 1999).

Gionfriddo (1995b) reported that dogs in which nictitating membrane gland was removed had an increased incidence of KCS.

2.3 OPHTHALMIC EXAMINATION AND DIAGNOSTIC MODALITIES

2.3.1 Ophthalmic examination

Bedford (1982) opined that oblique illumination was effective in determining corneal epithelial loss and posterior synechiae. He described the superficial appearance of diseased eye. A 'wet eye', a 'red eye' or a 'blue/steamy' eye indicated keratitis, uveitis or glaucoma and a 'dry eye' indicated KCS.

Wilkie and Whittaker (1997) opined that the culture and sensitivity, Schirmer's tear test (STT), cytology, fluorescein stain test (FST), and complete anterior segment examination should be considered as part of the routine examination in dogs with corneal ulcer.

Ultrasound biomicroscopy provided cross-sectional information of iridocorneal angle to diagnose the early stages of glaucoma. It also provided information on the pathogenesis, prognosis and preferred management of glaucoma (Gibson *et al.*, 1998).

Sansom (2000) opined that good illumination and magnification enabled detection of early changes in the clarity of aqueous humor or depth of the anterior chamber, indicative of ocular diseases. The aqueous humor being an ultra filtrate of plasma, changes in its composition indicated the presence of systemic diseases.

Bowersox and Croix (2001) described the use of noninvasive techniques for the examination of posterior segment of eye like direct and indirect ophthalmoscopy, ultrasonography and electroretinography.

Felchle and Urbanz (2001) suggested thorough ophthalmic examination of anterior and posterior segment for evaluation of ocular diseases along with STT, FST and tonometry. As ocular involvement might indicate systemic diseases, a general physical examination should precede the ophthalmic examination.

Moore (2001) opined that a good light source such as a pen light was helpful in determining the location, colour, shape and pattern of corneal lesions, which provided valuable information about the underlying cause.

Mandell and Holt (2005) pointed out that every patient presented with ocular diseases should undergo a complete ocular examination, including evaluation of the periocular structures, conjunctiva, cornea, anterior chamber, lens, vitreous and fundus.

Williams (2008) pointed out the importance of careful ophthalmic examination including STT in older dogs, especially of brachycephalic breeds like Chinese pug, Pekingese, Shih-tzu *etc*, dogs with previous endocrinopathies and any dog with corneal ulceration, corneal inflammation or red eye.

Mitchell (2011) described a step-by-step approach to ocular examination. He suggested that sedation should be avoided during ophthalmic examination unless it was absolutely necessary, as it could cause enophthalmos, downward rotation of globe, protrusion of the third eyelid, changes in pupil size and alteration in the results of quantitative tests.

2.3.2 Ocular Reflexes

According to Magrane (1977), the pupillary light reflex was helpful in determining optic nerve and retinal function. The direct and indirect reflexes were recorded as normal, sluggish, incomplete, or absent.

Bedford (1982) opined that a normal pupil should respond to bright focal illumination by contracting down if the retina, the visual pathway to the level of pupillary control centre and oculomotor nerve were functioning normally. Also, the blindness in the presence of reflexes indicated a lesion of the visual cortex.

Bedford (1987) pointed out that mydriasis indicated severe optic nerve damage or disruption of the parasympathetic supply to the iris sphincter musculature or both.

According to Felchle and Urbanz (2001), the pupillary light reflex was helpful in evaluating the function of retina, optic nerve, midbrain, oculomotor nerve and the iris sphincter muscle.

Menace response, a threatening, sudden movement presented near the eye, elicited a blink. Before testing for menace test, be sure that the facial nerve was intact by eliciting a blink reflex through the palpebral or corneal reflex. On menace testing, care should be taken to avoid air currents that might stimulate the blink reflex (Martin, 2001).

Moore (2001) opined that a normal direct pupillary light response indicated intact optic and oculomotor nerve. A normal consensual pupillary light response indicated a functional retinal and intact optic nerve in the fellow eye, provided oculomotor nerve and iris must be functional in the eye being evaluated.

Bath and Dua (2006a) graded the corneal, palpebral and pupillary reflexes as strong (++), weak (+) and absent (0) respectively in a study conducted in nine clinical cases of glaucoma in dogs.

Ollivier *et al.* (2008) stated that menace test evaluated the function of optic nerve, the facial and abducent nerve. This test was performed by making a menacing gesture with hand towards eye, without touching the vibrissae or causing excessive air currents to avoid false positive result. If the animal was able to see, it should have blinked or moved its head away.

Mitchell (2011) suggested that it was important to establish a normal blink response before judging the results of the menace response and dazzle reflex. A normal result confirmed an intact sensory pathway (trigeminal nerve) and motor pathway (facial nerve). A lack of blink indicated poor sensation or facial nerve paralysis.

2.3.3 Schirmer's Tear Test (STT)

Bedford (1982) opined that fifteen millimetres of a standard filter paper strip should become wet over a period of one minute, when placed in lower conjunctival sac of a normal dog.

Kaswan *et al.* (1995) suggested that STT should be performed in all breeds predisposed to KCS like Chinese pug, Lhasa apso, Cocker spaniel, Pekingese, English bull dog and West Highland white terrier as part of their yearly physical examination. The stated normal value was 15-25 mm/min. Values between 0-9 mm/min was considered to have KCS and those between 10-14 mm/min was considered as border line tear deficiency.

Hamor *et al.* (2000) described the advantage of STT II i.e. STT testing with the use of topical anaesthetic as it inhibits the unilateral reflex tear production due to stimulation of the trigeminal nerve. Also, the test values should be correlated with the patient's history, results of slit lamp examination, tear break-up time and results of serial STT measurements.

Felchle and Urbanz (2001) pointed out that painful conditions like corneal ulceration cause rise in the STT value because of reflex lacrimation, possibly masking an underlying KCS.

Miller (2001) stated that STT was indicated in corneal ulcer because a normal tear film was required to maintain an intact corneal epithelium.

Munro (2001) reported that STT should be performed in unsedated patients early on ophthalmic examination. Sedation, general anaesthesia, topical anaesthesia and para-sympatholytic agents would significantly reduce the reading.

Moore (2003) described STT as the most common test for pre corneal tear film and it assessed the quantitative production of the aqueous portion of the tear film. He opined that dogs with STT value less than 15 mm/min had decreased aqueous component of tear film.

Mitchel (2011) pointed out that STT was indicated in every case presented with ocular discharge, conjunctivitis and keratitis. He pointed out that the test should be carried out before administration of any local anaesthetic.

2.3.4 Fluorescein Staining Test (FST)

Miller and Crenshaw (1988) advocated the use of fluorescein dye strips for determining corneal integrity and patency of nasolacrimal duct.

According to Wilkie and Whittaker (1997), the hydrophilic nature of corneal stroma was responsible for the retention of the water soluble sodium fluorescein dye.

Felchle and Urbanz (2001) pointed out that sterile fluorescein strips were best suited over fluorescein solutions, as these solutions readily enabled bacterial growth because of neutralization of common preservatives added.

Miller (2001) described FST as the primary test for diagnosing corneal ulcers, and the most commonly used form of fluorescein stain was the impregnated paper strip. The moistened strip was placed on the superior bulbar conjunctiva and allowed the animal to blink several times to distribute the stain across the cornea and the excess stain was flushed out. Fluorescein stained intracellular spaces in the corneal stroma gained access through and outlining breaks in the epithelium.

Moore (2001) opined that FST should be done before application of the topical anaesthetic, as it could produce changes in corneal epithelial surface due to their epithelial toxicity.

Mandell and Holt (2005) opined that red and painful eye should be stained routinely with fluorescein dye to check for the presence of corneal ulceration.

Mitchell (2011) observed that exposed Descemet's membrane did not retain water soluble fluorescein dye, leaving a clear area at the base of the deep

defect in the cornea, indicating a descemetocoele. Also, the highly lipophilic and hydrophilic fluorescein dye would not penetrate an intact corneal epithelium with its lipid cell membranes.

2.3.5 Culture and sensitivity test

Bedford (1982) opined ocular discharge should always be submitted for microbiological examination and related antibiotic sensitivities.

Startup (1984) reported that the melting ulcers caused by *Pseudomonas* spp. were most frequently observed in brachycephalic breeds.

Gerding *et al.* (1988) opined that the selection of antibiotic therapy before obtaining culture and sensitivity results could be made on the basis of clinical signs, Gram's staining and a history of previous antimicrobial therapy. Knowledge of most commonly isolated microorganisms from the canine eye in a geographic location and their antibiotic susceptibility provided the most efficacious antimicrobial treatment.

Wolfer and Grahn (1994) isolated *Pseudomonas aeruginosa* from melting corneal ulcer in a brachycephalic dog. The isolate was found to be sensitive to gentamicin.

According to Sweeny and Irby (1996), the infectious ulcerative keratitis was due to opportunistic bacterial infection by the resident conjunctival flora after trauma and ulceration.

Wilkie and Whittaker (1997) opined that culture and sensitivity should be considered for all corneal ulcers and perforations.

Massa *et al.* (1999) collected corneal specimens from the centre and periphery of the epithelial defect using sterile swabs and found out that *Streptococcus* spp. and *Staphylococcus* spp. as the most prevalent bacterial organisms, followed by *Corynebacterium* spp., *Pasteurella* spp. and *Pseudomonas*

spp. The most common fungal isolates included *Aspergillus* spp. and *Candidan* spp. Topical use of antimicrobials interfered with the ability of the test to obtain definite diagnosis.

Whitley (2000) isolated *Staphylococcal* spp. (39%), *streptococcus* spp. (25%), *Pseudomonas* spp. (9.4%), *E. coli* (4.7%), *Corynebacterium* spp. (3.9%) and *Bacillus aureus* (2.4%) from the eyes of dogs with external ocular diseases. Chloramphenicol, gentamicin, amikacin and ciprofloxacin were found effective in most of the infections.

Morreale (2003) opined that culture should be performed before instilling any medications including topical anaesthetics onto the eye as they might inhibit bacterial growth.

Prado *et al.* (2005) isolated bacterial organisms from a total of 18 dogs with corneal ulcers, but only from 39% of dogs with healthy eyes. The dogs with corneal ulcers had higher number of colony forming units compared to the dogs with healthy eyes. Gram positive bacteria (86.5%) predominated over Gram negative bacteria (13.5%) in all dogs. Among the dogs with corneal ulcers *Staphylococcus* spp. was the predominant bacteria (56.8%) followed by Gram positive bacilli (18.9%), *Enterobacteriaceae* (13.5%) and *Streptococcus* spp (10.8%). They opined that the normal microflora became potentially pathogenic during immunosuppression or corneal tissue damage.

2.3.6 Microflora in cornea

Sansom (1988) pointed out that the normal bacterial flora of canine eye was influenced by geographical location, climate, seasonal factors, breed and technique used. All the microorganisms isolated from conjunctival sac were potentially pathogenic and the pathogenicity was altered by trauma and lack of tears or excessive use of corticosteroids.

The most common bacterial colonies isolated by Salisbury *et al.* (1995) from cornea in dogs with KCS were *Staphylococcus* spp. (69%), *Streptococcus*

spp. (56%), *E. coli* (15%), *Proteus* spp. (8%), *Klebsiella* spp. (7%) and *Pseudomonas* spp. (5%). He also found a significant decrease in corneal bacterial isolates in dogs that responded to topical cyclosporine treatment.

Ollivier (2003) described that Gram-positive organisms were most common isolate in the normal flora of the canine and feline eye. *Staphylococcus* spp., *Streptococcus* spp., *Corynebacterium* spp., *Bacillus* spp., *Pseudomonas aeruginosa*, *Escherichia coli*, *Klebsiella*, *Neisseria* spp., and *Fusobacterium* spp. were the only anaerobic bacteria that occasionally found in normal canine eye.

Mandell and Holt (2005) reported that Gram positive and Gram-negative organisms were capable of infecting the cornea. Gram-negative rods produced proteases (collagenases), which could result in rapid progressive destruction (“melting”) of corneal tissue.

Wang *et al.* (2008) found *Staphylococcus intermedius* as the most common isolate in the conjunctival sac of clinically normal dogs and dogs with ulcerative keratitis in China, so *Staphylococcus intermedius* belonged to resident flora in the conjunctival sac of normal dogs. If corneal ulcer developed, regardless of causes, *Staphylococcus* spp., *Streptococcus* spp. and *Pseudomonas* spp. were likely to invade the ocular surface of dogs.

2.3.7 Exfoliative cytology

According to Krawitz (1963), low number of leukocytes usually found in the basal cells increased during keratitis.

Lavach *et al.* (1977) obtained conjunctival scraping from both normal and infected eyes. Keratinised epithelial cells and leucocytes were uncommon in normal eyes. In inflamed conjunctiva the initial inflammatory cell response was seen specific according to the case. Leucocytes were the predominant cell type in all cases of acute and chronic bacterial conjunctivitis.

Massa *et al.* (1999) compared the usefulness of cytological evaluation of

aerobic microbial culture of corneal specimens in cases of ulcerative keratitis from different species of animals. Cytological evaluation was found superior to microbial culture for demonstrating fungi.

Felchle and Urbanz (2001) pointed out that culture and sensitivity testing and conjunctival or corneal cytology were useful in establishing a diagnosis and determining the appropriate antimicrobial therapy in corneal and conjunctival diseases. Instruments like cotton or Dacron swabs, cytobrush and malleable platinum spatula or the blunt end of a scalpel blade were found useful.

Hamor (2001) pointed cytologic examination as a valuable tool to establish the cause of ocular disease and to direct immediate therapy. It also revealed the cellular response associated with specific disease conditions.

Morreale (2003) pointed that the immediate information provided by cytologic evaluation was useful for designing an initial treatment regimen.

2.3.8 Tonometry

Magrane (1977) found out that tonometry was useful in the early detection of glaucoma. The normal IOP in dogs varied between 15 to 30 mm of Hg.

According to Bedford (1982), measurement of IOP was of value in two clinical situations: uveitis and glaucoma.

Gionfriddo (1995c) pointed out that tonometry was contraindicated in cases of descemetocoele or deep melting ulcer.

Gelatt and Mackay (1998) reported that no differences in IOP existed among dog breeds. The mean IOP for the normal dog is 19.0 mmHg with a range of 11 and 29 mmHg.

Moore (2001) reported that IOP less than 10 mm of Hg and a difference of 5 mm of Hg or more between the IOP of the two eyes were suggestive of anterior uveitis.

The diagnosis of glaucoma in dogs could be confirmed by measuring the IOP (Woerdt, 2001).

Broadwater *et al.* (2008) performed measurement of IOP in routine ophthalmic examinations because its values were important indicators of ocular health and diseased status. He opined that body position affects measurement of IOP.

Mitchell (2011) pointed out that gentle patient handling was essential to avoid induction of temporary rise in IOP as a result of jugular compression, which could lead to a false diagnosis of glaucoma.

2.3.9 Haematology

Sansom (2000) suggested that blood sampling for routine haematology, biochemistry and serology should be carried out in combination with thorough clinical examination when an underlying systemic disease associated with uveitis was suspected.

According to Schalm *et al.* (2000), normal values of various haematological parameters such as differential leukocyte count were, neutrophils 60-77 per cent (av 70%), band cells 0-3 per cent (av 0.8%), lymphocytes 12-30 per cent (av 20%), monocytes 3-10 per cent (av 5.2%), eosinophils 2-10 per cent (av 4.0%) and basophils rare. Haemoglobin concentration will be between 12 and 18 g/dl (av 14.9g/dl) and packed cell volume 37 -55 per cent (av 45.54%).

A recheck on complete blood count should be made for patients in whom RBC, WBC or platelet abnormalities were previously diagnosed (Metzger and Rebar, 2004).

In a study of glaucoma in dogs, Bath and Dua (2006a) observed that the mean haematological values were within normal physiological range.

The hematology in animals with keratitis did not vary much from the normal

range to demonstrate any underlying disease process (Resmi, 2008).

In a study of glaucoma in dogs, Priya (2009) observed that the mean haemotological values were within normal physiological range.

2.3.10 Blood glucose level

Kaswan *et al.* (1995) opined that Keratoconjunctivitis sicca may be associated with autoimmune disorders like diabetes mellitus.

Whitley *et al.* (1991) stated that Keratoconjunctivitis sicca might occur with endocrinopathies like diabetes mellitus.

Cullen *et al.* (2005) reported that diabetic dogs have significantly altered keratoconjunctival characteristics compared to non-diabetic dogs.

Williams *et al.* (2007) observed decreased corneal sensitivity in diabetic animals.

Williams (2008) stated that exposure keratopathy in brachycephalic breeds with lagophthalmos and decreased tear production might be associated with endocrinopathies like diabetes mellitus, hyperadrenocorticism and hypothyroidism.

2.4 TREATMENT

Sansom (1988) opined that in severe ocular infections, both systemic and topical treatments were essential. Once an intraocular infection was established, it would be difficult to treat and the outcome was often disastrous due to blood-ocular barrier and the absence of lymphatics.

Whitley *et al.* (1991) stated that the therapy for KCS should be designed to eliminate the cause, stimulate tear production, replace tear film, control and prevent bacterial infection and decrease inflammation.

Gionfriddo (1995b) stated that the treatment goal for all preocular tear film deficiency diseases was providing moisture and lubrication to the cornea and conjunctiva and preventing bacterial invasion of these structures.

Treatments of corneal ulcers depend upon their depth and rate of progression. The general therapeutic principles were applied to treat the ulcer, superficial or deep, but rapidly progressing deep stromal ulcers warrant surgical intervention in order to give support and reduce the risk of perforation (Renwick, 1996).

Miller (2001) opined that the treatment for exposure keratitis consisted of treating the underlying cause, medically and surgically by a temporary tarsorrhaphy or permanent canthoplasty

Miller (2003) stated that the primary goal in treatment for all forms of glaucoma was to reduce the IOP to a safer level that would halt the death of retinal ganglion cells, so that progressive visual impairment could no longer occur.

The most important step in the treatment of corneal ulceration was to determine and eliminate the cause, followed by attempts to create an ideal environment for lesion repair, prevention of progression, and surgical treatment to prevent corneal rupture (Kim *et al.*, 2009).

2.4.1 Medical management

Martin (1971) studied corneal epithelial healing in dogs treated with topical antibiotic, mineral oil-antibiotic, vitamin A-antibiotic, subconjunctival repositol corticosteroid and vitamin A-corticosteroids and found that the corneas treated with topical vitamin A and mineral oil-antibiotic combination healed faster than antibiotic treated group.

Treatment with vitamin A, orally or local application was beneficial especially in corneal erosion syndrome and in cases of indolent ulcers, vitamin C and E proved effective (Startup, 1984).

According to Bedford (1987), topical and systemic broad spectrum antibiotics together with cycloplegics and mydriatics therapy were essential for treatment of corneal perforation. Also, the use of corticosteroid therapy to treat the subsequent uveitis should be investigated.

Sansom (1988) opined that ointments delayed the corneal healing and were contraindicated in perforating corneal wounds. Their use in KCS was inadvisable as they disrupted the tear film and trapped exudates

Wilkie and Latimer (1991) noticed that topical administration of 0.5% timolol maleate at 12 hour interval resulted in reduction of IOP in treated and contra lateral eyes. Also the 'cross over effect' and 'systemic uptake' of timolol maleate resulted in bilateral miosis because of inhibitory effects of drug on β -adrenergic fibers on canine iris sphincter muscle.

According to Collins *et al.* (1995) high intervention of cornea caused substantial postoperative pain and hence analgesics were essential to improve animal's comfort and to minimize the tendency to self trauma.

Gionfriddo (1995d) opined that topical non-steroidal anti-inflammatory

drugs (NSAIDs) might be safely administered in eyes with concurrent uveitis and ulcerative keratitis, whereas topical and systemic corticosteroids should not be used for uveitis with concurrent keratitis.

Early diagnosis of KCS and treatment with ophthalmic cyclosporine may avert a major cause of blindness in dogs. The resolution of corneal vascularization and pigmentation took place gradually over a period of three to 12 months or more (Kaswan *et al.*, 1995).

According to Wilkie and Whittaker (1997) the medical treatment for corneal ulceration included topical application of artificial tears, broad spectrum antibiotics, mydriatics-cycloplegic agents and anti-inflammatory or immune modulating drugs. The topical medications should be administered before ointments at a frequency ranging from 2 to 8 hours, depending on severity of infection, spaced 5 to 15 minutes apart. Corneal pigmentation could be better managed through prevention and treatment of the inciting causes like KCS, lagophthalmos and lid abnormalities rather than keratectomy.

Whitley (2000) reported the effectiveness of topical cyclosporine in the treatment of KCS owing to the increase in the tear production characterized by the decrease in number of *pseudomonas* spp. and coliform isolates from KCS affected eyes.

Gelatt and Mackay (2001) found that 2% dorzolamide instilled twice or thrice daily caused significant decrease in IOP.

Hasegawa *et al.* (2001) reported that lower IOP could be maintained with medical therapy alone for a long period in dogs with open angle glaucoma.

Hendrix *et al.* (2001) in a study on the effect of antibiotics on the morphologic characteristics and migration of canine corneal epithelial cells in tissue culture, pointed out that gentamicin inhibited cell migration and exhibited more cytopathic effect than tobramycin.

Munro (2001) described that the antibiotics were indicated in the treatment of corneal ulceration, as the loss of the epithelial barrier exposed the corneal stroma. The topical application provided therapeutic concentration of the drug and markedly increased penetration in corneal epithelial cells. Flurbiprofen had good corneal penetrability and was used to control uveitis after intraocular procedures.

Miller (2001) opined that superficial ulcers demonstrated by positive FST usually healed rapidly with antibiotic and cycloplegic therapy. Such patients should be re-evaluated within a week to assess healing. Topical instillation of 1% atropine ophthalmic solution two to three times daily paralyzed the ciliary body muscles and reduced ocular discomfort. He also pointed out that solutions were preferred over ointments especially in cases where there were chances of corneal perforation.

Woerdt (2001) pointed out that topical carbonic anhydrase inhibitors reduced IOP without significant systemic side effects like metabolic acidosis, fatigue, hypokalemia, loss of appetite, gastrointestinal disturbances and renal calculi that were observed with orally administered drugs.

Gelatt and Mackay (2002) observed that the selective α_2 -adrenergic agonist, brimonidine tartrate (0.2%) decreased the IOP with significant miosis and limited ocular hypotension. They opined that because of the limited drug induced ocular hypotension; brimonidine should be combined with other drugs. Also, it rendered neuroprotection of the optic nerve head and the retinal ganglion cells.

According to Willis *et al.* (2002), carbonic anhydrase enzyme inhibitors reduced the synthesis of aqueous humor by decreasing the production of bicarbonate ions from water and carbon dioxide in epithelium of ciliary body. By reducing the production of bicarbonate ions, less sodium and water moved into posterior chamber, thus lowering the IOP.

Stiles *et al.* (2003) opined that repeated topical application of anaesthetic was contraindicated in corneal ulceration due to its toxic effect on the corneal

epithelium and delayed re-epithelialisation of corneal wound in spite of the mild analgesic effect.

Ollivier (2003) opined that even though ulcers were not usually infected, topical broad spectrum antibiotics should be used to prevent secondary, opportunistic bacterial infection as the epithelial integrity was compromised. Recurrent superficial ulcers required additional medical treatments like serum, growth factors and vitamins or surgical treatment.

Giuliano (2004) observed that topical NSAIDs represented a more selective modality to treat ocular inflammation and a more efficient therapeutic choice for treating ulcerokeratouveitis or stromal abscesses and controlled postoperative pain and inflammation after intraocular procedures. Systemic NSAIDs were useful in treating uveitic conditions for which corticosteroids were contraindicated such as infectious diseases and diabetes mellitus.

Maehara *et al.* (2004) observed that topical application of timolol maleate decreased pulse rate and blood pressure significantly until the end of study period in glaucomatous patients. It reduced the IOP by decreasing production and also increasing the uveoscleral outflow.

Malmberg and Lupo (2004) observed that ophthalmic solutions affected vision and corneal healing minimally and were easy to administer.

Mandell and Holt (2005) suggested topical therapy with corticosteroids in the treatment of anterior uveitis. Topical mydriatic drugs like atropine could be used to control ciliary body spasm and to prevent synechiae formation, but was contraindicated in elevated IOP, where a drug with rapid action and short duration like tropicamide could be used.

The medical treatment regimen for keratitis in dogs included the use of topical medications like gentamicin, atropine sulphate and vitamin A. Corticosteroid therapy was instituted only in cases of corneal opacity or vascularization after developing complete healthy corneal epithelium. Use of

corticosteroid helped in clearing of cornea and thus improved vision (Bath and Dua, 2006b).

According to Plummer *et al.* (2006), a fixed combination of 0.5% timolol maleate and 2% dorzolamide twice daily was efficacious at reducing IOP in glaucomatous dogs, compared to either timolol or dorzolamide alone.

Tolar *et al.* (2006) observed that the resistance to fluoroquinolone antimicrobials was minimal among all isolates from dogs with bacterial keratitis and pointed out that ciprofloxacin was still an effective treatment for bacterial keratitis in dogs especially against *Staphylococcus* spp., *Streptococcus* spp. and *Pseudomonas* spp.

Ledbetter *et al.* (2007) studied the susceptibility of *Pseudomonas aeruginosa* isolates from 27 dogs with ulcerative keratitis to fluoroquinolones in vivo. The percentage of susceptible isolates was highest for ciprofloxacin and levofloxacin (100%) and lowest for moxifloxacin (88.9%).

Ollivier *et al.* (2007) recommended protease inhibitors for the treatment of ulcerative keratitis to reduce the progression of stromal ulcers, speed epithelial healing and minimize corneal scarring and it included autologous serum, which contains broad protease inhibitors like α_2 macroglobulin and α_1 proteinase inhibitor. The other protease inhibitors recommended were N-acetyl cysteine, doxycycline, Itonastat and ethylene diamine tetra acetic acid (EDTA), which chelates the zinc and calcium, the cofactors and stabilizing ions required for the metalloproteinases.

According to Townsend (2007), the benefit of lacrimostimulant, cyclosporine in patients with KCS stems from its selective T-helper lymphocyte suppression. He also advised to use lacrimomimetics like polyvinyl alcohol, hydroxyl propyl methyl cellulose, dextran and sodium hyaluronate or chondroitin sulphate based preparations with aqueous base more similar to natural tears to improve lubrication and provide comfort until sufficient tear production was

attained in cases of KCS.

Hendrix and Cox (2008) studied the pharmacokinetics of topical ciprofloxacin in tears and concluded that mean tear concentration of ciprofloxacin remained above the MIC₉₀ levels for most pathogenic bacteria for 6 hours in normal mesocephalic and brachycephalic dogs.

Palmer *et al.* (2008) pointed out that topical administration of non selective β -adrenergic receptor antagonist, 0.5% timolol maleate produced side effects like bradycardia and broncho-constriction due to systemic uptake.

The medical treatment for *Pseudomonas aeruginosa* associated keratitis in dogs included protease inhibitors, antimicrobials, tropicamide and oral administration of NSAIDs (Wang *et al.*, 2008)

Williams (2008) pointed out that cyclosporine being a specific immunomodulator with immunosuppressive effects, had a lacrimogenic effect and also a hypothesized lacrimomimetic effect.

Ivermectin at the rate of 50 μ g/kg body weight administered orally completely cleared the larval stages of *Dirofilaria* sp. (Bowman and Atkins, 2009)

Gilmour and Lehenbauer (2009) stated that topically, orally and parenterally administered NSAIDs have the ability to suppress prostaglandin-mediated anterior uveitis. Orally administered NSAIDs were effective in controlling pain and postoperative anterior uveitis.

Kim *et al.* (2009) used 2 % cyclosporine to stimulate tear production for the treatment of corneal ulceration associated with KCS in addition to topical mydriatics, antibiotics and anti-inflammatory agents. Elizabethan collars were used to prevent self trauma to the eye until the ulcers were healed.

Ledbetter *et al.* (2009) successfully treated *Pseudomonas aeruginosa* ulcerative keratitis in dogs with topical ciprofloxacin 0.3% ophthalmic solution

with or without additional surgical procedures.

Townsend *et al.* (2009) reported that topical fluoroquinolones were effective in treating infected corneal ulcers or stromal abscesses especially against gram negative organisms.

Hartley (2010) indicated supportive care of the cornea with false tear preparation and/or contact lens, with antibacterial therapy for exposure keratopathy.

Smith *et al.* (2010) suggested that the topical application of a single ocular hypotensive agent might not be able to achieve the desired degree of IOP reduction and hence a combination of two or more anti-glaucomal drugs might be required. He also mentioned the combination drugs like dorzolamide-timolol and brimonidine-timolol.

Pirie *et al.* (2011) mentioned that topical NSAID, flurbiprofen (0.3%) was effective as it had good intraocular permeability, minimal significant intraocular metabolism and potency of inhibition of cyclooxygenase.

2.4.2 Surgical management

Surgical treatment for weakened corneas included tarsorrhaphy and membrana nictitans or bulbar conjunctival flap techniques. Membrana nictitans flap was more convenient than the tarsorrhaphy procedure because of the short time required for the procedure. The modalities such as eye patches, complete or temporary tarsorrhaphies, membrana nictitans or bulbar conjunctival flaps were described and opined that they should be inexpensive, easily applied and well tolerated and should provide direct support to the weakened cornea (Anderson *et al.*, 1976).

According to Bedford (1980), membrana flapping in which the membrana nictitans was sutured to the dorsal bulbar conjunctiva was a simple and effective way of covering and protecting the whole corneal surface. Fornix based

conjunctival hood flaps and the pedicle conjunctival flap or grafts were used to cover specific areas of the cornea.

Helper (1981) described the procedure for temporary tarsorrhaphy. One or two interrupted horizontal mattress sutures were used for temporary tarsorrhaphy. A small opening was left at the medial canthus to facilitate medication. A prolapsed third eyelid gland seldom remains in normal position and hence removal of third eyelid gland was indicated.

Startup (1984) described various techniques for the treatment of corneal ulcers, which included conjunctival flaps, third eyelid flaps, hydrophilic contact lenses, and superficial keratectomy. Nictitating membrane flap was observed as the most useful single treatment for corneal ulcers where there was a deep defect or a large area was involved. The author recommended suturing of third eyelid flap to the bulbar conjunctiva in breeds with protruding eyes or membrane flap of limited size, because there was a great tendency for sutures to pull out if the flap was sutured to the eyelid.

Kaswan and Martin (1985) suggested tacking of third eyelid gland for its repositioning that involved the suturing of the prolapsed gland to the periosteum of the ventral orbital rim.

Roberts *et al.* (1986) studied the disinfecting property of povidone-iodine concentrations of 1:2, 1:10, 1:50, 1:100 in eye. It was concluded that 1:50 povidone-iodine solution was effective in eliminating bacterial contamination of the external ocular tissue without causing tissue reaction.

In iris prolapse, the protruding iris material should be replaced wherever possible or removed when adhesions prevented its replacement. The anterior chamber should be washed clean of blood and fibrin and reformed using sterile balanced salt solution. The wound should be repaired using interrupted 8-0 absorbable or nylon sutures placed 1 millimeter apart. The use of membrane flap was helpful (Bedford, 1987).

In case of KCS with exposure keratopathy secondary to lagophthalmos, euryblepharon or facial paralysis, a permanent lateral canthoplasty was found to be effective than parotid transposition. Also, the permanent lateral canthoplasty shortening reduced the exposed surface area of the globe (Whitley *et al.*, 1991).

Geasey *et al.*, (1992) reported that collagen shields proved to be highly oxygen permeable, promoted epithelial healing, decreased inflammatory cell infiltration and reduced corneal oedema.

Morgan *et al.* (1993) described the various imbrication techniques for the replacement of prolapsed third eyelid gland in dogs.

Stanley and Kaswan (1994) introduced the modification in the orbital rim anchorage method for surgical replacement of the prolapsed third eyelid gland in dogs.

Collins *et al.*, (1995) described the effect of anaesthetic drugs in ocular patients. The potential benefit of anticholinergic treatment was prevention or reversal of oculocardiac reflex. In ketamine administration, the palpebrae remained open, globe was centrally positioned and the palpebral and corneal reflex persisted. Diazepam reduced the ketamine induced increase in intraocular pressure. Topically administered anaesthetic may be irritating and could cause transient conjunctival hyperaemia.

In dogs with KCS, lagophthalmos and exophthalmos and in brachycephalic dogs like Chinese pugs, with dense pigmentary keratitis or subsequent pigment accumulation even after cyclosporine therapy, due to excessive corneal exposure, Kaswan *et al.* (1995) advised lateral lid shortening or parotid duct transposition in addition to ophthalmic corticosteroids.

Wilkie and Whittaker (1997) suggested that topical broad spectrum antibiotics should be administered 6 to 12 hours prior to surgery especially in infectious keratitis and corneal perforation. The irrigation of cornea with sterile balanced salt solution throughout the surgical process prevented drying of the

cornea, preserved corneal epithelium, improved tissue handling and visualization. The main complications associated with corneal perforation included phthisis bulbi and infection. However, maintenance of corneal curvature or shape was deemed to be less important.

Stanley *et al.* (1998) reported the complications of superficial keratectomy which included perforation, permanent scar formation, infection, corneal vascularisation and fibrosis similar to that reported by Wilkie and Whittaker (1997) in brachycephalic breeds. They also opined that the third eyelid flap helped in minimizing postoperative discomfort and infection.

Hansen and Guandalini (1999) stated that the principle advantage of the techniques lies in the ready availability of material to fill and strengthen the stromal defect. The techniques available to treat the corneal lesions included tissue adhesives and biological grafts.

Featherstone and Sansom (2000) indicated the application of biodegradable collagen based material small intestinal submucosa (SIS) in the management of ulcerative keratitis, in which preservation of vision was desired as it promoted tissue healing with negligible scar formation.

Sansom (2000) reported that the surgical repair of the wound and replacement of the iris produced satisfactory result in iris prolapse. The aqueous clot formed over the wound should not be removed prior to general anaesthesia and repair. Penetration of anterior chamber caused aqueous humor leakage and acute pain.

Whitley (2000) indicated debridement of the loose epithelium with a cotton tipped swab in the treatment of refractory corneal ulcers.

Deep corneal defects should be managed surgically for optimum results and techniques available included conjunctival autografts, cyanoacrylate tissue adhesive, corneal-scleral-conjunctival transpositions, lamellar keratoplasty, and penetrating keratoplasty. The tectonic corneal grafts and the non-corneal grafts

including split-thickness dermal grafts, equine pericardium, peritoneum, equine amniotic membrane, human amniotic membrane in rabbits, equine renal capsule, and expanded polytetrafluoroethylene could also be used (Featherstone *et al.*, 2001).

Miller (2001) opined that debridement was inexpensive and possessed little risk to the eye. Also, it could be easily accomplished without sedation.

According to Herring (2003), the corneal surgical procedures were directed towards preserving or restoring maintenance of physical integrity, clarity, shape and curvature of cornea. A temporary tarsorrhaphy leaving the medial canthus open allowed postoperative topical medication. It also provided short-term protection of the surgical site and improved postoperative epithelial healing. Elizabethan collar to prevent self-trauma and broad-spectrum antibiotics until complete healing were also advised.

Hollingsworth (2003) suggested enucleation in cases of severe intraocular damage such as globe collapse accomplished by extrusion of intraocular contents like lens, vitreous and retina. In case of a prolapsed uveal tissue through a corneal laceration, it should be replaced with the use of viscoelastic material or gentle manipulation, if the tissue was healthy. But if the tissue was severely contaminated, then it should be excised.

Moore (2003) recommended debridement for the treatment of SCCEDs as it stimulated corneal healing by removal of the abnormal corneal epithelium, promoted epithelial attachment to basement membrane and removal of debris from the exposed stroma. Different bandaging procedures, use of contact lenses, collagen shields, cyanoacrylate tissue adhesives, temporary tarsorrhaphy, third eyelid flap, and conjunctival flaps to protect cornea after various surgical procedures were also discussed. For exposure keratitis, the surgical treatment recommended was temporary tarsorrhaphy or permanent canthoplasty.

Ollivier (2003) mentioned debridement of loose epithelium and grid

keratectomy for the surgical management of recurrent superficial erosions. Surgical management of progressive deep ulcerations included conjunctival grafts or flaps and corneal grafts.

According to Bussieres *et al.* (2004), collagen based material had the advantage of being cost effective, commercially available and easy to handle. SIS acted as a scaffold for the repair and provided valuable tectonic support and helped in epithelialization of small intestinal submucosa because of its collagenous nature that mimicked the stromal surface. He also mentioned the complications with the use of SIS grafts in dogs which included aqueous leakage, conjunctival flap dehiscence, SIS laceration, chronic uveitis and hyphaema.

Morgan (2004a) described steps for preparation of eyelid surgeries. He suggested using diluted aqueous povidone-iodine solution (diluted to 1:10 to 1:50 with 0.9 % irrigating saline) for the preparation of eyelid and adnexa and sterile cotton swabs soaked in diluted povidone-iodine solution to remove residual material from conjunctiva and ocular surfaces. The povidone-iodine scrub and chlorhexidine diacetate solutions being irritants for eye should not be used for preparing the eyelids.

Morgan (2004b) pointed out the advantage of suturing the third eyelid to the bulbar conjunctiva especially in brachycephalic breeds in which the third eyelid was too short to suture to the upper eyelid. Since the globe and third eyelid moved together, less motion occurred between the third eyelid and the cornea. He described the techniques for correction of entropion. Modified Hotz-Celsus operation was found to be applicable for all types of entropion.

The first line of treatment in case of prolapsed third eyelid gland is the replacement of gland. The removal of gland could be performed in case of recurrence (Herrera, 2005).

Renwick (2007) suggested eye shortening in brachycephalic breeds with signs of corneal disease due to globe exposure. The smaller palpebral fissure thus

created resulted in improved blink function, better tear film distribution and decreased tendency to corneal desiccation. Eyelid shortening could be done at the medial or lateral canthus. The later enabled removal of the hairy caruncle and the frequently in turned section of medial lower eyelid. Also, it might be sufficient to perform a medial lower lid Hotz-Celsus procedure for medial lower lid entropion or might be necessary to remove the nasal folds, if it exacerbated the degree of entropion or if nasal fold trichiasis was present.

Vanore *et al.* (2007) described the standard procedure for surgical repair of deep melting corneal ulcers which included debridement of necrotic and collagenolytic tissues, fixation of SIS grafts on the cornea using 9/0 polyglactin 910 placed at 12, 3, 6 and 9'O clock position with simple interrupted sutures followed by nictitating membrane flap to protect corneal repair. The three important stages in SIS integration and corneal wound healing were corneal neovascularization, proliferation of epithelial and stromal tissue and remodeling of the extracellular matrix to produce corneal transparency and preservation of corneal integrity.

According to Gilger *et al.* (2008) conjunctival tissue might not have adequate strength to maintain a water tight seal and a formed anterior chamber after surgery in a full thickness corneal defect. They advised to use cornea or another material having more structural integrity than conjunctival tissue, to overcome these problems.

Krohne (2008) suggested nasal fold reduction by either complete or partial resection for nasal fold trichiasis or a lateral or medial blepharoplasty for large palpebral fissure. He opined that complete resolution of clinical signs associated with eyelid and adnexal abnormalities was uncommon, but improved the comfort of the patient.

Resmi (2008) noticed disruption of third eyelid flap even after suturing on to the bulbar conjunctiva.

In a retrospective study of ulcerative keratitis in 32 dogs, Kim et al. (2009) used debridement, tarsorrhaphy and conjunctival flap construction for the surgical treatments of corneal ulcerations. Enucleation was the treatment of choice in cases of ulceration with well defined suppurative endophthalmitis.

Collagen sheet of bovine intestinal origin was found to be very effective in the treatment of staphyloma, subsequent to corneal ulcers in dogs (Anoop *et al.*, 2010).

In a study on prolapse of membrane nictitans gland in dogs, recurrence was noticed in all the dogs in which repositioning of gland was tried, which were subsequently excised (Bharathi, 2010).

Chandler *et al.* (2010) suggested grid keratotomy as the most commonly preferred surgical procedures used to treat refractory corneal ulcers in dogs.

According to Chinchu (2010), the collagen sheet placed on the newly prepared recipient bed of cornea was well supported by temporary tarsorrhaphy sutures.

Hartley (2010) pointed out the paradoxical effect seen with dilution of povidone-iodine, with more dilute solutions exhibiting greater antibacterial action. He recommended dilutions of 1 in 10 for eyelids and 1 in 50 dilutions for ocular surfaces based on tissue tolerance and bacterial counts. He also pointed out that povidone-iodine solutions should not be used in the preparation of a ruptured globe as it was not tolerated by the corneal endothelium, where sterile normal saline was used

Materials and Methods

3. MATERIALS AND METHODS

The study was conducted in 22 dogs with eye affections belonging to the breed Chinese pug, presented to the surgery outpatient unit of University Veterinary Hospitals, Mannuthy and Kokkalai.

3.1 SELECTION OF CASES

All the Chinese pugs presented with the history of eye affections were subjected to detailed clinical and ophthalmological examination and were categorised based on the disease condition for which they were presented or diagnosed. The treatment was selected depending upon the type and severity of the condition, based on the observations made on the first day of presentation. The conditions which responded to the medical management were treated medically and were observed for a period of 60 days. Those conditions which were refractory to medical management or required immediate surgical intervention were subjected to surgical treatment. Among 84 cases presented and subjected to treatment, cases of 22 dogs, the post treatment observations of which were available for a period of 60 days, were selected for the study. The cases were designated as Case Nos. I, II, III, IV, V, VI, VII, VIII, IX, X, XI, XII, XIII, XIV, XV, XVI, XVII, XVIII, XIX, XX, XXI and XXII. Post treatment observations were made on the 10th, 25th, 40th and 60th day to study the effectiveness of treatment adopted.

3.2 MEDICAL MANAGEMENT

The medical treatment for ulcerative keratitis included topical instillation of antibiotics, anti-inflammatory agents like furbiprofen¹ and cycloplegic agents

1. Flur eye drops (0.3%), Nicholas Piramal, Dhar, Madhya Pradesh

like tropicamide². The topical antibiotic used initially was ciprofloxacin³ in all the cases and was changed, if required, based on the clinical outcome. Changes were made based on results of the culture and sensitivity test of the ocular discharges or corneal swab. Eye drops were instilled four times daily at an interval of three to four hours. In addition to the medications mentioned above, oral antibiotics (cephalexin⁴) at the rate of 22 mg/kg body weight for seven days was also advised in case of deep lesions and uveitis. In all cases, oral supplementation of multivitamins was also advised. In those cases where the eye was found to have an elevated intraocular pressure, antiglaucoma therapy was given with topical instillations of carbonic anhydrase inhibitors like dorzolamide⁵ and β blockers like timolol maleate⁶ or a combination of α_2 adrenergic agonist, brimonidine tartrate and β blockers like timolol maleate⁷ were advised. The treatment adopted for the management of tear deficiency associated with Keratoconjunctivitis sicca (KCS) included topical instillation of lacrimomimetics (tear substitute) like carboxymethyl cellulose sodium⁸ and lacrimostimulants like cyclosporine⁹ two to three times daily. In case of tear deficiency due to globe exposure, tear substitute alone was advised. Elizabethan collar (Plate 3) was advised in all the cases to avoid self mutilation, till complete healing.

2. Tropicamet eye drops (1%), Milmet Lab Pvt. Ltd., Baroda

3. Ciplox eye drops (0.3%), Cipla Ltd., Verna, Goa

4. Sporidex DS (250mg tablets), Ranbaxy laboratories Ltd., A.P.

5. Dorzox eye drops (2%), Cipla Ltd., Verna, Goa

6. Glucomol eye drops (0.5%), Allergan India Ltd., Bangalore

7. Brimotim eye drops, Ophtho Remedies (P) Ltd., Mumfordganj, Allahabad

8. CCS eye drops (0.5%), Ophtho Remedies (P) Ltd., Mumfordganj, Allahabad

9. Cyclomune (0.1%), Sun pharmaceuticals Ind. Ltd., Halol, Gujarat

3.3 SURGICAL TREATMENT

All the surgical treatments were performed under general anaesthesia with standard preoperative preparations.

3.3.1 Preoperative preparation of patient

Topical instillation of antibiotic, ciprofloxacin eye drops was advised four times daily and was started three days prior to surgery. Solid food was withheld for twelve hours and liquid food for four hours before the surgery in all the cases.

3.3.2 Preoperative preparation of surgical site

The affected eye was thoroughly irrigated with sterile normal saline and the periocular area was cleaned with sterile cotton to remove accumulated ocular discharge, dirt and tissue debris. The eyelashes were clipped close to the palpebral border. The periocular area was carefully clipped and thoroughly scrubbed with povidone iodine solution (1:50). In dog XVI, for the correction of nasal fold trichiasis, the hair around the entire nasal fold to be removed was shaved and in case of dog XVII, for the correction of lower eyelid entropion and to reduce the globe exposure, the hair around the lower eyelid and lateral canthus was shaved followed by thorough scrubbing with povidone iodine solution (1:50). The eyes were irrigated with sterile isotonic normal saline solution and the periocular area was painted with povidone iodine solution. The animals were positioned, draped and instilled a few drops of povidone iodine (5% w/v) ophthalmic solution¹⁰ (Slatter and Dietrich, 2003).

3.3.3 Preparation of collagen sheet

Collagen was used as a potential biomaterial for the repair of corneal lesions. The collagen sheet of bovine small intestinal origin was procured from M/s Animal Byproducts Ltd., Chennai. It was prepared from submucosal layer

10. Apidine-5 (5% w/v), Appasamy Ocular Devices Pvt. Ltd., Pondichery

of intestine. Submucosal layer of intestine was separated by repeated scraping and washing of both sides and dried it. Collagen sheet appears as semiluscent sheet when dry and preserved in air tight packets. When moistened the collagen sheet became more transparent and pliable (Plate 2).

3.3.4 Anaesthesia

All the dogs were premedicated with atropine sulphate¹¹ at the rate of 0.045 mg/kg body weight followed by xylazine hydrochloride¹² at the rate of 1.5 mg/kg body weight, both given intramuscularly. After 15 minutes, general anaesthesia was induced with ketamine hydrochloride¹³ at the rate of 5 mg/kg body weight, administered intramuscularly. Anaesthesia was maintained by intravenous infusion of a combination of xylazine hydrochloride and ketamine hydrochloride, equal quantity by volume and diazepam¹⁴ at the rate of 0.5 mg/kg body weight, given 'to effect' (Collins *et al.*, 1995).

3.3.5 Surgical technique

All the surgical procedures were conducted with the help of an operating microscope¹⁵ (Plate 2), with a magnification of 10 X.

The surgical technique was selected according to the nature of the lesion. One of the following surgical techniques was performed in the selected cases.

3.3.5.1 Corneal debridement

All the dogs were positioned in lateral recumbency with the affected eye above and were draped. Eyelids were kept retracted with the help of an eye speculum (Plate 2). Dry

11. Atropine sulphate injection (0.6 mg/ml), Mount Mettur pharmaceuticals Ltd., India

12. Xylazine injection (20 mg/ml), Indiam Immunologicals Ltd., Andra Pradesh

13. Ketmin-50 injection (50 mg/ml), Themis Medicare Ltd., Mumbai

14. Calmpose injection (5 mg/ml), Ranbaxy, NewDelhi

15. Ophthalmic operating microscope, AA OM 10, Appasamy Associates, Chennai

cotton tipped applicator was moved in a circular manner beginning from the margin of the lesion and removed the abnormal epithelium and debris from the stromal surface. The fine corneal scissors (Plate 2) were used to facilitate the removal of the loose epithelium. The defect was aggressively debrided until epithelium no longer dislodged easily from the stroma. The epithelium was removed about 1-2 mm away from the margin of the ulcer (Miller, 2001).

3.3.5.2 Iridectomy and suturing

The dogs were positioned in lateral recumbency with the affected eye above and were draped. Iridectomy was performed in cases with irreducible or necrosed staphyloma. The temporary seal formed by the aqueous clot over staphyloma was removed manually using corneal scissors (Plate 2). Attempts were made to separate the adhesion of the iris from the borders of perforated cornea. In cases, where the adhesion could not be separated, the prolapsed part of the iris was excised and removed using corneal scissors. The haemorrhage was controlled by instilling 1:10,000 adrenaline. The anterior chamber was gently irrigated with the sterile balanced salt solution¹⁶ and the clots were removed. The corneal wound was then sutured and the shape of the globe was reestablished using the sterile balanced salt solution. In case where the corneal wound could not be sutured, collagen sheet was applied (Hollingsworth, 2003).

3.3.5.3 Third eyelid flap

The dogs were positioned and the eye was prepared as above. For performing the third eyelid flap, three horizontal mattress sutures were preplaced in the third eyelid using 4/0 braided silk¹⁷. The sutures were passed through the anterior surface of the third eye lid about 2 to 3 mm from the free margin and then into the upper eyelid. The direction was then reversed and the sutures were then passed from inner surface of the nictitans membrane to the outer surface. The sutures were tightened so that the third eyelid was pulled dorsally to

16. Irgan, Klar Sehan Pvt. Ltd., Kolkata

17. Ethicon, Johnson & Johnson Ltd., Aurungabad

cover the lesion and knotted. Two or three similar sutures were used to attach the nictitating membrane to the upper eyelid (Morgan, 2004b).

3.3.5.4 Application of collagen sheet

The dogs were positioned in lateral recumbency with the affected eye above and were draped. The eyelids were kept retracted with the help of an eye speculum. Collagen sheet was cut into the desired size and shape with scissors. It was made large enough to cover the cornea and was thoroughly washed with sterile normal saline. Then, it was dipped in gentamicin¹⁸ eye drops for about 15 minutes and kept over the newly prepared corneal recipient bed. To ensure the retention of the collagen sheet in the eye, it was sutured onto the bulbar conjunctiva using 4/0 braided silk followed by temporary tarsorrhaphy using 1/0 braided silk sutures (Chinchu, 2010).

3.3.5.5 Temporary tarsorrhaphy

In all the surgical ulcerative keratitis cases, temporary tarsorrhaphy was done to protect the cornea. To create a temporary tarsorrhaphy, one or two horizontal mattress sutures were preplaced through the eyelids. The sutures were placed three millimeters from the upper lid margin with a distance of five millimeters between the sutures. Sutures were placed penetrating skin, extend not deeper than the tarsal plate and crossed the upper eyelid margin anterior to the orifices of the Meibomian glands. Sutures were taken through the lower eyelid and returned in the similar manner to the upper eyelid and knotted. The suture material used was 2/0 silk. A small opening was left at the medial canthus to facilitate medication. The sutures were removed after 7 days for review of the case (Morgan, 2004b).

18. Genticyn eye drops (0.3%), Allergan India Private Limited, Dhar, Madhya Pradesh

3.3.5.6 Lateral lid shortening

This was performed in cases of exposure keratitis due to exophthalmos. Dogs were positioned in lateral recumbency with the affected eye above and were draped. The necessary length of the lid to be shortened, about one fourth to one third of the lid length was estimated. The dorsolateral and ventrolateral lid margins were excised with tenotomy scissors (Plate 2). A triangular area of ventral lid skin was outlined with a Bard Parker blade No. 11 with its base along the lid margin and it was removed by tenotomy scissors. The dorsolateral lid was everted and a triangular portion of the palpebral conjunctiva was excised. The subcutaneous layer including the cut conjunctival margins were apposed in a simple continuous pattern using 4/0 polyglactin 910¹⁹. The lateral canthus was carefully recreated by suturing the lateral edges of the dorsal and ventral canthal margins with a single horizontal mattress sutures using 4/0 polyglactin 910. The dorsal skin triangle was sutured into the ventral skin excision using 4/0 polyglactin 910. The sides of the reapposed skin triangle was sutured with simple interrupted sutures using 4/0 polyglactin 910. A temporary tarsorrhaphy was placed to reduce the tension and potential for dehiscence (Kaswan *et al.*, 1995).

3.3.5.7 Nasal fold resection

The dogs were positioned in lateral recumbency with the affected eye above and were draped. The necessary length of the fold to be resected, which was already estimated with the animal awake, was excised along the direction of the fold. The skin wound was apposed by simple interrupted sutures using fine nylon (Renwick, 2007).

3.3.5.8 Modified Hotz-Celsus operation for anatomical entropion

The dogs were positioned in lateral recumbency with the affected eye above and were draped. The amount of tissue to be removed was already estimated by manually everting the affected eyelid with the animal awake. The eyelid was tensioned intra-operatively by fingers and a proximal incision was made through the full thickness of the

19. Vicryl, Johnson & Johnson Ltd., Aurungabad

skin about 2 mm from the eyelid margin using a Bard Parker blade No. 11. A second incision was made distal and parallel to the first one and was contoured ventrally to include the extra skin as needed. A crescent shaped piece of skin of adequate size to correct the degree of eyelid inversion was excised. A strip of underlying subcutis and orbicularis oculi muscle was also excised. The elliptical skin wound was apposed by simple interrupted sutures using fine nylon (Morgan, 2004b).

3.3.5.9 Resection of prolapsed membrana nictitans gland

The dogs were positioned in lateral recumbency with the affected eye above and were draped. The junction of the gland and the nictitans was crushed with a haemostatic mosquito forceps (Plate 2). The gland was excised with a small scissors. The haemorrhage was controlled by applying light pressure on the surgical wound for a few minutes (Helper, 1981).

3.3.6 Postoperative care

Elizabethan collar was applied to all the animals to prevent self mutilation after the surgical treatment. Parenteral administration of ceftriaxone²⁰ at the rate of 20 mg/kg body weight was given in all the cases on the day of surgical intervention. Topically antibiotics, anti-inflammatory drugs and cycloplegic agents were instilled four times daily till the remission of symptoms. Topical antibiotic ciprofloxacin was changed, if required according to the results of culture and sensitivity of the corneal swab taken on the first day. Orally, cephelexin at the rate of 22 mg/kg body weight for seven days and oral supplementation of multivitamins were advised in all the cases. In those cases where the contra lateral eye was found to have an elevated intraocular pressure, topical instillations of carbonic anhydrase inhibitors like dorzolamide and β blockers like timolol maleate or a combination of α_2 adrenergic agonist, brimonidine tartrate and β blockers like timolol maleate were also advised.

20. Intacef injection (250 mg), Intas Pharmaceuticals Ltd., Ahmedabad, India

3.4 MAIN ITEMS OF OBSERVATION

3.4.1 Signalment

The age, sex, signs noticed by the owner, duration of illness and details of previous medications, if any, were recorded.

3.4.2 Physiological parameters

The rate of respiration (per minute), pulse rate (per minute) and rectal temperature (°C) were recorded in all the cases.

3.4.3 Clinical examination

3.4.3.1 *General condition of the patient*

The general condition of the animal was visually assessed and categorized as excellent, good, fair and poor.

3.4.3.2 *Wet film examination*

One drop of blood was placed on a glass slide, covered with cover slip without air bubbles and observed immediately under low power objective of microscope to identify the presence of any moving blood parasites and observations were recorded on the day of presentation. A re-examination was done after one week in positive cases.

3.4.3.3 *Condition of the eye*

The eye was observed for gross appearance of cornea, presence and nature of ocular discharge, type and extent of lesion, visual function, corneal clarity, Schirmer tear test, corneal opacity/oedema, fluorescein dye test result, vascularization of cornea, conjunctival changes, if any and any other relevant observations.

3.4.3.3.1 Appearance of eye, eyelids and cornea

Gross appearance of the eye and cornea was observed for blepharitis, blepharospasm, photophobia, exophthalmos, enophthalmos, periorbital dermatitis, signs of trauma or scratching, anisocoria, trichiasis, dermoids, entropion etc. were recorded.

3.4.3.3.2 Nature of discharge

The eyes were observed for the presence of lacrimation or discharge. If present, the nature of the discharge was recorded as serous, mucoid and purulent.

3.4.3.3.3 Type and extent of lesion

The type and extent of lesions were assessed and categorized in to superficial/deep, spreading/stationary, epithelial/stromal, descemetocoele and staphyloma.

3.4.3.3.4 Visual function tests

The visual function status of the affected animals was assessed based on the menace reflex and pupillary light reflex.

3.4.3.3.5 Corneal clarity

The clarity of the cornea was assessed based on visual examination. The clarity was categorized as clear (++++), hazy (+++), moderate opacity (++) and complete opacity (+).

3.4.3.3.6 Corneal oedema

The presence of corneal oedema in all the cases was recorded as present (+) or absent (-).

3.4.3.3.7 Fluorescein dye test

The presence of corneal defects and the extent of lesions were assessed by the use of fluorescein dye in the form of sterile impregnated strips²¹ (Plate 3). The strips were moistened with sterile normal saline and were touched on the dorsal bulbar conjunctiva. Care was taken not to touch the cornea to avoid false positive results. Then the dog was allowed to blink a few times or the eyelids were closed to disperse the stain over the cornea. The excess stain was flushed out from the eye with sterile normal saline. The corneal stroma stained fluorescent green whenever there was an epithelial defect but deep corneal defects that extended to the level of Descemet's membrane did not take stain (Miller, 2001).

3.4.3.3.8 Vascularisation of cornea

All the affected eyes were examined for the presence of neovascularisation and its changes till the end of the observation period and were recorded.

3.4.3.3.9 Conjunctival changes

All the affected eyes were observed for conjunctival changes which included generalized congestion, injection of vessels, follicular conjunctivitis and oedema.

3.4.3.3.10 Pigmentation of cornea

All the affected eyes were examined for the presence of pigmentation till the end of the observation period and were recorded.

21. OptiGlo, Ophtechincs Unlimited, Gurgaon, Haryana

3.4.3.3.11 Schirmer tear test (STT)

Schirmer tear test assesses the quantitative tear production of the aqueous portion of the tear film by the use of sterile Schirmer tear test strip²² (Plate 3) calibrated with a millimeter scale. The strip was bent along a preformed notch before it was removed from the package. The strip was placed in the medio-ventral to lateral third of the conjunctival fornix for exactly one minute. Then the strip was removed and the distance of wetness was immediately measured in millimeters. The normal Schirmer tear test value in dogs ranges from 14 to 25 mm of wetting per minute (Moore, 2003).

3.4.3.3.12 Intra ocular pressure (IOP)

The Schioetz indentation tonometer (Plate 3) was used to measure the IOP. Lignocaine hydrochloride 4% solution²³ was instilled one drop each at an interval of five minutes for half an hour to desensitize the cornea. The dog was restrained in a sitting position and the eyes were directed upward so that the cornea was kept in a horizontal plane. The eyelids were separated, the tonometer was allowed to rest by its own weight on the near centre of the up-turned cornea without applying force on the globe and the plunger released with 5.5 gm weight in place. The 7.5 gm and 10 gm weights were used, if necessary. Three consecutive scale readings, which were inversely proportional to the IOP, were recorded. The average value was then converted into mm of Hg using the calibration table provided by Peiffer *et al.*, (1977). The normal IOPs in dogs are 20 ± 5 mm of Hg (Jones, 2004).

3.4.3.3.13 Ophthalmoscopic examination

Ophthalmoscopic examination of the eye was done using direct ophthalmoscope (Plate 3) to detect the presence of anterior synechia, posterior synechia, conditions of lens, retinal detachment, hyphema, hypopion and uveitis.

22. OpStrip, Opthechnics Unlimited, Gurgaon, Haryana

23. Xylocaine Topical (4%), Astra Zeneca Pharma Ltd., Bangalore

3.4.3.3.14 Other relevant observations

Observations like aqueous flare, corneal pigmentation and other specific findings, if any, will be recorded.

3.4.4 Haematological parameters

Blood samples collected in EDTA and were used for haematological evaluation *viz.*, haemoglobin concentration (Hb) by Sahli's acid haematin method, volume of packed red cells (VPRC), erythrocyte sedimentation rate (ESR) by Wintrobe haematocrit method and total leucocyte count (TLC). Blood samples without EDTA were prepared for differential leucocyte count (DLC) and the results were recorded (Metzger and Rebar, 2004).

3.4.5 Biochemical parameters

Random blood glucose level was estimated in all the cases on the day of presentation by using the random blood glucose monitoring system²⁴ (Plate 3).

3.4.6 Culture and sensitivity test of corneal swab

The swabs for culture and sensitivity test (Plate 3) were collected before instillation of any medication. The eyelids were gently retracted and the sides of the sterile swabs were rolled over the area of ulceration. Care was taken not to touch the eyelid margin or eye lashes. The samples were inoculated in Brain Heart Infusion Agar quadrant-streaking method. All the plates were incubated at 37°C for 24 hours and were examined for the presence of bacterial growth. Antibiotic sensitivity test was carried out by Kirby-bauer disc diffusion technique (Ollivier, 2003).

24. Gluco Chek, Aspen Diagnostics (P) Ltd., Delhi

3.4.7 Exfoliative cytology of impression smears from corneal ulcer

The eyelids were gently retracted and a swab was rolled over the ulcerated area of the cornea. Then the swab was gently rolled over a glass slide to prepare the smear. Then the smear was air dried and then stained with Wrights stain to demonstrate the exfoliated cells of the cornea.

3.4.8 Complications

The cases were observed for post-treatment complications like infection, mutilation, pigmentation, dehiscence of sutures and others, if any was recorded. Suitable modifications in the treatment were made accordingly.

All the observations were carried out on the day of first presentation of the case and subsequently on the 10th, 25th, 40th and 60th days, except wet film examination and culture and sensitivity of the corneal swab, which was performed on the first day of presentation only.

3.5 STATISTICAL ANALYSIS

The results were statistically analyzed (Montiani-Ferreira *et al.*, 2004).



- | | |
|-------------------------------------|---|
| A. Lignocaine hydrochloride 4% | G. Dorzolamide hydrochloride ophthalmic solution 2% |
| B. Xylazine hydrochloride injection | H. Timolol maleate eye drops 0.5% |
| C. Atropine sulphate injection | I. Flurbiprofen sodium ophthalmic solution |
| D. Ketamine hydrochloride injection | J. Cyclosporine eye drops 0.1% |
| E. Diazepam injection | K. Carboxymethyl cellulose sodium eye drops 0.5% |
| F. Gentamicin eye drops | L. Ciprofloxacin eye drops |

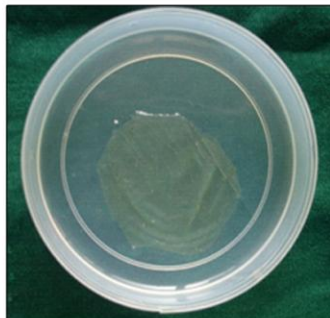
Plate 1: Anaesthetic and therapeutic medicines used in the study



A. Ophthalmic operating microscope



B. Surgical set

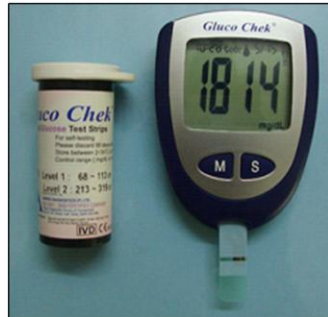


C. Collagen sheet after soaking in gentamicin

Plate 2: Ophthalmic surgical instruments



A. Schiøtz tonometer



B. Random blood glucose monitoring system



C. Ophthalmoscope



D. Schirmer tear test strip
E. Fluorescein dye test strip
F. Sterile corneal swab



G. Elizabethan collar

Plate 3: Ophthalmic diagnostic instruments and postoperative accessories

Results

4. RESULTS

4.1. SELECTION OF CASE (Table 1)

All the Chinese pug dogs presented to the surgery outpatient unit of Veterinary College Hospital, Mannuthy and Kerala Veterinary and Animal Sciences University Hospital, Kokkalai with eye affections were thoroughly examined and were categorised based on the disease condition for which they were presented or diagnosed. All the dogs were subjected to detailed clinical and ophthalmic examination.

The treatment was selected depending upon the type and severity of the conditions, based on the observations made on the first day of presentation. The conditions which responded to the medical management were treated medically and were observed for a period of 60 days. Those conditions which were refractory to medical management or required immediate surgical intervention were subjected to surgical treatment.

Among 578 Chinese pug dogs presented during the period of study from June 2011 to May 2012, 84 dogs had ocular affections. The dogs presented with eye affections comprised 14.53% of the total dogs presented (Fig. 1). Out of the 84 Chinese pugs presented with ocular problems, 40 dogs were diagnosed to have ulcerative keratitis, 10 had glaucoma, 10 had concurrent glaucoma and ulcerative keratitis, 12 dogs had conjunctivitis, seven dogs had pigmentary keratitis, one was presented with proptosis, one had cherry eye, two dogs had concurrent pigmentary keratitis and glaucoma and one had concurrent ulcerative keratitis and keratoconjunctivitis sicca (Fig. 2).

Among the cases presented with ocular affections and subjected to treatment, 22 cases in which the post treatment observations available for a period of 60 days were selected for the study (Fig. 3). The dogs were numbered as Dog

No. I, II, III, IV, V, VI, VII, VIII, IX, X, XI, XII, XIII, XIV, XV, XVI, XVII, XVIII, XIX, XX, XXI and XXII.

The Dogs I to XV had ulcerative keratitis, Dogs XVI and XVII had pigmentary keratitis, Dog XVIII was presented with cherry eye, Dogs XIX to XXII were diagnosed to have glaucoma. The Dogs I, II, IV, VI and XIII with ulcerative keratitis were diagnosed to have glaucoma on contra lateral eyes. The Dog XVII was having concurrent pigmentary keratitis and glaucoma (right eye) and the Dog X was having concurrent bilateral ulcerative keratitis and KCS.

Post treatment observations of the selected cases were made on the 10th, 25th, 40th and 60th day to study the effectiveness of treatment adopted.

4.2 MEDICAL MANAGEMENT

4.2.1 Ulcerative keratitis

The Dogs I and IX with superficial keratitis (Fig. 4 & Fig. 34) and the Dog X with stromal ulcerative keratitis (Fig. 30 & Fig. 31) were given medical treatment. The medical treatment for ulcerative keratitis included topical instillation of antibiotics, anti-inflammatory agents (furbiprofen) and cycloplegic agents (tropicamide) instilled four times daily at an interval of three to four hours. In addition to the medications mentioned above, cephelexin at the rate of 22 mg/kg body weight orally for seven days was also advised for Dog X. Topical tear substitute (0.5% carboxy methyl cellulose sodium) and oral supplementation of multivitamins were advised in all the cases. The topical antibiotic selected initially was ciprofloxacin and was continued in all the cases as culture and sensitivity and clinical response suggested the same. All the animals under medical treatment tolerated the medications and responded to the treatments given.

4.2.2 Glaucoma

Dogs with numbers XIX, XX, XXI and XXII, were diagnosed to have bilateral glaucoma, were treated with topical carbonic anhydrase inhibitors (0.5%

dorzolamide) and β blockers (0.2% timolol maleate), instilled twice daily. In Dogs I, II, IV, VI and XIII with ulcerative keratitis of contra lateral eye, the left eye of Dogs I and IV and the right eye of Dogs II, VI and XIII, diagnosed to have glaucoma were also subjected to treatment with anti-glaucoma drugs. The right eye of the Dog XVII with concurrent pigmentary keratitis, was diagnosed to have glaucoma, was also given the same treatment. Topical instillation of lacrimomimetics or tear substitute (0.5% carboxy methyl cellulose sodium) two to three times daily was also advised in all the cases. All the dogs showed considerable reduction in IOP during the observation period. No side effects of clinical importance were noticed during the period of treatment after administration of anti-glaucomal drugs.

4.2.3 Keratoconjunctivitis sicca

Topical instillation of lacrimomimetics or tear substitute (0.5% carboxy methyl cellulose sodium) two to three times daily was advised on the first day for the Dog X with concurrent bilateral ulcerative keratitis, diagnosed to have KCS. Also, lacrimostimulants (0.1% cyclosporine) two to three times daily was advised on 25th day of observation, when the corneal ulcer was found to be healed. The medications were started for Dog XVIII, with cherry eye, from the seventh postoperative day.

4.2.4 Pigmentary keratitis

The Dog XVI and XVII were subjected to medical management for pigmentary keratitis. The treatment included topical instillation of antibiotic (ciprofloxacin), anti-inflammatory agents (furbiprofen), immunosuppressive agents (0.1% cyclosporine) and tear substitutes (0.5% carboxymethyl cellulose sodium) two to three times daily. The topical application of 0.1% cyclosporine was started for the right eye of Dog XVII, with concurrent glaucoma, on the first day of presentation itself. The treatment for the left eye of Dogs XVI and XVII was started only from the 10th day of observation, after the suture removal, for their respective eyelid and adnexal surgeries.

Elizabethan collar was advised in all the cases to avoid self trauma, until complete healing was observed.

4.3 SURGICAL TREATMENT

Dogs with numbers II, IV, XI and XIV with stromal ulcerative keratitis, Dogs III, VIII and XII with descemetocoele and Dogs V, VI, VII, XIII and XV with staphyloma were subjected to surgical treatment. The dogs with pigmentary keratitis, i.e., Dog XVI, with nasal fold trichiasis of the left eye and Dog XVII, with lower eyelid entropion and exposed globe of the left eye were also subjected to surgical treatment. Surgical treatment was opted for Dog XVIII presented with cherry eye. All the surgical procedures were carried out under general anaesthesia with proper preoperative preparation. All the surgical procedures were conducted with the help of an operating microscope, with a magnification of 0.4X.

4.3.1 Preoperative preparation of patient

All the cases were put under medical therapy with topical instillation of ciprofloxacin eye drops four times for three days prior to surgery. Solid food was withheld for twelve hours before surgery and liquid food for four hours before the surgery in all the cases.

4.3.2 Preoperative preparation of surgical site

The affected eye was thoroughly irrigated with sterile normal saline and the periocular area was cleaned with sterile cotton to remove the accumulated ocular discharge, dirt and tissue debris. The eyelashes were clipped close to the palpebral border. The periocular area was carefully clipped and thoroughly scrubbed with povidone iodine solution (1:50). In Dog XVI, for the correction of nasal fold trichiasis, the hair around the entire nasal fold to be removed was shaved and in case of Dog XVII, for the correction of lower eyelid entropion and to reduce the globe exposure, the hair around the lower eyelid and lateral canthus was shaved followed by thorough scrubbing with povidone iodine solution (1:50). Eyes were irrigated with sterile isotonic normal saline solution and the periocular

area was painted with povidone iodine solution. All the animals were positioned, draped (Fig. 61) and instilled few drops of povidone iodine (5% w/v) ophthalmic solution. The method of preparation of the site for surgery was found to be satisfactory.

4.3.3 Preparation of collagen sheet

The collagen sheet of bovine small intestinal origin became pliable and transparent after soaking in gentamicin eye drops. Thus it enabled easy placement of the sheet over the newly prepared recipient bed on the cornea.

4.3.4 Anaesthesia

All the dogs were subjected to general anaesthesia. Anaesthetic regimen included premedication with atropine sulphate at the rate of 0.045 mg/kg body weight followed by xylazine hydrochloride at the rate of 1.5 mg/kg body weight, both given intramuscularly. General anaesthesia was induced after 15 minutes using ketamine hydrochloride at the rate of 5 mg/kg body weight, administered intramuscularly. A combination of a mixture of xylazine hydrochloride and ketamine hydrochloride, equal quantity by volume and diazepam at the rate of 0.5 mg/kg body weight were found effective in maintaining the anaesthesia. The anaesthetic regimen adopted was found to be satisfactory for the surgical manipulation of cornea in all the cases. Induction and recovery in all the cases were found to be smooth and uneventful.

General anaesthesia following the same regimen was administered in cases of Dog XVI and XVII for suture removal after their respective eyelid and adnexal surgeries. Anaesthesia was not administered in all the cases for the removal of temporary tarsorrhaphy sutures and for measurement of intraocular pressure. In these cases topical administration of 4% lignocaine was done, one drop instilled four times at an interval of five minutes.

4.3.5 Surgical technique

All the surgical procedures were conducted with the help of an operating microscope, with a magnification of 10 X. It provided better visualisation of operating field and facilitated better manipulation of the cornea.

Debridement was done in Dogs II and XI with stromal ulcer (Fig. 6 & Fig. 22) using dry cotton tipped applicator and removed the abnormal epithelium and debris from the stromal surface. The collagen sheet was placed over the newly prepared recipient bed on the cornea and was sutured on to the bulbar conjunctiva using 4/0 silk (Fig.7), followed by placement of temporary tarsorrhaphy sutures (Fig. 58).

Dog IV with melting stromal ulcer (Fig. 16) was subjected to Debridement and temporary tarsorrhaphy suture placement. Since the condition progressed into a staphyloma by the seventh postoperative day (Fig. 17), collagen sheet was placed followed by temporary tarsorrhaphy suture placement.

In Dog XIV with stromal ulcer (Fig. 25), debridement alone was done followed by placement of temporary tarsorrhaphy suture placement.

Dogs III and VIII presented with small descemetocoele (Fig. 10 & Fig. 28) were subjected to temporary tarsorrhaphy suture placement alone after irrigation with normal saline.

The Dog XII with descemetocoele had an extensive stromal ulcer (Fig. 36) and was subjected to debridement of the stroma followed by collagen sheet placement. The eye was protected by temporary tarsorrhaphy suture placement. Due to the development of hypopion noticed on the seventh postoperative day, the anterior chamber was flushed with balanced salt solution followed by temporary tarsorrhaphy.

Among the Dogs V, VI, VII, XIII and XV presented with staphyloma (Fig. 13, Fig. 20 & Fig. 40), the protruded iris was found to be covered with fibrin clot except in Dog VII (Fig. 48). Fibrin clots were removed using corneal scissors. Iris

in all these cases were severely adhered to the cornea and no seepage of the aqueous humor was noticed. As the adhesions between the iris and cornea were difficult to be separated, iridectomy was performed. Since the corneal wound edges were not apposed on suturing, it was kept as such in all these dogs. The edges of the corneal wound were scarified with the tip of BP blade No. 11. In Dogs V, VI and VII, collagen sheet was placed over the newly prepared cornea and was sutured on to the bulbar conjunctiva. In the case of Dog XIII, a third eyelid flap was placed and was sutured on to the upper eyelid. Temporary tarsorrhaphy sutures were placed in all the dogs to protect the cornea.

Dog XV, presented with staphyloma, had extensive stromal injury in addition to lens extrusion. Iridectomy was performed and balanced salt solution was infused into the space between the adhered iris and Descemet's membrane. Debridement of the remnants of corneal tissue was carried out using dry cotton tipped applicator. Collagen sheet was sutured over the cornea followed by temporary tarsorrhaphy suture placement.

The Dog XVI with pigmentary keratitis was found to have nasal fold trichiasis in the left eye (Fig. 52). Nasal fold resection was done in this case. The skin wound was sutured in horizontal mattress suture pattern using nylon (Fig.53).

In Dog XVII with pigmentary keratitis, an exposed globe with lower eye lid entropion was noticed in the left eye (Fig. 60). Surgical correction of lower eyelid entropion of left eye was done using modified Hotz-Celsus operation for anatomical entropion. In order to reduce the exposed surface area of the globe of the same eye, permanent lateral canthal shortening was performed (Fig. 62).

The prolapsed membrana nictitans gland in Dog XVIII presented with cherry eye (Fig. 56) was treated with surgical excision of the gland (Fig. 57).

The surgical procedure in all the cases was uneventful without any complication.

Temporary tarsorrhaphy was done in all the surgical cases with ulcerative keratitis and was found to be effective in protecting and supporting the corneal healing. Also, it provided an additional support to the collagen sheet sutured and fixed over the cornea. Partial disruption of the tarsorrhaphy sutures were noticed in Dog IV, XII and XIII. The sutures in all other cases were intact and were removed on the 7th postoperative day.

The collagen sheet was sutured over the bulbar conjunctiva after covering the corneal defect completely. Suturing enabled proper placement and fixation of the sheet over the cornea. Also, the temporary tarsorrhaphy sutures supported the sheet. Complete dissolution of collagen sheet was noticed by the seventh postoperative day (Fig. 8).

The nasal fold resection done in Dog XVI with nasal fold trichiasis enabled to avoid irritation and further damage to the cornea.

The smaller palpebral fissure created in Dog XVII by permanent lateral canthal shortening improved blink function, provided better tear film distribution and decreased the tendency for corneal desiccation.

The surgical wound in cases of Dog XVI and XVII was found to heal without any complication and the sutures were removed on the seventh postoperative day (Fig. 54 & Fig. 63).

4.3.6 Postoperative care

Elizabethan collar was applied to all the animals during the postoperative period. It was found very effective in preventing self mutilation. In order to counteract infection and inflammation, topical and systemic antibiotics and topical anti-inflammatory agents were administered. Parenteral administration of ceftriaxone at the rate of 20 mg/kg body weight was done in all the cases on the day of surgical intervention. Topically, ciprofloxacin and flurbiprofen were instilled four times daily at an interval of 10 minutes till complete healing. Orally, cephelexin at the rate of 22 mg/kg body weight was administered for the first

seven postoperative days. Tropicamide was instilled topically four times a day till the remission of inflammation was noticed. Oral supplementation of multivitamins was advised in all the cases.

Tropicamide applied topically was successful in preventing anterior synechiae formation in all the cases except in Dogs V and XII. Dog V had anterior synechiae on the day of presentation itself and Dog XII developed anterior synechiae during the course of surgical treatment subsequent to the development of hypopion (Fig. 38).

The Dogs II, IV, VI and XIII, that underwent surgical treatment for ulcerative keratitis and Dog XVII for eyelid surgery, were found to have an elevated intraocular pressure in the contra lateral eye. These dogs were treated medically for glaucoma.

4.4 MAIN ITEMS OF OBSERVATION

4.4.1 Signalment (Table 2, 3 and 4)

The age of the Chinese pug dogs selected in this study ranged from 2 months to 60 months with an average of 16.14 ± 2.93 months. Out of the 22 cases selected for the study, 15 dogs had ulcerative keratitis, 10 dogs had glaucoma, 2 dogs had pigmentary keratitis and one dog had cherry eye. Among the dogs with ulcerative keratitis, 5 dogs had concurrent glaucoma on contralateral eye and one had concurrent bilateral KCS. Among the dogs with pigmentary keratitis, one dog had concurrent unilateral glaucoma. Nine out of the 22 dogs selected for the study were males and the rest were females.

Among the dogs with ulcerative keratitis, nine were females and the rest were males with an average age of 14.00 ± 2.45 , ranging from 2 months to 36 months. The dog presented with concurrent KCS was a puppy of 2 months. Out of the 10 dogs with glaucoma, nine were females with an average age of 18.10 ± 2.74 , ranging from 8 months to 36 months. The ages of the dogs presented with

pigmentary keratitis were 36 and 60 months. Prolapse of membrana nictitans gland was noticed in a puppy aged 3 months.

The average duration of illness of dogs with ulcerative keratitis was 5.27 ± 0.77 days, ranging from 2 to 11 days, before presenting the case to the hospital. Among them 4 dogs received topical medication with corticosteroids. The duration of illness of dogs with pigmentary keratitis was 6 and 7 months.

4.4.2 Physiological parameters (Table 5)

4.4.2.1 Rate of respiration

The mean rate of respiration (per minute) in all the cases selected in the study was 26.73 ± 1.33 on the day of presentation. It was 28.09 ± 1.23 , 27.55 ± 0.90 , 27.45 ± 0.87 and 27.09 ± 0.82 on the 10th, 25th, 40th and 60th day of observations, respectively. All the values were within the normal range.

4.4.2.2 Rectal temperature

The mean rectal temperature (°C) in all the cases selected in the study was 38.95 ± 0.10 on the day of presentation. It was 38.87 ± 0.07 , 38.84 ± 0.06 , 38.78 ± 0.06 and 38.76 ± 0.06 on the 10th, 25th, 40th and 60th day of observations, respectively. All the values were within the normal range, except for dog XIII and XVI on the day of presentation, which on subsequent observation day was found to be normal.

4.4.2.3 Pulse rate

The mean pulse rate (per minute) in all the cases selected in the study was 90.73 ± 2.16 on the day of presentation. It was 90.45 ± 1.87 , 91.00 ± 1.91 , 90.64 ± 1.74 and 90.09 ± 1.38 on the 10th, 25th, 40th and 60th day of observations, respectively. All the values were within the normal range.

4.4.3 Clinical examination

4.4.3.1 General condition of the patient

All the dogs selected in the study were found to have a general condition scored good except Dog XVII, which had poor condition on the first day and recovered good health

4.4.3.2 Wet film examination

Moving blood parasites were not detected preoperatively in any of the cases except in Dog XIII and XVI. On reexamination on the 10th day, no moving blood parasites could be detected.

4.4.3.3 Condition of the eye

4.4.3.3.1 Appearance of eye, eyelids and adnexa and cornea (Table 6)

The eye appeared to be normal on the day of presentation in all the dogs selected in the study except in Dogs IV, XVII, XIX, XX and XXII with glaucoma which appeared buphthalmic. Relative exophthalmos was noted in all the dogs with keratitis. Phthisis bulbi was noticed in the right eye of Dog XVI. Blepharospasm was noticed in Dogs I, III, IX, XI, XII and XIV on the day of presentation which resolved completely by the tenth day of observation except in Dog XII. In Dog III and XII, blepharodema was also noticed on the tenth day of observation which resolved by the next day of observation. Episcleral vessel congestion was noticed in all dogs with glaucoma on the first day of observation. Complete pigmentation of both eyes was noticed in dogs with pigmentary keratitis (Fig. 52 & Fig. 59). The left eye of the Dog XVI with pigmentary keratitis was found to have nasal fold trichiasis and the left eye of the Dog XVII with pigmentary keratitis was found to have lower eyelid entropion. In both the dogs with pigmentary keratitis, accumulation of hair, dirt and debris was noticed on the central cornea in addition to xerophthalmia (Fig. 52). Pigmentation of cornea was

also noticed in Dog XXII with glaucoma (Fig. 55). Corneal oedema was noticed in all dogs with ulcerative keratitis and in Dog XXII with glaucoma.

4.4.3.3.2 Nature of discharge

Epiphora was noticed in Dogs I, III, IV, VIII, IX, XII and XIV with ulcerative keratitis, which was found to have subsided by the tenth day of observation. The nature of discharge was clear in all the cases except in Dogs IV, V, X, XIII and XV. The nature of discharge in Dogs IV, V, X, XIII and XV was mucopurulent. The discharge in these dogs became clear by the tenth day of observation except in Dog X with concurrent KCS. The discharge in this dog became clear by the 25th day of observation.

4.4.3.3.3 Type and extent of lesion

The Dogs I and IX with superficial keratitis, treated medically healed completely with a mild scar in the center by the tenth day of observation (Fig. 5 & Fig. 35).

The depth and extent of stromal ulcer in dog X treated medically and Dogs II, XI and XIV treated surgically got reduced by the 25th day of observation (Fig.27). Stromal melting was observed in Dog IV with stromal ulcer and on the tenth day of observation, the ulcer was found to have progressed into a staphyloma without anterior synechiae (Fig. 17). On subsequent observations, progressive reduction in the depth and extent of the ulcer was noticed.

The depth and extent of descemetocele in Dogs III and VIII treated surgically showed complete healing by 25th day of observation. The Dog XII, with descemetocele, had an extensive stromal ulcer treated surgically. The descemetocele had progressed into a small healing staphyloma that was noticed on the tenth day of observation. Even though the ulcer was found to granulate, the eye developed hypopion. By 25th day of observation, the hypopion was found to have resolved completely, but resulted in permanent anterior synechiae. The Dog

VIII with ulcerative keratitis was found to have a superficial ulcer when presented on the 60th day of observation (Fig. 29).

In all the dogs presented with staphyloma, the protruded iris was found to have covered with fibrin clot and debris, except in Dog VII. The dog V with staphyloma was presented with an already collapsed anterior chamber with anterior synechiae. Corneal healing was noticed in this dog by the tenth day of observation (Fig. 44) and was completed by 25th day, but could not regain its shape. In Dogs V, VI, VII and XIII, the protruded iris was found to seal the corneal wound and thereby the leakage of aqueous humor was blocked. The lesion in centre of cornea of all these dogs became shallow on tenth day of observation (Fig. 14, Fig. 41 & Fig. 49) and by 25th day, the cornea healed completely. But, in Dog XIII, a small facet was noticed on the central cornea by 40th day of observation (Fig. 43). Dog XV was presented with staphyloma in addition to extensive stromal injury and lens extrusion. The iris was found to be infected and covered with fibrin clot and debris. On 25th day of observation, an elastic membrane simulating the Descemet's membrane was found bulging with a newly formed anterior chamber (Fig. 45). Healing cornea with granulation was noticed by 40th day of observation with an aqueous filled anterior chamber (Fig. 46).

In both the dogs with pigmentary keratitis, the sclera was found to be pigmented in addition to the densely pigmented cornea. By day 25, further accumulation of corneal pigments was not observed.

4.4.3.3.4 Visual function tests (Table 7)

All the dogs selected in the study had normal palpebral and corneal reflexes throughout the observation period.

In all the dogs with ulcerative keratitis, menace reflex was found to be sluggish in the affected eye on the day of presentation and was absent in Dogs V and XV, throughout the observation period. Pupillary light reflex could not be assessed in dogs with ulcerative keratitis from the day of presentation until the cornea attained clarity. The menace and pupillary light reflex in all the dogs with

ulcerative keratitis could be assessed as normal by day 25, except in Dogs IV, V, XII, XIII and XV. In Dogs IV and XIII, both the reflexes were assessed to be normal by 40th day. The Dog X lost menace reflex by 40th day of observation due to complete bilateral corneal pigmentation. By day 60, all the dogs with ulcerative keratitis regained normal visual function except Dogs V, X, XII and XV.

In dogs with pigmentary keratitis, menace reflex was absent in both the eyes throughout the observation period. In these dogs, the pupillary light reflex could not be assessed. Also, the blinking of eyes in these dogs was found to be incomplete.

The pupillary light reflex was found to be sluggish in all the dogs with glaucoma on the day of presentation except in Dog XVII, with complete corneal pigmentation, where the reflex could not be assessed. In dogs with glaucoma, where the contralateral eye was found to have ulcerative keratitis, the pupillary light reflex could not be assessed. All the dogs showed normal pupillary light reflex during the subsequent days of observation.

4.4.3.3.5 Corneal clarity (Table 8)

Dogs with numbers I, II, III, VI and VIII were presented with moderate opacity and all the other dogs with ulcerative keratitis were presented with complete opacity.

The cornea attained clarity in all the cases presented with ulcerative keratitis by the 60th day of observation except in Dogs IV, XII, XIII and XV.

In Dogs I and IX presented with superficial ulcers, cornea became completely clear with a mild scar at the center by the 10th day after presentation. The scar resolved later.

Among the five dogs presented with stromal ulcers, corneal clarity was noticed in Dogs X and XIV by the 25th day of observation, while Dogs II and XI showed central haziness and Dog IV had moderate opacity (Fig. 18). Dog II

achieved corneal clarity by the 40th day of observation (Fig. 9) and the cornea in Dogs XI became clear by the 60th day of observation (Fig. 24). Central haziness remained in Dog IV on the 60th day of observation (Fig. 19).

In Dogs III and VIII, with descemetocele, corneal clarity was noticed by the 25th day of observation. The Dog XII with descemetocele which had an extensive stromal ulcer, progressed into a staphyloma by the 10th day of observation. Moderate opacity was noticed in this dog during epithelialisation that remained on the 60th day of observation also.

Among the dogs presented with staphyloma, only the Dog V could achieve corneal clarity by the 60th day of observation. In Dog XV, complete damage of cornea was noticed on the day of presentation. This dog also regained corneal clarity by the 40th day of observation except at a central zone of complete opacity showing epithelialisation in progression (Fig. 46) that remained as such on the 60th day of observation (Fig. 47). Moderate opacity was noticed in Dogs VI and VII by the 10th day of observation and in Dog XIII by the 25th day of observation. Both the Dogs VI and VII showed a central haziness on the 60th day of observation. In Dog XIII, the cornea remained moderately opaque with granulation even on the 60th day of observation.

4.4.3.3.6 Corneal oedema (Table 9)

Corneal oedema was noticed in all the cases, presented with ulcerative keratitis on the day of presentation, with complete absence noticed by 40th day of observation.

Complete absence of oedema was noticed in dogs I and IX presented with superficial ulcers by the 10th day of observation.

Among the five dogs presented with stromal ulcers, corneal oedema was found to be resolved in Dogs II, X, XI and XIV by the 25th day of observation. Oedema resolved completely in dog IV by 40th day of observation.

In Dogs III, VIII and XII presented with descemetocoele, complete absence of oedema was noticed by the 25th day of observation, except in Dog XII. Oedema resolved completely in dog XII by 40th day of observation.

All the dogs presented with staphyloma showed complete absence of corneal oedema by the 25th day of observation except Dog XIII and XV, which was found to be resolved during the subsequent day of observation.

Among the dogs with glaucoma, corneal oedema was noticed in Dog XXII (Fig. 55), which was found to be resolved by the 25th day of observation.

4.4.3.3.7 Fluorescein staining test (Table 10)

FST was positive in all the dogs presented with ulcerative keratitis on the day of presentation. Absence of fluorescein dye retention by the 10th day of observation was noticed only in Dogs I and IX with superficial ulcer. On the 25th day of observation, fluorescein dye retention was not noticed in any of the dogs with ulcerative keratitis in the study except Dog XII (Fig. 37), XIII (Fig. 42) and XV (Fig. 45). By 40th day of observation, these two dogs were also stained negative (Fig. 38, Fig. 43 & Fig. 46).

4.4.3.3.8 Vascularisation of cornea (Table 11)

Vascularisation of cornea was noticed in Dogs II, V, VI, X, XI, XIII, XIV and XV with ulcerative keratitis on the day of presentation itself.

Complete absence of vascularisation was noticed throughout the observation period in Dogs I and IX presented with superficial ulcers.

All the dogs with stromal ulcer had vascularisation on the day of presentation itself, except dog IV. Vascularisation of cornea in dog IV was noticed by the seventh postoperative day and got intensified by the 25th day of observation. By 25th day of observation, vascularisation in all the dogs with stromal ulcer was found to have resolved except in Dog IV. In this dog regression was noticed by the 40th day of observation.

All the dogs with descemetocoele did not show vascularisation on the day of presentation. In these dogs vascularisation was noticed by the seventh postoperative day. The vascularity disappeared in dogs with descemetocoele by 25th day of observation, except in Dog XII. In this dog, complete absence of vascularity was noticed by the 40th day of observation.

Among the dogs presented with staphyloma, all the dogs had vascularisation on the day of presentation itself, except in Dog VII. In this dog, vascularisation was noticed by the seventh postoperative day (Fig. 49). Deep corneal vessels which formed 360^o perilimbal pattern were noticed in Dog XIII (Fig. 42). In Dogs V and VII, complete absence of vascularization of cornea was noticed by the 25th day of observation and in dog VI, the avascularity was achieved by 40th day of observation. Vascularisation persisted in Dogs XIII and XV (Fig. 43 & Fig. 46) even on the 40th day of observation, but complete regression was noticed by the 60th day of observation.

The Dog XVI, presented with pigmentary keratitis, showed vascularisation of cornea on the day of presentation and the 10th day of observation, which regressed by the 25th day of observation.

4.4.3.3.9 Conjunctival changes (Table 12)

Conjunctival hyperaemia was noticed in all the dogs with keratitis on the day of presentation. Chemosis was noticed in Dog V and XII on the day of presentation. Chemosis in Dog V resolved by the subsequent observation day and in Dog XII, by the 25th day of observation. In Dogs I and IX, with superficial keratitis, conjunctival hyperaemia was absent on the 10th day of observation. All the other dogs with ulcerative keratitis showed conjunctival hyperaemia on the 10th day of observation also. Mild conjunctival hyperaemia persisted on the 25th day of observation in dog IV with stromal ulcer, Dog XII with descemetocoele and Dogs VI, XIII and XV with staphyloma. In these dogs, conjunctival hyperaemia resolved completely by the 40th day of observation.

Conjunctival hyperaemia was found resolved by the 10th day of observation in both the dogs with pigmentary keratitis.

4.4.3.3.10 Pigmentation of cornea (Table 13)

All the dogs in the study with ulcerative keratitis showed pigmentation during the subsequent observations (Fig. 12, Fig. 19, Fig. 24 & Fig. 51), but of varying degrees, except in Dogs VIII and IX. In all these dogs, pigmentation was found to begin from the medial and ventral limbal region by the 25th day of observation except in Dog VI. In Dog XV, pigmentation was noticed on the day of presentation itself. No further accumulation of pigment was noticed in these dogs once complete corneal healing was observed except in Dogs V, VI and X. In Dogs V and X, complete pigmentation of cornea, and in Dog VI, mild pigmentation was noticed by the 60th day of observation.

In Dog X with concurrent bilateral ulceration and KCS, complete pigmentation of both the eyes was observed by the 25th day of observation (Fig. 32 & Fig. 33). The subsequent observation also showed the same, without further pigment deposition.

In dogs with Pigmentary keratitis, Dog XVI and XVII, no further accumulation of pigments were noticed throughout the entire period of observation.

Corneal pigmentation was noticed in Dog XVIII with cherry eye on the day of presentation itself. The pigmentation remained as such till the final observation period.

The pigmentation noticed in the cornea of Dog XXII, with glaucoma on the day of presentation remained as such till the final observation day (Fig. 55).

4.4.3.3.11 Schirmer tear test (Table 14)

The mean values of STT readings of the right eye were 16.32 ± 0.88 , 16.37 ± 0.77 , 15.95 ± 0.68 , 16.00 ± 0.50 and 16.23 ± 0.37 on the day of presentation,

10th, 25th, 40th and 60th day respectively. The mean values of STT readings of the left eye were 17.50 ± 0.99 , 17.36 ± 0.86 , 16.36 ± 0.72 , 16.82 ± 0.38 and 16.73 ± 0.23 on the day of presentation, 10th, 25th, 40th and 60th day respectively.

All the values were within the normal range except in Dogs X, XVI, XVII and XVIII. Higher STT values were noticed in dogs with ulcerative keratitis on the day of presentation. The STT values of right eye in Dog X were 3, 4 and 5 and that of left eye were 4, 3 and 3 on the day of presentation, 10th and 25th day of observation respectively. In this dog a considerable increase in tear production was noticed during the 40th and 60th day of observation.

In the dogs with pigmentary keratitis, Dog XVI and XVII, the STT values were less than the normal range on the day of presentation. A considerable rise in STT values were noticed in these dogs by 25th day and the subsequent days of observations.

In Dog XVIII, with cherry eye, STT value of the affected eye was found to be reduced on the 10th day of observation. On subsequent observation the values were found to lie within the normal range.

4.4.3.3.12 Intra ocular pressure (Table 15)

The mean IOP in the right eye and left eye of all the dogs with glaucoma was found to be 56.03 ± 3.38 and 49.61 ± 4.59 respectively on the day of presentation. A drastic reduction in mean IOP was noticed in both the eyes by the 10th day of observation and subsequent observations showed gradual reduction in IOP. By day 60, the mean IOP in the right eye and left eye was found to be 30.94 ± 0.61 and 30.41 ± 0.74 respectively.

4.4.3.3.13 Ophthalmoscopic examination

The ophthalmic examination did not reveal any significant abnormalities in dogs with glaucoma. Mild uveitis accompanied all the dogs with ulcerative keratitis. Among the dogs with ulcerative keratitis, severe uveitis with hypopion

was detected in Dog XII on the seventh postoperative day which resolved by the 25th day of observation but developed anterior synechiae.

4.4.3.3.14 Other relevant observations

Xerophthalmia was noticed in Dogs XVI and XVII presented with pigmentary keratitis on the day of presentation. Absence of corneal drying was noticed in these dogs by the 10th day of observation. In Dog XVII, improved lid function was noticed by the seventh postoperative day.

4.4.4 Haematological parameters (Table 16)

4.4.4.1 Haemoglobin concentration

The mean values haemoglobin concentration (g/dl) of all the dogs selected in the study were 12.70 ± 0.33 , 13.26 ± 0.25 , 13.58 ± 0.30 , 13.12 ± 0.29 and 13.12 ± 0.3 on the day of presentation, 10th, 25th, 40th and 60th day respectively. All the values were within the normal range.

4.4.4.2 Volume of packed red cells

The mean values of volume of packed red cells (%) of all the dogs selected in the study were 37.14 ± 1.01 , 39.09 ± 0.81 , 40.41 ± 0.93 , 39.14 ± 0.88 and 39.09 ± 0.93 on the day of presentation, 10th, 25th, 40th and 60th day respectively. All the values were within the normal range.

4.4.4.3 Erythrocyte sedimentation rate

The mean values of erythrocyte sedimentation rate (mm/hr) of all the dogs selected in the study were 6.36 ± 0.28 , 6.68 ± 0.34 , 6.86 ± 0.42 , 7.18 ± 0.38 and 7.23 ± 0.38 on the day of presentation, 10th, 25th, 40th and 60th day respectively. All the values were within the normal range.

4.4.4.4 Total leucocyte count

The mean values of total leucocyte count ($\times 10^3/\text{cmm}$) of all the dogs selected in the study were 11.83 ± 0.45 , 11.30 ± 0.37 , 11.73 ± 0.36 , 11.91 ± 0.52 and 11.24 ± 0.36 on the day of presentation, 10th, 25th, 40th and 60th day respectively. All the values were within the normal range.

4.4.4.5 Differential leucocyte count

The mean differential count for neutrophils (%) of all the dogs selected in the study were 74.33 ± 1.98 , 73.77 ± 1.76 , 73.33 ± 1.62 , 72.33 ± 2.56 and 73.33 ± 1.48 on the day of presentation, 10th, 25th, 40th and 60th day respectively. The mean percentages for lymphocytes were 27.76 ± 1.44 , 26.52 ± 1.26 , 26.12 ± 1.64 , 26.32 ± 1.22 and 26.0 ± 1.21 on the day of presentation, 10th, 25th, 40th and 60th day respectively. The mean percentages for eosinophils were 0.5 ± 0.22 , 0.5 ± 0.33 , 0.33 ± 0.21 , 0.66 ± 0.33 and 0.66 ± 0.33 on the day of presentation, 10th, 25th, 40th and 60th day respectively. The mean percentage for monocytes was 0.16 ± 4.47 , 0.16 ± 4.51 , 0.16 ± 4.47 , 0.16 ± 4.63 and 0.16 ± 4.58 on the day of presentation, 10th, 25th, 40th and 60th day respectively. All the values were within the normal range.

4.4.5 Random blood glucose level (Table 17)

The mean random blood glucose level (mg/dl) of all the dogs selected in the study on the day of presentation was 97.00 ± 2.41 . All the values were within the normal range.

4.4.6 Culture and sensitivity test of corneal swab (Table 18)

No growth was observed in Dogs I, VI, VIII and IX after incubation for 24 hours. The predominant isolate was Gram positive cocci with colony characteristics of *Staphylococcus* spp. isolated from Dogs III, V, VII, X, XI, XII, XIV and XV. The other isolates included Gram negative bacilli, isolated from Dogs II, IV and X and Gram positive bacilli which was isolated from Dog XIII. All the isolates were found sensitive to ciprofloxacin except Dogs II and X. The

subsequent observation in these dogs showed clinical response to ciprofloxacin and hence the antibiotic was not changed.

4.4.7 Exfoliative cytology of impression smears of corneal ulcer (Table 19)

Nucleated epithelial cells and anuclear keratinized cells were seen in all the dogs on the day of presentation. Nucleated epithelial cells were seen in higher quantity in dogs with deep defects. The number of these cells was found to decrease gradually during the healing process. Anuclear keratinized cells were seen in the smear of all the dogs till the completion of corneal healing. Polymorphonuclear cells were noticed only in dogs with deep and full thickness defects. These cells disappeared by the 25th day of observation in all the dogs except in Dogs IV, VI, XII, XIII and XV. By 60th day of observation, polymorphonuclear cells disappeared in these dogs also.

4.4.8 Complications (Table 20)

Partial disruption of temporary tarsorrhaphy sutures were noticed in Dogs IV, XII and XIII (Fig. 17) and dislodgement of third eyelid flap was observed in Dog XIII by the 7th postoperative day. The melting ulcer in dog IV, and the descemetocele in Dog XII, progressed into a small staphyloma by the 10th day of observation. Dog V had an already formed anterior synechiae and resulted in phthisis bulbi by the 60th day of observation (Fig. 21). Dog XII developed severe uveitis with hypopion by the seventh postoperative day. Anterior synechiae formation was noticed in this dog on the 25th day of observation and phthisis bulbi was appreciated by the 60th day of observation (Fig. 39). In Dog XV, the cornea was covered with deeply pigmented granulation tissue with resultant enophthalmos by the 60th day of observation. In Dog X, complete bilateral pigmentation and in Dog XI and Dog XIII, dense pigmentation was noticed in the cornea of affected eye by the 60th day of observation. Complete bilateral pigmentation persisted in Dogs XVI and XVII throughout the observation period. In Dog XVIII, with cherry eye, decrease in tear production was noticed after the removal of third eyelid gland by the 10th observation day.

Table 1: Anamnesis of Chinese pugs selected for study

SL No.	Dog No.	Age (Months)	Sex	Condition of the eye
1	I	18	Female	Ulcerative keratitis and glaucoma
2	II	24	Female	Ulcerative keratitis and glaucoma
3	III	4	Female	Ulcerative keratitis
4	IV	9	Female	Ulcerative keratitis and glaucoma
5	V	18	Male	Ulcerative keratitis
6	VI	18	Female	Ulcerative keratitis and glaucoma
7	VII	18	Male	Ulcerative keratitis
8	VIII	5	Female	Ulcerative keratitis
9	IX	8	Male	Ulcerative keratitis
10	X	2	Male	Ulcerative keratitis and keratoconjunctivitis sicca
11	XI	21	Male	Ulcerative keratitis
12	XII	3	Female	Ulcerative keratitis
13	XIII	18	Female	Ulcerative keratitis and glaucoma

Table 1: Anamnesis of Chinese pugs selected for study (Contd...)

SL No.	Dog No.	Age (Months)	Sex	Condition of the eye
14	XIV	8	Male	Ulcerative keratitis
15	XV	36	Female	Ulcerative keratitis
16	XVI	60	Male	Pigmentary keratitis
17	XVII	36	Female	Pigmentary keratitis and glaucoma
18	XVIII	3	Male	Cherry eye
19	XIX	8	Female	Glaucoma
20	XX	12	Male	Glaucoma
21	XXI	18	Female	Glaucoma
22	XXII	8	Female	Glaucoma
Mean ± SE		16.14±2.93		

Table 2: Anamnesis of Chinese pugs with ulcerative keratitis selected for the study

SL No.	Dog No.	Age (Months)	Sex	Condition of the eye	Eye affected	Duration of illness (days)	Previous medications, if any	Treatment adopted
1	I	18	Female	Superficial ulcer	Right	3	Nil	Medical
2	II	24	Female	Stromal ulcer	Left	8	Nil	Surgical
3	III	4	Female	Descemetocele	Left	5	Pyochlor dexta eye drops	Surgical
4	IV	9	Female	Stromal melting ulcer	Right	3	Nil	Surgical
5	V	18	Male	Staphyloma	Right	10	Ciplox-D eye drops	Surgical
6	VI	18	Female	Staphyloma	Left	7	Ciplox eye drops	Surgical
7	VII	18	Male	Staphyloma	Left	2	Nil	Surgical
8	VIII	5	Female	Descemetocele	Right	3	Ofloxacin eye drops	Surgical

Table 2: Anamnesis of Chinese pugs with ulcerative keratitis selected for the study (Contd...)

SL No.	Dog No.	Age (Months)	Sex	Condition of the eye	Eye affected	Duration of illness (days)	Previous medications, if any	Treatment adopted
9	IX	8	Male	Superficial ulcer	Left	3	Nil	Medical
10	X	2	Male	Stromal ulcer	Bilateral	7	Nil	Medical
11	XI	21	Male	Stromal ulcer	Left	7	Pyochlor dexta eye drops	Surgical
12	XII	3	Female	Descemetocoele	Right	2	Nil	Surgical
13	XIII	18	Female	Staphyloma	Left	6	Nil	Surgical
14	XIV	8	Male	Stromal ulcer	Left	2	Nil	Surgical
15	XV	36	Female	Staphyloma	Left	11	Ciplox-D eye drops	Surgical
Mean ± SE		14.00±2.45				5.27 ± 0.77		

Table 3: Anamnesis of Chinese pugs with glaucoma selected for the study

SL No.	Dog No.	Age (Months)	Sex	IOP (mm of Hg)		Buphthalmos	Treatment adopted
				Right eye	Left eye		
1	I	18	Female	×	43.4	Absent	Medical
2	II	24	Female	46.3	×	Absent	Medical
3	IV	9	Female	×	55.4	Present	Medical
4	VI	18	Female	52.3	×	Absent	Medical
5	XIII	18	Female	46.3	×	Absent	Medical
6	XVII	36	Female	69.9	28.4	Present	Medical
7	XIX	8	Female	69.9	62.2	Present	Medical
8	XX	24	Male	58.7	62.2	Present	Medical
9	XXI	18	Female	55.4	52.3	Absent	Medical
10	XXII	8	Female	49.4	43.4	Present	Medical
Mean ± SE		18.10 ± 2.74		56.03 ± 3.38	49.61 ± 4.59		

×Could not be assessed due of the presence of corneal ulcer

Table 4: Anamnesis of Chinese pugs with pigmentary keratitis selected for the study

SL No.	Dog No.	Age (Months)	Sex	Eyelid and adnexal abnormalities	Eye affected	Duration of illness (months)	Previous medications, if any	Treatment adopted
1	XVI	60	Male	Nasal fold trichiasis	Left	7	Chlormycetin applicaps Ciplox-D eye drops	Surgical
2	XVII	36	Female	Lower lid entropion Lagophthalmos (left eye)	Bilateral	6	Nil	Medical (right eye) Surgical (left eye)
Mean ± SE		48 ± 12				6.5 ± 0.5		

Table 5: Mean values of physiological parameters of the cases studied

Parameters	Days of observation				
	1st day	10th day	25th day	40th day	60th day
Rate of respiration (per minute)	26.73 ± 1.33	28.09 ± 1.23	27.55 ± 0.90	27.45 ± 0.87	27.09 ± 0.82
Rectal temperature (°C)	38.95 ± 0.10	38.87 ± 0.07	38.84 ± 0.06	38.78 ± 0.06	38.76 ± 0.06
Pulse rate (per minute)	90.73 ± 2.16	90.45 ± 1.87	91.00 ± 1.91	90.64 ± 1.74	90.09 ± 1.38

Table 6: General and clinical appearance of the right eye (R) and left eye (L) of the cases studied

SL No.	Dog No.	Eye	Eyelids and adnexa	Conjunctiva	Cornea	Sclera
1	I	R	Normal	Congested	Superficial ulcer with corneal opacity	Episcleral congestion
		L	Normal	Normal	Normal	Episcleral congestion
2	II	R	Normal	Normal	Normal	Episcleral congestion
		L	Normal	Congested	Stromal ulcer with mild corneal opacity	Episcleral congestion
3	III	R	Normal	Normal	Normal	Normal
		L	Normal	Congested	Descemetocele with corneal opacity	Normal
4	IV	R	Normal	Congested	Melting ulcer with corneal opacity	Normal
		L	Normal	Normal	Normal	Episcleral congestion
5	V	R	Normal	Swollen	Staphyloma	Normal
		L	Normal	Normal	Normal	Normal
6	VI	R	Normal	Normal	Normal	Episcleral congestion
		L	Normal	Congested	Staphyloma	Episcleral congestion

Table 6: General and clinical appearance of the right eye (R) and left eye (L) of the cases studied (Contd...)

SL No.	Dog No.	Eye	Eyelids and adnexa	Conjunctiva	Cornea	Sclera
7	VII	R	Normal	Normal	Normal	Normal
		L	Normal	Congested	Staphyloma with corneal opacity	Normal
8	VIII	R	Normal	Congested	Descemetocele with corneal opacity	Normal
		L	Normal	Normal	Normal	Normal
9	IX	R	Normal	Normal	Normal	Normal
		L	Normal	Congested	Superficial ulcer with corneal opacity	Normal
10	X	R	Normal	Inflamed	Stromal ulcer with corneal opacity	Normal
		L	Normal	Congested	Stromal ulcer with corneal opacity	Normal
11	XI	R	Normal	Normal	Normal	Normal
		L	Normal	Congested	Stromal ulcer with corneal opacity	Normal
12	XII	R	Normal	Inflamed	Descemetocele with mild opacity	Normal
		L	Normal	Normal	Normal	Normal

Table 6: General and clinical appearance of the right eye (R) and left eye (L) of the cases studied (Contd...)

SL No.	Dog No.	Eye	Eyelids and adnexa	Conjunctiva	Cornea	Sclera
13	XIII	R	Normal	Congested	Normal	Episcleral congestion
		L	Normal	Congested	Staphyloma	Normal
14	XIV	R	Normal	Normal	Normal	Normal
		L	Normal	Congested	Stromal ulcer with opacity	Normal
15	XV	R	Normal	Normal	Normal	Normal
		L	Normal	Congested	Staphyloma	Normal
16	XVI	R	Normal	Normal	Pigmented	Normal
		L	Nasal fold trichiasis	Congested	Pigmented	Normal
17	XVII	R	Normal	Congested	Pigmented	Episcleral congestion
		L	Lower lid entropion	Congested	Pigmented	Normal
18	XVIII	R	Normal	Normal	Normal	Episcleral congestion
		L	Normal	Normal	Normal	Episcleral congestion

Table 6: General and clinical appearance of the right eye (R) and left eye (L) of the cases studied (Contd...)

SL No.	Dog No.	Eye	Eyelids and adnexa	Conjunctiva	Cornea	Sclera
19	XIX	R	Normal	Normal	Normal	Episcleral congestion
		L	Normal	Normal	Normal	Episcleral congestion
20	XX	R	Normal	Normal	Normal	Episcleral congestion
		L	Normal	Normal	Normal	Episcleral congestion
21	XXI	R	Normal	Normal	Normal	Episcleral congestion
		L	Normal	Normal	Normal	Episcleral congestion
22	XXII	R	Normal	Congested	Oedema Pigmented	Episcleral congestion
		L	Normal	Normal	Normal	Episcleral congestion

Table 7: Visual function of right eye (R) and left eye (L) in the dogs studied

Dog No.	Eye	Days of observation																			
		1 st day				10 th day				25 th day				40 th day				60 th day			
		P	C	M	PL	P	C	M	PL	P	C	M	PL	P	C	M	PL	P	C	M	PL
I	R	✓	✓	+	0	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
II	R	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	+	0	✓	✓	+	0	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
III	R	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	+	0	✓	✓	+	0	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
IV	R	✓	✓	+	0	✓	✓	+	0	✓	✓	+	0	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
V	R	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0
	L	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
VI	R	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	+	0	✓	✓	+	0	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
VII	R	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	+	0	✓	✓	+	0	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
VIII	R	✓	✓	+	0	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

Present (✓), Sluggish (+), Absent (-) and Could not be assessed due to corneal opacity/pigmentation (0)

Palpebral reflex (P), Corneal (C), Menace reflex (M) and Pupillary light reflex (PL)

Table 7: Visual function of right eye (R) and left eye (L) in the dogs studied (Contd...)

Dog No.	Eye	Days of observation																			
		1 st day				10 th day				25 th day				40 th day				60 th day			
		P	C	M	PL	P	C	M	PL	P	C	M	PL	P	C	M	PL	P	C	M	PL
IX	R	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√
	L	√	√	+	0	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√
X	R	√	√	+	0	√	√	+	0	√	√	√	√	√	√	-	0	√	√	-	0
	L	√	√	+	0	√	√	+	0	√	√	√	√	√	√	-	0	√	√	-	0
XI	R	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√
	L	√	√	+	0	√	√	+	0	√	√	√	√	√	√	√	√	√	√	√	√
XII	R	√	√	+	0	√	√	+	0	√	√	-	0	√	√	-	0	√	√	-	0
	L	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√
XIII	R	√	√	√	+	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√
	L	√	√	+	0	√	√	+	0	√	√	+	0	√	√	√	√	√	√	√	√
XIV	R	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√
	L	√	√	+	0	√	√	+	0	√	√	√	√	√	√	√	√	√	√	√	√
XV	R	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√	√
	L	√	√	-	0	√	√	-	0	√	√	-	0	√	√	-	0	√	√	-	0

Present (√), Sluggish (+), Absent (-) and Could not be assessed due to corneal opacity/pigmentation (0)

Palpebral reflex (P), Corneal (C), Menace reflex (M) and Pupillary light reflex (PL)

Table 7: Visual function of right eye (R) and left eye (L) in the dogs studied (Contd...)

Dog No.	Eye	Days of observation																			
		1 st day				10 th day				25 th day				40 th day				60 th day			
		P	C	M	PL	P	C	M	PL	P	C	M	PL	P	C	M	PL	P	C	M	PL
XVI	R	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0
	L	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0
XVII	R	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0
	L	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0	✓	✓	-	0
XVIII	R	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
XIX	R	✓	✓	✓	+	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	+	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
XX	R	✓	✓	✓	+	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	+	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
XXI	R	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
XXII	R	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	L	✓	✓	✓	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

Present (✓), Sluggish (+), Absent (-) and Could not be assessed (0)

Palpebral reflex (P), Corneal (C), Menace reflex (M) and Pupillary light reflex (PL)

Table 8: Degree of corneal clarity in the dogs with ulcerative keratitis studied

SL No.	Dog No.	Days of observation				
		1 st day	10 th day	25 th day	40 th day	60 th day
1	I	2+	4+	4+	4+	4+
2	II	2+	3+	3+	4+	4+
3	III	2+	1+	4+	4+	4+
4	IV	1+	1+	2+	3+	3+
5	V	1+	2+	3+	3+	4+
6	VI	2+	2+	3+	3+	3+
7	VII	1+	2+	3+	3+	3+
8	VIII	2+	3+	4+	4+	4+
9	IX	1+	4+	4+	4+	4+
10	X	1+/1+(R/L)	2+/2+(R/L)	4+/4+(R/L)	4+/4+(R/L)	4+/4+(R/L)
11	XI	1+	1+	3+	3+	4+
12	XII	1+	1+	2+	2+	2+
13	XIII	1+	1+	2+	2+	2+
14	XIV	1+	2+	4+	4+	4+
15	XV	1+	1+	2+	2+	1+/4+

Complete opacity (1+), Moderate opacity (2+), Hazy (3+) and Clear (4+)

R/L - Right eye/Left eye

Table 9: Corneal oedema in cases of ulcerative keratitis studied

SL No.	Dog No.	Days of observation				
		1 st day	10 th day	25 th day	40 th day	60 th day
1	I	+	-	-	-	-
2	II	+	+	-	-	-
3	III	+	+	-	-	-
4	IV	+	+	+	-	-
5	V	+	+	-	-	-
6	VI	+	+	-	-	-
7	VII	+	+	-	-	-
8	VIII	+	+	-	-	-
9	IX	+	-	-	-	-
10	X	++ (R/L)	++ (R/L)	-/- (R/L)	-/- (R/L)	-/- (R/L)
11	XI	+	+	-	-	-
12	XII	+	+	+	-	-
13	XIII	+	+	+	-	-
14	XIV	+	+	-	-	-
15	XV	+	+	+	-	-

Present (+) and Absent (-)

R/L - Right eye/Left eye

Table 10: Fluorescein dye retention in the ulcerative keratitis cases studied

SL No.	Dog No.	Days of observation				
		1 st day	10 th day	25 th day	40 th day	60 th day
1	I	+	-	-	-	-
2	II	+	+	-	-	-
3	III	+	+	-	-	-
4	IV	+	+	-	-	-
5	V	+	+	-	-	-
6	VI	+	+	-	-	-
7	VII	+	+	-	-	-
8	VIII	+	+	-	-	-
9	IX	+	-	-	-	-
10	X	++ (R/L)	++ (R/L)	-/- (R/L)	-/- (R/L)	-/- (R/L)
11	XI	+	+	-	-	-
12	XII	+	+	+	-	-
13	XIII	+	+	+	-	-
14	XIV	+	+	-	-	-
15	XV	+	+	+	-	-

Present (+) and Absent (-)

R/L - Right eye/Left eye

Table 11: Vascularisation of the cornea in cases of ulcerative and pigmentary keratitis studied

SL No.	Dog No.	Days of observation				
		1 st day	10 th day	25 th day	40 th day	60 th day
1	I	-	-	-	-	-
2	II	+	+	-	-	-
3	III	-	+	-	-	-
4	IV	-	+	+	-	-
5	V	+	+	-	-	-
6	VI	+	+	+	-	-
7	VII	-	+	-	-	-
8	VIII	-	+	-	-	-
9	IX	-	-	-	-	-
10	X	+/+ (R/L)	+/+ (R/L)	-/- (R/L)	-/- (R/L)	-/- (R/L)
11	XI	+	+	-	-	-
12	XII	-	+	+	-	-
13	XIII	+	+	+	+	-
14	XIV	+	+	-	-	-
15	XV	+	+	+	+	-
16	XVI	+	+	-	-	-
17	XVII	-	-	-	-	-

Present (+) and Absent (-)

R/L - Right eye/Left eye

Table 12: Conjunctival changes in cases of ulcerative and pigmentary keratitis studied

SL No.	Dog No.	Days of observation				
		1 st day	10 th day	25 th day	40 th day	60 th day
1	I	+	-	-	-	-
2	II	+	+	-	-	-
3	III	+	+	-	-	-
4	IV	+	+	+	-	-
5	V	+	+	-	-	-
6	VI	+	+	+	-	-
7	VII	+	+	-	-	-
8	VIII	+	+	-	-	-
9	IX	+	-	-	-	-
10	X	+/+ (R/L)	+/+ (R/L)	-/- (R/L)	-/- (R/L)	-/- (R/L)
11	XI	+	+	-	-	-
12	XII	+	+	+	-	-
13	XIII	+	+	+	-	-
14	XIV	+	+	-	-	-
15	XV	+	+	+	-	-
16	XVI	+	-	-	-	-
17	XVII	+	-	-	-	-

Present (+) and Absent (-)

R/L - Right eye/Left eye

Table 13: Pigmentation of cornea in cases of ulcerative and pigmentary keratitis studied

SL No.	Dog No.	Days of observation				
		1 st day	10 th day	25 th day	40 th day	60 th day
1	I	-	-	+	+	+
2	II	-	-	+	+	+
3	III	-	-	+	+	+
4	IV	-	-	+	+	+
5	V	-	-	+	+	+
6	VI	-	-	-	-	+
7	VII	-	-	+	+	+
8	VIII	-	-	-	-	-
9	IX	-	-	-	-	-
10	X	-/- (R/L)	-/- (R/L)	+/+ (R/L)	+/+ (R/L)	+/+ (R/L)
11	XI	-	-	+	+	+
12	XII	-	-	+	+	+
13	XIII	-	-	+	+	+
14	XIV	-	-	+	-	+
15	XV	+	+	+	+	+
16	XVI	+	+	+	+	+
17	XVII	+	+	+	+	+

Present (+) and Absent (-)

R/L - Right eye/Left eye

Table 14: Schirmer tear test readings of right eye (R) and left eye (L) in the dogs studied (millimeters/minute)

SL No.	Dog No.	Days of observation									
		1 st day		10 th day		25 st day		40 th day		60 th day	
		R	L	R	L	R	L	R	L	R	L
1	I	22	17	20	18	19	18	19	17	18	18
2	II	16	20	17	18	17	17	18	17	16	17
3	III	16	22	17	19	16	18	16	17	18	17
4	IV	24	19	21	18	19	19	18	19	17	16
5	V	19	20	18	17	16	17	18	19	16	18
6	VI	18	19	18	20	17	18	17	18	18	18
7	VII	18	25	18	25	18	20	17	20	17	18
8	VIII	20	18	18	18	17	18	17	16	18	16
9	IX	17	22	17	18	17	16	16	17	16	17
10	X	3	4	4	3	5	3	9	11	13	14
11	XI	16	21	16	18	16	17	16	17	16	16
12	XII	16	17	16	18	16	14	15	16	14	15

Table 14: Schirmer tear test readings of right eye (R) and left eye (L) in the cases studied (Contd.)

SL No.	Dog No.	Days of observation									
		1 st day		10 th day		25 st day		40 th day		60 th day	
		R	L	R	L	R	L	R	L	R	L
13	XIII	18	22	17	20	18	18	17	16	16	18
14	XIV	16	20	17	20	16	17	16	18	16	17
15	XV	16	19	17	21	16	20	16	17	15	17
16	XVI	11	10	12	13	11	15	11	17	12	17
17	XVII	12	11	13	14	12	16	13	16	15	18
18	XVIII	13	15	11	16	14	16	16	16	15	16
19	XIX	18	17	19	18	18	16	17	17	18	16
20	XX	17	16	18	17	19	16	18	16	17	16
21	XXI	16	15	18	17	17	15	16	18	18	16
22	XXII	17	16	18	16	17	16	16	15	18	17
Mean ± SE		16.32 ± 0.88	17.50 ± 0.99	16.37 ± 0.77	17.36 ± 0.86	15.95 ± 0.68	16.36 ± 0.72	16.00 ± 0.50	16.82 ± 0.38	16.23 ± 0.37	16.73 ± 0.23

Table 15: Intraocular pressure of the eyes in the cases with glaucoma (mm of Hg)

SL No.		1	2	3	4	5	6	7	8	9	10	Mean \pm SE	
Case No.	Eye	I	II	IV	VI	XII	XVII	XIX	XX	XXI	XXII		
Days of observation	1st day	R	×	46.3	×	52.3	46.3	69.9	69.9	58.7	55.4	49.4	56.03 \pm 3.38
		L	43.4	×	55.4	×	×	28.4	62.2	62.2	52.3	43.4	49.61 \pm 4.59
	10th day	R	×	31.9	×	33.9	30.1	36	38.3	31.9	33.9	31.9	33.49 \pm 0.93
		L	33.9	×	36	×	×	28.4	33.9	36	33.9	33.9	33.71 \pm 0.96
	25th day	R	31.9	30.1	×	31.9	31.9	33.9	36	31.9	31.9	30.1	32.18 \pm 0.61
		L	31.9	×	31.9	×	×	26.9	33.9	33.9	33.9	31.9	32.04 \pm 0.94
	40th day	R	31.9	30.1	×	33.9	30.1	33.9	31.9	31.9	30.1	28.4	31.36 \pm 0.62
		L	33.9	×	31.9	×	×	26.9	33.9	33.9	31.9	30.1	31.78 \pm 0.98
	60th day	R	30.1	28.4	×	31.9	30.1	33.9	31.9	31.9	31.9	28.4	30.94 \pm 0.61
		L	30.1	×	30.1	×	×	28.4	33.9	31.9	30.1	28.4	30.41 \pm 0.74

Right eye (R) and Left eye (L)

×Could not be measured due to corneal ulcer

Table 16: Mean haematological values in the cases studied

Parameters	Days of observation					
	1 st day	10 th day	25 th day	40 th day	60 th day	
Hb (g/dl)	12.70±0.33	13.26 ±0.25	13.58±0.30	13.12±0.29	13.12±0.3	
VPRC (%)	37.14±1.01	39.09±0.81	40.41±0.93	39.14±0.88	39.09±0.93	
ESR (mm/hr)	6.36±0.28	6.68±0.34	6.86±0.42	7.18±0.38	7.23±0.38	
TLC (×10³/cmm)	11.83±0.45	11.30±0.37	11.73±0.36	11.91±0.52	11.24±0.36	
DLC (%)	N	74.33±1.98	73.77±1.76	73.33±1.62	72.33±2.56	73.33±1.48
	L	27.76±1.44	26.52±1.26	26.12±1.64	26.32±1.22	26.0±1.21
	E	0.50±0.22	0.50±0.33	0.33±0.21	0.66±0.33	0.66±0.33
	M	0.16±4.47	0.16±4.51	0.16±4.47	0.16±4.63	0.16±4.58

Table 17: Random blood glucose level (RBGL) in the cases studied

SL No.	Dog No.	RBGL (mg/dl)	SL No.	Dog No.	RBGL (mg/dl)
1	I	82	13	XIII	104
2	II	106	14	XIV	91
3	III	93	15	XV	101
4	IV	108	16	XVI	86
5	V	102	17	XVII	110
6	VI	83	18	XVIII	87
7	VII	91	19	XIX	116
8	VIII	104	20	XX	97
9	IX	85	21	XXI	82
10	X	108	22	XXII	106
11	XI	80	Mean ± SE		97.00 ± 2.41
12	XII	112			

Table 18: Antibiogram of the corneal swabs in cases of ulcerative keratitis studied

Dog No.	Organism cultured	Sensitive to	Resistant to
I	No growth		
II	Gram negative bacilli	Ceftriaxone +++ Cefotaxim ++ Gentamicin +	Ciprofloxacin Amoxicillin Penicillin-G
III	Gram positive cocci	Ceftriaxone +++ Chloramphenicol ++ Ciprofloxacin ++	Amoxicillin Cefotaxim
IV	Gram negative bacilli	Ciprofloxacin ++ Gentamicin ++ Ceftriaxone +	Sulphadiazine Oxytetracycline
V	Gram positive cocci	Ciprofloxacin +++ Gentamicin +++ Cefotaxim ++	Sulphadiazine
VI	No growth		
VII	Gram positive cocci	Ciprofloxacin +++ Gentamicin ++	Ceftriaxone Cefotaxim Amoxicillin
VIII	No growth		
IX	No growth		

Table 18: Antibigram of the corneal swabs in cases of ulcerative keratitis studied (Contd.)

Dog No.	Organism cultured	Sensitive to	Resistant to
X	Gram positive cocci Gram negative bacilli	Ceftriaxone +++ Gentamicin + Amoxicillin +	Ciprofloxacin Cefotaxim Penicillin-G
XI	Gram positive cocci	Ciprofloxacin +++ Gentamicin +++	Amoxicillin Cefotaxim Penicillin-G
XII	Gram positive cocci	Gentamicin +++ Ciprofloxacin ++ Ceftriaxone ++	Amoxicillin Cefotaxim
XIII	Gram positive bacilli	Ciprofloxacin ++ Cefotaxim ++	Gentamicin Amoxicillin Chloramphenicol
XIV	Gram positive cocci	Gentamicin +++ Amoxicillin +++ Ciprofloxacin ++	Tetracycline
XV	Gram positive cocci	Ciprofloxacin +++ Amoxicillin ++ Gentamicin +	Ceftriaxone

Table 19: Exfoliative cytology of lacrimal smear of dogs with ulcerative keratitis

Dog No.		I	II	III	IV	V	VI	VII	VIII	IX	X	XI	XII	XIII	XIV	XV	
Days of observation and Exfoliative cell types	1 st	E	+	++	++	++	+++	+++	+++	+	+	++	++	+++	++	++	+++
		A	+	+	+	+	+++	++	++	+	+	++	++	++	+++	+	+++
		P	-	+	-	+	+++	+++	+++	-	-	+	+	+	+++	+	+++
	10 th	E	-	+	+	++	+	+	-	+	-	-	+	++	+	+	+
		A	-	++	+	+	++	++	++	++	-	++	++	++	+++	++	+++
		P	-	+	-	++	++	++	++	-	-	+	+	++	++	+	++
	25 th	E	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-
		A	-	+	+	++	+	+	+	+	-	+	+	++	++	+	++
		P	-	-	-	+	-	+	-	-	-	-	-	+	+	-	+
	40 th	E	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
		A	-	-	-	+	-	+	-	-	-	-	-	+	++	-	++
		P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	60 th	E	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-
		A	-	+	-	-	-	-	-	-	+	-	-	-	-	-	+
		P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Epithelial cells (E), Anuclear keratinized cells (A) and Polymorphonuclear cells (P)

Present (+) and Absent (-)

Table 20: Complications encountered in the cases studied

SL No.	Dog No.	Complications encountered
1	IV	Partial disruption of temporary tarsorrhaphy sutures and progression of ulcer to small staphyloma.
2	V	Anterior synechiae and phthisis bulbi
3	X	Bilateral pigmentation of cornea
4	XI	Dense pigmentation of cornea
5	XII	Partial disruption of temporary tarsorrhaphy sutures, descemetocoele progressed to small staphyloma, hypopion, anterior synechiae and phthisis bulbi
6	XIII	Partial disruption of temporary tarsorrhaphy sutures, dislodgement of third eyelid flap and deep pigmentation of cornea with granulation
7	XV	Enophthalmos with deeply pigmented granulation tissue
8	XVI	Complete bilateral pigmentation of cornea persisted
9	XVII	Complete bilateral pigmentation of cornea persisted
10	XVIII	Decrease in tear production after removal of third eyelid gland

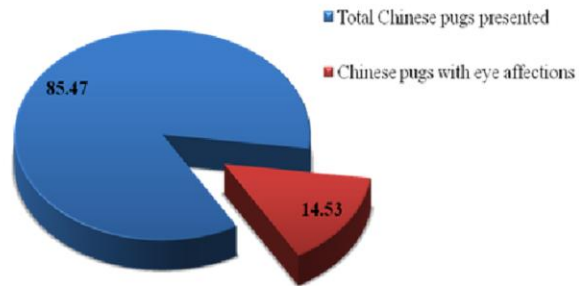


Fig. 1: Incidence of eye affections in Chinese pugs

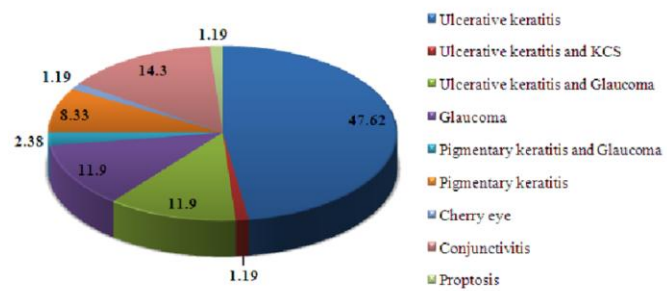


Fig. 2: Distribution of different conditions in Chinese pugs

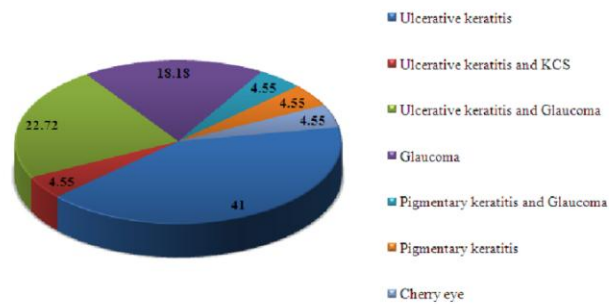


Fig. 3: Distribution of different conditions in Chinese pugs selected for the study

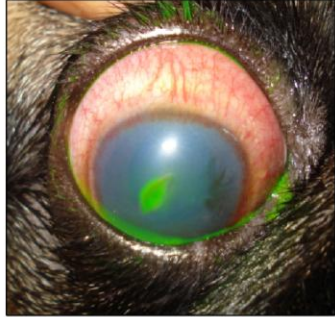


Fig. 4: Dog No.I Superficial ulcer (Day 1)



Fig. 5: Dog No. I Superficial ulcer (Day 10)

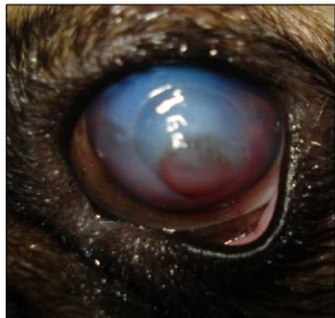


Fig. 6: Dog No.II Stromal ulcer (Day 1)



Fig. 7: Dog No. II Collagen sheet sutured on to bulbar conjunctiva (Day 3)



Fig. 8: Dog No. II Completely dissolved collagen sheet (Day 10)



Fig. 9: Dog No.II Completely cleared cornea with mild opacity (Day 60)



Fig. 10: Dog No. III Descemetocoele (Day 1)

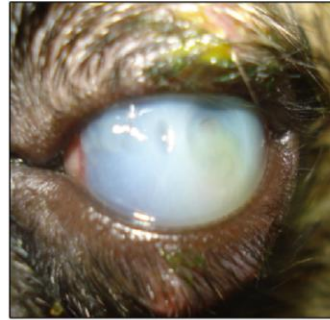


Fig. 11: Dog No. III Corneal oedema (Day 10)



Fig. 12: Dog No. III Completely cleared cornea with pigmentation (Day 60)



Fig. 13: Dog No. VI Staphyloma (Day 1)

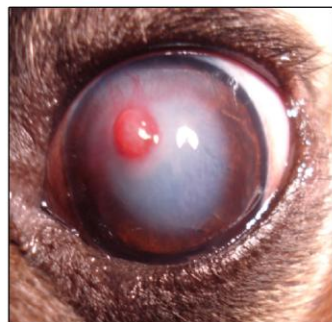


Fig. 14: Dog No. VI Mild corneal oedema (Day 10)

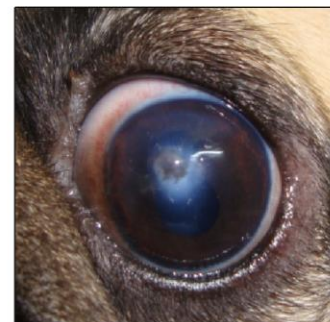


Fig. 15: Dog No. VI Mild opacity of central cornea (Day 60)



Fig. 16: Dog No.IV Melting stromal ulcer (Day 1)



Fig. 17: Dog No.IV Suture disruption with progression to staphyloma (Day 10)



Fig. 18: Dog No. IV Healing cornea with vascularisation and pigmentation (Day 25)



Fig.19: Dog No. IV Cleared cornea with pigmentation (Day 60)



Fig. 20: Dog No. V Necrosed staphyloma (Day 1)

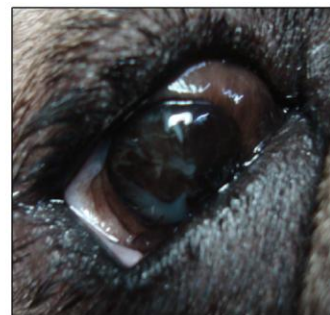


Fig. 21: Dog No. V Phthisis bulbi (Day 60)

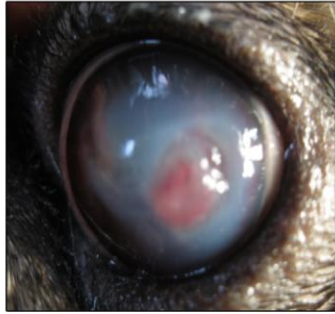


Fig. 22: Dog No. XI Stromal ulcer (Day 1)

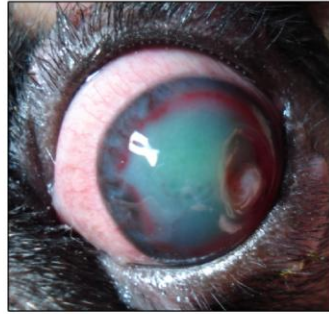


Fig. 23: Dog No. XI Vascularisation of cornea (Day 10)



Fig. 24: Dog No. XI Cleared cornea with pigmentation (Day 60)

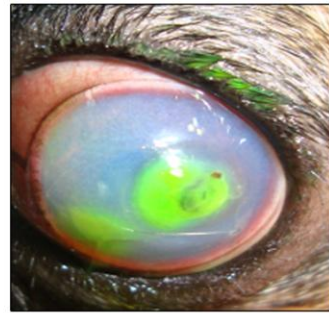


Fig. 25: Dog No. XIV Stromal ulcer (Day 1)



Fig. 26: Dog No. XIV Vascularisation of cornea (Day 10)

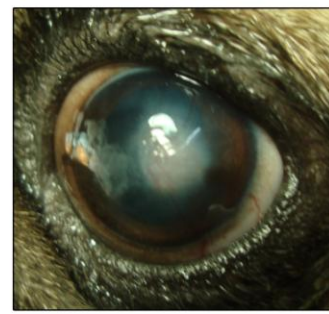


Fig. No. 27: Dog No. XIV Reduction in depth of corneal ulcer (Day 25)



Fig. 28: Dog No. VIII Descemetocoele (Day 1)



Fig. 29: Dog No. VIII Completely cleared cornea with a recurrent superficial ulcer (Day 60)

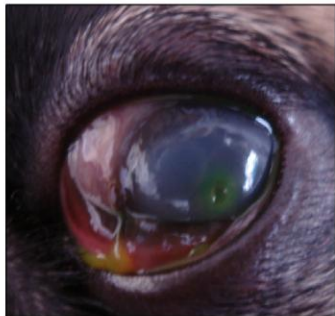


Fig. 30: Dog No. X Stromal ulcer in left eye (Day 1)

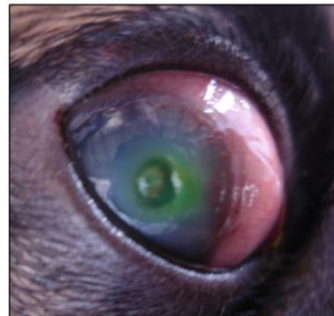


Fig. 31: Dog No. X Stromal ulcer with conjunctival hyperaemia of right eye (Day 1)



Fig. 32: Dog No. X Complete corneal pigmentation of left eye (Day 60)



Fig. 33: Dog No. X Complete corneal pigmentation of right eye (Day 60)



Fig. 34: Dog No. IX Superficial ulcer (Day 1)



Fig. 35: Dog No. IX Cleared cornea with a central scar (Day 10)



Fig. 36: Dog No. XII Descemetocoele (Day 1)



Fig. 37: Dog No. XII Fluorescein dye retention (Day 25)

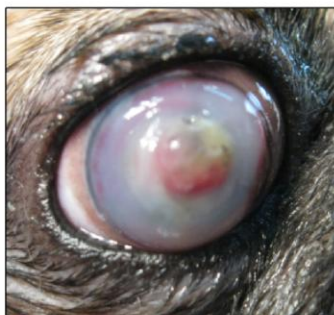


Fig. 38: Dog No. XII Absence of fluorescein dye retention with anterior synechiae (Day 40)

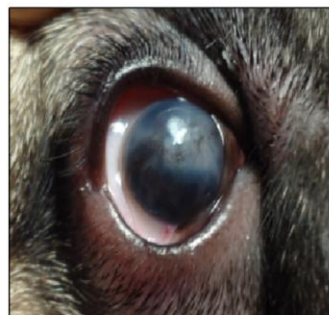


Fig. 39: Dog No. XII Phthisis bulbi (Day 60)



Fig. 40: Dog No. XIII Necrosed staphyloma (Day 1)



Fig. 41: Dog No. XIII Fluorescein dye retention (Day 10)

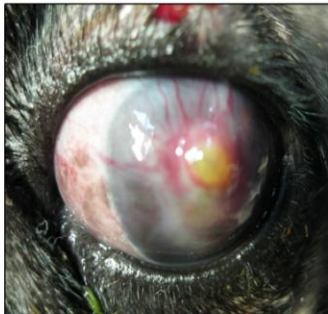


Fig. 42: Dog No. XIII Mild fluorescein dye retention with vascularisation (Day 25)



Fig. 43: Dog No. XIII Persistence of vascularisation with central opacity (Day 40)



Fig. 44: Dog No. XV Fluorescein dye retention with vascularisation (Day 10)



Fig. 45: Dog No. XV Protruded Descemet's membrane with reformed anterior chamber (Day 25)



Fig. 46: Dog No. XV Clearing of corneal defect with granulation tissue (Day 40)



Fig. 47: Dog No. XV Corneal clearing on progression (Day 60)



Fig. 48: Dog No. VII Staphyloma (Day 1)



Fig. 49: Dog No. VII Vascularisation of cornea (Day 10)

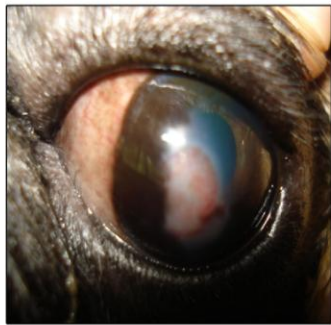


Fig. 50: Dog No. VII Pigmentation of cornea (Day 25)

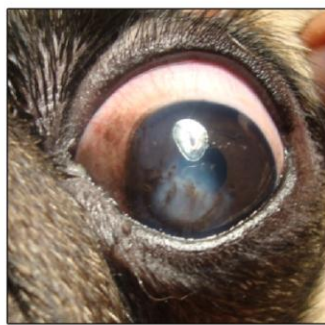


Fig. 51: Dog No. VII Completely cleared cornea with mild opacity and pigmentation (Day 40)

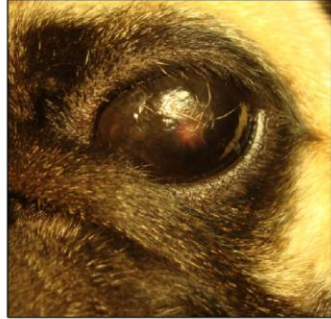


Fig. 52: Dog No. XVI Pigmentary keratitis with nasal fold trichiasis (Day 1)



Fig. 53: Dog No. XVI Postoperative appearance (Day 3)



Fig. 54: Dog No. XVI Postoperative appearance after suture removal (Day 10)

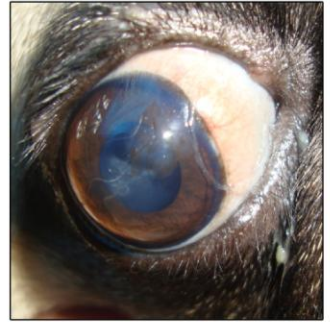


Fig. 55: Dog No. XXII Corneal oedema and pigmentation (Day 1)



Fig. 56: Dog No. XVIII Prolapse of membrana nictitans gland (Day 1)

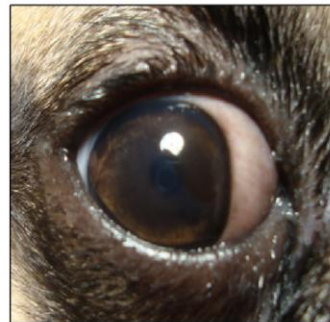


Fig. 57: Dog No. XVIII Postoperative appearance (Day 10)



Fig. 58: Dog No. XI Postoperative appearance after temporary tarsorrhaphy (Day 3)



Fig. 59: Dog No. XVII Bilateral pigmented keratitis with exophthalmos (Day 1)



Fig. 60: Dog No. XVII Preoperative appearance of eye before lateral lid shortening and correction of medial entropion (Day 3)

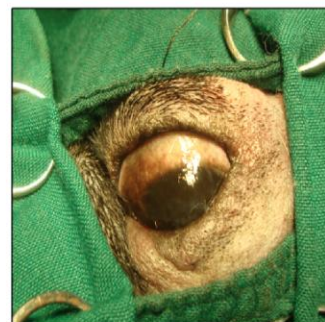


Fig. 61: Dog No. XVII Preoperative draping of surgical site (Day 3)



Fig. 62: Dog No. XVII Postoperative appearance after lateral lid shortening and correction of medial entropion (Day 3)

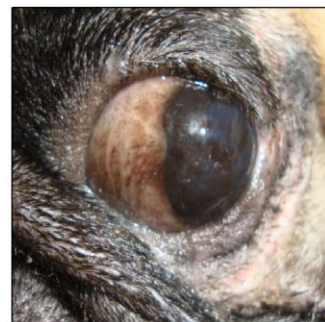


Fig. 63: Dog No. XVII Postoperative appearance (Day 10)

Discussion

5. DISCUSSION

5.1 SELECTION OF CASE

The study was conducted in Chinese pug dogs presented to the surgery outpatient unit of University Veterinary Hospital, Mannuthy and Kokkalai with eye affections. All the dogs were thoroughly examined and were categorised based on the disease condition for which they were presented or diagnosed. The dogs were subjected to detailed clinical and ophthalmic examination.

The treatment was selected depending upon the type and severity of the condition, based on the observations made on the first day of presentation. The conditions which responded to the medical management were treated medically and were observed for a period of 60 days. Those conditions which were refractory to medical management or required immediate surgical intervention were subjected to surgical treatment.

Among 578 Chinese pug dogs presented during the period of study from June 2011 to May 2012, 84 dogs had ocular affections. The increased incidence of eye affections in Chinese pugs was also observed by Whitley *et al.* (1991) and Bharathi *et al.* (2011). The higher incidence of ocular problems in short headed dogs was also observed by Krawitz (1963).

Out of the 84 Chinese pug dogs presented with ocular problems, 40 dogs were diagnosed to have ulcerative keratitis, 10 had glaucoma, 10 had concurrent glaucoma and ulcerative keratitis, 7 dogs had pigmentary keratitis, 12 dogs had conjunctivitis, one was presented with proptosis, one had cherry eye, 2 dogs had concurrent pigmentary keratitis and glaucoma and one had concurrent ulcerative keratitis and keratoconjunctivitis sicca.

In total, 51 dogs were presented with ulcerative keratitis accounting 60.71% of the total cases presented. Among them, 10 dogs had concurrent glaucoma and one had concurrent KCS. The increased incidence of ulcerative keratitis in Chinese pugs was also reported by Tolar *et al.* (2006) and Chinchu (2010). This increased incidence can be attributed to the predisposition of this brachycephalic breed, due to the relative exophthalmos (Renwick, 2007) in addition to inherited corneal insufficiency, poor corneal reflex and lack of protective eye consciousness (Startup, 1984), shallow orbit and large palpebral fissure (Bedford, 1987), globe prominence (Wolfer and Grahn, 1994) and lagophthalmos (Moore, 2003 and Williams, 2008). Also, the rise in intraocular pressure (Resmi, 2008 and Chinchu, 2010), KCS (Whitley *et al.*, 1991 and Kaswan *et al.*, 1995), central area of tear film lipid deficiency (Williams, 2008), poor tear film distribution, presence of foreign materials and lid abnormalities like entropion and nasal fold trichiasis (Renwick, 2007) led to ulcerations. The excessive corneal exposure (Kaswan *et al.*, 1995) and exposure keratopathy (Hartley, 2010) in Chinese pugs due to the absence of effective blinking also exaggerated the severity of corneal lesions and resulted in corneal ulcerations (Carrington *et al.*, 1989 and Renwick, 2007).

The incidence of glaucoma in Chinese pugs in the present study was found to be 26.19%. Even though, an increased occurrence of this condition in Chinese pugs was reported by Gelatt and Mackay (2004a), Resmi (2008) and Bharathi *et al.* (2011), literatures on breed predisposition were not found. About 45.45% of the dogs with glaucoma had concurrent ulcerative keratitis and 9.09% had concurrent pigmentary keratitis. They were 11.9% and 2.38%, respectively, of total Chinese pugs presented. Increased intraocular pressure in Chinese pugs presented with corneal affections was previously reported by Resmi (2008), Chinchu (2010) and Venugopal (2011).

Pigmentary keratitis accounted for about 10.71% of the total Chinese pugs presented with eye affections. The congenital predisposition of this breed to corneal pigment deposition (Bedford, 1982 and Whitley *et al.*, 1995), the lid

abnormalities like entropion and nasal fold trichiasis along with exposure due to lagophthalmos, poor tear film distribution and presence of foreign materials resulted in corneal pigmentation (Peiffer *et al.*, 1987 and Renwick, 2007). The central area of tear film deficiency (Williams, 2008) and the absence of an effective blink (Kaswan *et al.*, 1995) due to lagophthalmos greatly exaggerated the severity of corneal pigmentation. A variable degree of medial pigmentary keratitis was noticed by Carrington *et al.* (1989) in all the brachycephalic dogs presented with tear film deficiency.

Even though Chinese pugs are predisposed to traumatic proptosis, only one dog was presented with this condition. The prominent eye along with the shallow orbit and a large palpebral fissure predisposes brachycephalic breeds to complete prolapse of the globe through the palpebral fissure on orbital trauma (Bedford, 1987 and Tolar *et al.*, 2006).

Only one dog was presented with KCS. This dog had concurrent bilateral corneal ulceration. The over-presentation of Chinese pugs with eye affections due to KCS was reported by Whitley *et al.* (1991). The breed predisposition to KCS was mentioned by Kaswan *et al.* (1995).

The percentage of dogs presented with conjunctivitis was 14.29% and that with cherry eye was 1.19%. Also, the brachycephalic breeds are predisposed to cherry eye (Herrera, 2005)

Among the cases presented with ocular affections and subjected to treatment, cases of 22 dogs, the post treatment observations of which were available for a period of 60 days, were selected for the study. The dogs were numbered as Dog No. I, II, III, IV, V, VI, VII, VIII, IX, X, XI, XII, XIII, XIV, XV, XVI, XVII, XVIII, XIX, XX, XXI and XXII.

The Dogs I to XV had ulcerative keratitis, Dogs XVI and XVII had pigmentary keratitis, Dog XVIII was presented with cherry eye, Dogs XIX to XXII were diagnosed to have glaucoma. The Dogs I, II, IV, VI and XIII with

ulcerative keratitis were diagnosed to have glaucoma on contra lateral eyes. The Dog XVII was having concurrent pigmentary keratitis and glaucoma (right eye) and the Dog X was having concurrent bilateral ulcerative keratitis and KCS.

Post treatment observations of the selected cases were made on the 10th, 25th, 40th and 60th day to study the effectiveness of treatment adopted.

5.2 MEDICAL MANAGEMENT

5.2.1 Ulcerative keratitis

The Dogs I and IX with superficial keratitis and the Dog X with stromal ulcerative keratitis were subjected to medical treatment. The medical treatment for ulcerative keratitis included topical instillation of antibiotics, anti-inflammatory agents and cycloplegic agents instilled four times daily at an interval of three to four hours. In addition to the medications mentioned above, oral antibiotics for seven days were also advised for Dog X. Topical tear substitutes and oral supplementation of multivitamins were advised in all the cases. The treatment was similar to the course that was suggested by Bedford (1987) and Wilkie and Whittaker (1997). All the animals under medical treatment tolerated the medications and responded to the treatments given.

The topical antibiotic selected initially was ciprofloxacin and was continued in all the cases as culture and sensitivity and clinical response suggested the same. Even though superficial ulcers were not infected, topical broad spectrum antibiotic, ciprofloxacin helped to prevent secondary, opportunistic bacterial infection as the epithelial integrity was compromised (Ollivier, 2003). Topical fluoroquinolones were effective in treating infected corneal ulcers especially against gram negative organisms (Townsend *et al.*, 2009). The resistance to fluoroquinolone antimicrobials was minimal among all isolates from dogs with bacterial keratitis. Ciprofloxacin was still an effective antibiotic in the treatment for bacterial keratitis in dogs especially against *Staphylococcus* spp., *Streptococcus* spp. and *Pseudomonas* spp. (Tolar *et al.*, 2006). The in vitro studies

in the *Pseudomonas aeruginosa* isolates in dogs with ulcerative keratitis conducted by Ledbetter *et al.* (2007) found a hundred per cent susceptibility to ciprofloxacin. According to Hendrix *et al.* (2001), topical gentamicin affected corneal healing as it inhibited epithelial migration and exhibited cytopathic effects. Also, the mean tear concentration of ciprofloxacin remained above the MIC₉₀ levels for most pathogenic bacteria for 6 hours in brachycephalic dogs (Hendrix and Cox, 2008).

Topical flurbiprofen was used as the anti-inflammatory agent since corticosteroids were contraindicated in corneal ulcers (Giuliano, 2004). The topical NSAIDs can be safely administered in eyes with concurrent uveitis and ulcerative keratitis (Gionfriddo, 1995d). Flurbiprofen counteracted the clinically manifested anterior uveitis secondary to ulcerative keratitis due to trigeminal nerve stimulation and secondary axonal stimulation of inflammatory mediators (Wilkie and Whittaker, 1997). According to Giuliano (2004), topical NSAIDs suppressed ocular inflammation and hence remain as an efficient therapeutic choice for treating ulcerokeratouveitis. Also, topical NSAIDs have the ability to suppress prostaglandin-mediated anterior uveitis (Gilmour and Lehenbauer, 2009). Flurbiprofen (0.3%) was effective as it had good intraocular permeability, minimal significant intraocular metabolism and potency of inhibition of cyclooxygenase (Munro, 2001 and Pirie *et al.*, 2011). Use of corticosteroid was preferred only after the clearing of cornea and development of healthy epithelium (Bath and Dua, 2006b).

The use of topical analgesic and mydriatic agent, tropicamide helped to improve animal's comfort as high intervention of cornea caused substantial postoperative pain and thereby the tendency to self trauma (Collins *et al.*, 1995). Topical mydriatic drugs controlled the ciliary body spasm and helped to prevent synechiae formation. The rapid and short duration of action of tropicamide enabled its use in dogs with concurrent glaucoma also (Mandell and Holt, 2005). The importance of mydriatic-cycloplegic therapy was also pointed out by Wilkie and Whittaker (1997) and Wang *et al.* (2008).

Administration of oral antibiotic for seven days for Dog X at the rate of 22 mg/kg body weight twice daily was effective in controlling the infection. The use of systemic antibiotics in the treatment of severe ocular infections was suggested by Bedford (1987).

Multivitamins were supplemented in all the cases of ulcerative keratitis in order to promote corneal epithelial healing. In a study by Martin (1971), corneal epithelial healing in dogs treated with vitamin A supplementation healed faster than the group treated with antibiotic alone. Treatment with vitamin A was beneficial especially in corneal erosion syndrome and in cases of indolent ulcers, vitamin C and E proved effective (Startup, 1984).

Supportive care of the cornea with false tear preparation or tear substitutes for all the dogs with relative exophthalmos helped to control corneal drying and enabled protection against corneal desiccation and exposure keratopathy (Wilkie and Whittaker, 1997 and Hartley, 2010). Topical 0.5% carboxy methyl cellulose sodium substituted the preocular tear film deficiency and was found satisfactory in providing moisture and lubrication to the cornea and conjunctiva and preventing bacterial invasion of these structures (Townsend, 2007).

5.2.2 Glaucoma

The Dogs XIX, XX, XXI and XXII diagnosed to have bilateral glaucoma, Dogs I, II, IV, VI and XIII with ulcerative keratitis of contra lateral eye, and the right eye of the Dog XVII with concurrent pigmentary keratitis diagnosed to have glaucoma, were treated with topical carbonic anhydrase inhibitors (0.5% dorzolamide) and β blockers (0.2% timolol maleate), instilled twice daily. Even though a significant decrease in IOP was noticed with the use of either 2% dorzolamide (Gelatt and Mackay, 2001) or timolol maleate (Maehara *et al.*, 2004) instilled twice or thrice daily, a fixed combination of 0.5% timolol maleate and 2% dorzolamide twice daily was found efficacious at reducing IOP, compared to

either timolol or dorzolamide alone (Plummer *et al.*, 2006). The topical application of a combination of two or more anti-glaucomal drugs was also suggested by Smith *et al.* (2010) to achieve the desired degree of IOP reduction. Carbonic anhydrase enzyme inhibitors lowered the IOP by reducing the production of bicarbonate ions from water and carbon dioxide in epithelium of ciliary body thereby decreasing the synthesis of aqueous humor (Willis *et al.*, 2002). Timolol maleate inhibited the β -adrenergic fibers on canine iris sphincter muscle and reduced the IOP by decreasing production of aqueous humor and also by increasing the uveoscleral outflow (Maehara *et al.*, 2004).

In Dogs I, II, IV, VI and XIII with ulcerative keratitis of contra lateral eye, the left eye of Dogs I and IV and the right eye of Dogs II, VI and XIII, diagnosed to have glaucoma were also subjected to treatment with anti-glaucoma drugs. In these dogs, antiglaucoma drugs could not be applied on the ulcerated eye. But, with topical administration of 0.5% timolol maleate at 12 hour interval, reduction of IOP could be achieved in both treated and contra lateral eyes due to its 'systemic uptake' and 'cross over effect' (Wilkie and Latimer, 1991).

All the dogs in the study showed considerable reduction in IOP to a safer level during the observation period. Topical therapy with anti-glaucoma drugs alone maintained the IOP within safer levels for a long period (Hasegawa *et al.*, 2001). Significant side effects of clinical importance were not noticed with topical carbonic anhydrase inhibitors (Woerd, 2001) and β -blockers (Wilkie and Latimer, 1991 and Palmer *et al.*, 2008) during the period of treatment.

Topical administration of 0.5% carboxy methyl cellulose sodium helped to prevent corneal drying and exposure keratopathy due to buphthalmos (Hartley, 2010).

5.2.3 Keratoconjunctivitis sicca

Topical instillation of lacrimomimetics or tear substitute (0.5% carboxy methyl cellulose sodium) two to three times daily was advised on the first day for the Dog X with concurrent bilateral ulcerative keratitis, diagnosed to have KCS. The use of this methyl cellulose based preparations with aqueous base more similar to natural tears helped to improve lubrication and provided comfort until sufficient tear production was attained (Townsend, 2007). Lacrimostimulants (0.1% cyclosporine) two to three times daily was advised from 25th day of observation, when the corneal ulcer was found to be healed. The use of cyclosporine helped to stimulate tear production and it was suggested for the treatment of corneal ulceration associated with KCS by Kim *et al.* (2009). Rise in the tear production with the use of cyclosporine therapy characterized by the decrease in number of bacterial isolates from KCS affected eyes was observed by Whitley (2000). This specific immunomodulator exerted its beneficial lacrimogenic effects by its selective T-helper lymphocyte suppression (Townsend, 2007 and Williams, 2008). The medications were also found effective for Dog XVIII, with cherry eye, treated for iatrogenic KCS that was noticed by the seventh postoperative day (Gionfriddo, 1995b and Hartley *et al.* 2006).

5.2.4 Pigmentary keratitis

The right eye of the Dog XVII with concurrent glaucoma was subjected to medical management. The treatment included topical instillation of antibiotic (ciprofloxacin), immunosuppressive agents (0.1% cyclosporine) and tear substitutes (0.5% carboxymethyl cellulose sodium). The treatment for the left eye of Dogs XVI and XVII was started only from the 10th day of observation, after the suture removal, for their respective eyelid and adnexal surgeries in order to avoid the immunosuppressive effects of cyclosporine (Williams, 2008). The therapeutic strategy helped to control the ocular infection and inflammation and to substitute the tear film deficiency noticed in these dogs (Kaswan *et al.*, 1995). According to

Wilkie and Whittaker (1997), corneal pigmentation could be better managed through prevention and treatment of the inciting causes like KCS, lagophthalmos and lid abnormalities rather than keratectomy. Also, resolution of corneal vascularization and pigmentation usually take place gradually over a period of three to 12 months or more (Kaswan *et al.*, 1995). But in these dogs, the pigmentary keratitis was so dense and they remained blind throughout the observation period.

The topical medications were administered at a frequency ranging from 2 to 8 hours, depending on severity of infection, spaced at 5 to 15 minutes (Wilkie and Whittaker, 1997). The topical application provided therapeutic concentration of the drug and markedly increased the penetration in corneal epithelial cells (Munro, 2001). According to Sansom (1988), ointments delayed the corneal healing. At the same time, ophthalmic solutions affected vision and corneal healing minimally and were easy to administer (Malmberg and Lupo, 2004). Hence solutions were preferred over ointments (Miller, 2001). The use of ointments in KCS was also inadvisable as they disrupted the tear film and trapped exudates (Sansom, 1988).

Elizabethan collar used in all the cases and were found to be useful in preventing self trauma, until complete healing was observed (Kim *et al.*, 2009). Also, the collar was well tolerated by the patients.

5.3 SURGICAL TREATMENT

The Dogs II, IV, XI and XIV with stromal ulcerative keratitis, Dogs III, VIII and XII with descemetocele and Dogs V, VI, VII, XIII and XV with staphyloma were subjected to surgical treatment. Treatment of corneal ulcers was based on their depth and rate of progression. The general therapeutic principles were applied to treat the ulcer, superficial or deep, but rapidly progressing deep stromal ulcers warrant surgical intervention in order to give support and reduce the risk of perforation (Renwick, 1996). The dogs with pigmentary keratitis, i.e., Dog XVI, with nasal fold trichiasis of the left eye and Dog XVII, with lower eyelid

entropion and exposed globe of the left eye were also subjected to surgical treatment. Surgical excision was opted for Dog XVIII presented with cherry eye. All the surgical procedures were carried out under general anaesthesia with proper preoperative preparation. All the surgical procedures were conducted with the help of an operating microscope, with a magnification of 0.4X.

5.3.1 Preoperative preparation of patient

All the cases were put under medical therapy with topical instillation of ciprofloxacin eye drops four times for three days prior to surgery and the medication was sufficient to control infection. The administration of topical broad spectrum antibiotics 6 to 12 hours prior to surgery especially in infectious keratitis and corneal perforation was suggested by Wilkie and Whittaker (1997). Solid food was withheld for twelve hours before surgery and liquid food for four hours before the surgery in all the cases.

5.3.2 Preoperative preparation of surgical site

The preparation of eyelids, adnexa and ocular surfaces using povidone iodine solution and irrigation with sterile normal saline for ocular and adnexal surgeries were found to be satisfactory. The use of 1:50 povidone-iodine solution was effective in eliminating bacterial contamination of the external ocular tissue without causing tissue reaction (Roberts *et al.*, 1986) and to remove residual material from conjunctiva and ocular surfaces (Morgan, 2004a). The paradoxical effect with dilution of povidone-iodine solution was also noticed by Hartley (2010), with more dilute solutions exhibiting greater antibacterial action. In cases of preparation of ruptured globe, povidone-iodine solutions were not used as it was not tolerated by the corneal endothelium, where sterile normal saline was used.

5.3.3 Preparation of collagen sheet

The pliability and transparency achieved by soaking the collagen sheet in gentamicin eye drops enabled easy placement on the newly prepared recipient bed as observed by Anoop *et al.* (2010).

5.3.4 Anaesthesia

General anaesthesia following the regimen with atropine sulphate, xylazine hydrochloride, ketamine hydrochloride and diazepam provided satisfactory anaesthesia for the corneal, eyelid and adnexal surgeries. Induction and recovery in all the cases were found to be smooth and uneventful.

The potential benefit of anticholinergic treatment was prevention or reversal of oculocardiac reflex. In ketamine administration, the palpebrae remained open, globe was centrally positioned and the palpebral and corneal reflex persisted. Diazepam reduced the ketamine induced increase in intraocular pressure (Collins *et al.*, 1995). The irrigation of cornea with sterile balanced salt solution throughout the surgical process prevented drying of the cornea, preserved corneal epithelium, improved tissue handling and visualization (Wilkie and Whittaker, 1997)

Anaesthesia was not administered in any of the cases for the removal of temporary tarsorrhaphy sutures and measurement of intraocular pressure. In these cases topical administration of 4% lignocaine was done, one drop instilled for four times at an interval of 5 minutes. Topically administered anaesthetic may be irritating and could cause transient conjunctival hyperaemia (Collins *et al.*, 1995). But repeated use of topical anaesthetic was contraindicated in corneal ulceration due to its toxic effect on the corneal epithelium, affecting re-epithelialisation of corneal wound, in spite of the mild analgesic effect (Stiles *et al.*, 2003).

5.3.5 Surgical technique

5.3.5.1 Corneal debridement

Debridement was done in all the cases with ulcerative keratitis except in Dogs III and VIII. Debridement stimulated corneal healing by removal of the abnormal corneal epithelium, promoted epithelial attachment to basement membrane and aided in removal of debris from the exposed stroma (Moore, 2003 and Vanore *et al.*, 2007). Debridement of the loose epithelium was easily accomplished with a cotton tipped swab (Whitley, 2000), was found inexpensive and possessed little risk to the eye (Ollivier, 2003). Also, it could be easily accomplished without sedation (Miller, 2001). Saline irrigations were used in order to remove the dead and necrotized tissues in the eye. The aqueous clot formed over the wound in full thickness defects was removed only at the time of repair after anaesthetizing the animal (Sansom, 2000).

5.3.5.2 Iridectomy

Iridectomy was performed in cases V, VI, VII, XIII and XV with irreducible, necrosed staphyloma. The prolapsed portion of the iris was contaminated, necrosed and showed severe adhesions. The extrusion of the iris through the corneal wound sealed the wound but provided a potent tract to infections. Such wounds should be repaired as early as possible so that the chance of infection could be reduced (Bedford, 1987). The aqueous clot formed over the wound was removed only at the time of repair (Wilkie and Whittaker, 1997 and Sansom, 2000). Iridectomy was performed as per the procedure described by Hollingsworth (2003) since adhesions prevented its replacement (Bedford, 1987). Penetration of anterior chamber caused aqueous humor leakage and loss of shape of globe (Sansom, 2000). The shape of the globe was reconstituted using the sterile balanced salt solution to the possible extent. However, in full thickness defects, maintenance of corneal curvature or shape was deemed to be less important (Wilkie and Whittaker, 1997). Since the corneal wound edges were unable to be apposed by suturing, it was kept as such in all these dogs (Chinchu, 2010). The

irrigation of cornea with sterile balanced salt solution throughout the surgical procedure prevented drying of the cornea, preserved corneal epithelium, improved tissue handling and visualization (Wilkie and Whittaker, 1997).

5.3.5.3 Third eyelid flap

Third eyelid flap was placed in Dog XIII, with staphyloma after iridectomy as described by Bedford (1987). The membrane was sutured over the eyelid. The use of membrane flap was suggested for the treatment of corneal ulcers by Anderson *et al.* (1976), Startup, (1984), and Moore (2003). Nictitating membrane flap was found to be more useful as a single treatment for corneal ulcers where a deep defect or a large area was involved (Startup, 1984). The procedure took only less time and was inexpensive, well tolerated and provided direct support to the weakened cornea (Anderson *et al.*, 1976). Also, the flap covered and protected the whole corneal surface (Bedford, 1980).

Startup (1984) and Morgan (2004b) suggested the suturing of membrana nictitans on to the dorsal bulbar conjunctiva in brachycephalic breeds with protruding eyes or membrane flap of limited size, because there was a great tendency for sutures to pull out during motion between the third eyelid and the cornea. Even after suturing on to the bulbar conjunctiva, disruption of third eyelid flap was noticed by Resmi (2008).

The flap got dislodged on the seventh post-operative day. However, it helped in minimizing postoperative discomfort and infection (Stanley *et al.*, 1998).

5.3.5.4 Application of collagen sheet

The collagen sheet was placed on the cornea of Dogs V, VI, VII and XV with necrosed staphyloma, Dogs II, IV and XI, with stromal ulcers and Dog XII with descemetocele. The collagen sheet of bovine small intestinal origin became pliable and transparent after soaking in gentamicin eye drops. Thus it enabled easy

placement of the sheet over the newly prepared recipient bed on the cornea. The collagen sheet was sutured on to the bulbar conjunctiva using 4/0 silk at 12, 3, 6 and 9'O clock position with simple interrupted sutures, followed by placement of temporary tarsorrhaphy sutures (Vanore *et al.*, 2007). Suturing enabled proper placement and fixation of the sheet over the cornea. Also, the temporary tarsorrhaphy sutures supported the sheet (Chinchu, 2010).

Even though enucleation was advised in case of ulceration with severe suppurative endophthalmitis (Kim *et al.*, 2009) and severe intraocular damage accomplished with lens extrusion (Hollingsworth, 2003), as in case of Dog No. XV, collagen sheet placement along with proper management helped to retain the globe and also the integrity of cornea.

Collagen shields proved to be highly oxygen permeable, promoted epithelial healing, decreased inflammatory cell infiltration and reduced corneal oedema (Geasey *et al.*, 1992). SIS acted as a scaffold for the repair and provided valuable tectonic support and helped in epithelialisation because of its collagenous nature that mimicked the stromal surface (Bussieres *et al.*, 2004).

Deep corneal defects managed by the application of biodegradable collagen based material, SIS, enabled to attain optimum results and preserved vision as it promoted tissue healing with negligible scar formation (Featherstone and Sansom, 2000 and Featherstone *et al.*, 2001). Also, it helped to maintain corneal transparency and preservation of corneal integrity (Vanore *et al.*, 2007). Collagen sheet of bovine intestinal origin was also found very effective in the cases of staphyloma, subsequent to corneal ulcers (Anoop *et al.*, 2010). The principle advantage of the techniques was the ready availability of material to fill and strengthen the stromal defect (Hansen and Guandalini, 1999) in addition to the cost effectiveness, commercial availability and easiness to handle (Bussieres *et al.*, 2004).

The Dogs XII with descemetocoele in which the collagen sheet was placed developed hypopion with progression into staphyloma by the 10th day of

observation. A similar complication was mentioned by Bussieres *et al.* (2004).

5.3.5.5 Temporary tarsorrhaphy

Temporary tarsorrhaphy was done in all the surgical cases with ulcerative keratitis. Moore (2003) also suggested the use of tarsorrhaphy as an effective bandaging technique after intraocular surgeries. The medial canthus was left open that facilitated the instillation of topical postoperative medications and was found effective in providing short-term protection of the surgical site and supporting the corneal healing (Herring, 2003). Also, it provided an additional support to the collagen sheet that was sutured and fixed over the cornea (Chinchu, 2010). Temporary tarsorrhaphy was also inexpensive, easily applied, well tolerated and provided direct support to the weakened cornea (Anderson *et al.*, 1976). Partial disruption of tarsorrhaphy sutures was noticed in Dogs IV, XII and XIII.

5.3.5.6 Lateral lid shortening

Lateral lid shortening done in Dog XVII was found to be effective in reducing the exposed surface area of the globe (Whitley *et al.*, 1991). The technique was also advised by Kaswan *et al.* (1995) and Moore (2003) in dogs with KCS, lagophthalmos and exophthalmos and in brachycephalic dogs like Chinese pugs, with dense pigmentary keratitis or subsequent pigment accumulation even after cyclosporine therapy, due to excessive corneal exposure. The smaller palpebral fissure thus created resulted in improved blink function, better tear film distribution and decreased tendency to corneal desiccation (Renwick, 2007). Lid shortening also enabled better elimination of ocular mucus, hairs and debris, redistribution of the tear film lipid layer and reduced the evaporation rates over the central cornea (Carrington *et al.*, 1989) and the opportunity for the development of ulcerations (Tolar *et al.*, 2006).

The lid shortening was an effective permanent remedy for exposure keratopathy associated with euryblepharon. Even though complete resolution of clinical signs that had developed as a result of large palpebral fissure could not be

achieved, the technique improved the comfort of the patient (Krohne, 2008). Subsequent observations on cornea revealed complete control of inflammation characterized by the absence of keratinization and hypertrophy of the corneal epithelium with uniform redistribution and spread of tear film (Kaswan *et al.*, 1995).

The technique was combined with modified Hotz-Celsus operation for the medial lower lid entropion in this dog as it exacerbated the deleterious effects of globe exposure and poor blink function.

5.3.5.7 Nasal fold reduction

Nasal fold reduction in Dog XVI was effective in reducing chronic irritation and inflammation of cornea due to nasal fold trichiasis (Peiffer *et al.*, 1987). Resection of nasal fold for nasal fold trichiasis in brachycephalic breeds was also suggested by Renwick (2007). Even though complete resolution of pigmentation associated with nasal fold trichiasis could not be achieved, reduction of fold improved the comfort of the patient (Krohne, 2008).

5.3.5.8 Modified Hotz-Celsus operation for anatomical entropion

The modified Hotz-Celsus operation used for the correction of medial lower lid entropion in Dog XVII provided satisfactory result. Generally, the technique was used for the correction of all types of entropion (Morgan, 2004b). Even though the pigmentation associated with chronic irritation and inflammation persisted, the progression of pigmentation was controlled and provided better comfort to the patient (Krohne, 2008). Often, it might be necessary to remove the nasal folds, if it exacerbated the degree of entropion (Renwick, 2007).

5.3.5.9 Resection of prolapsed membrana nictitans gland

The third eyelid gland was removed in Dog XVIII presented with cherry eye. The STT readings of this dog on the 7th postoperative day showed decrease in tear production and indicated development of KCS (Gionfriddo, 1995b and Kaswan *et al.*, 1995).

Although the modern techniques of repositioning of the gland to its normal location by anchoring or tacking (Kaswan and Martin, 1985 and Stanley and Kaswan, 1994) and by imbrication or pocket technique (Morgan *et al.*, 1993) were described, none of the techniques have proved to be efficient and recurrence is common (Herrera, 2005 and Bharathi *et al.*, 2010). The removal of third eyelid gland was also suggested by Helper (1981) and Herrera (2005) as it seldom remains in normal position.

5.3.6 Postoperative care

Topical application of ciprofloxacin and oral cephalixin administered postoperatively, and parenterally administered ceftriaxone on the day of surgical intervention was effective in controlling infection in all the surgically treated cases in the present study. According to Sansom (1988), once an intraocular infection was established, it was difficult to treat due to blood-ocular barrier and the absence of lymphatics. All the cases of ulcerative keratitis treated surgically were deep and often were infected. Hence both systemic and topical treatments were considered.

Topical tropicamide helped to improve animal's comfort and thereby the tendency to self trauma (Collins *et al.*, 1995). It also controlled the ciliary body spasm and helped to prevent synechiae formation, especially in full thickness lesion, in all the cases except in Dogs V and XII. Dog V had anterior synechiae on the day of presentation itself and Dog XII developed anterior synechiae during the course of surgical treatment subsequent to the development of hypopion. The

significance of postoperative mydriatic-cycloplegic therapy was also pointed out by Wilkie and Whittaker (1997) and Wang *et al.* (2008).

Topically instilled NSAIDs were recommended to suppress ocular inflammation and postoperative anterior uveitis (Giuliano, 2004 and Gilmour and Lehenbauer, 2009). The NSAID, flurbiprofen (0.3%) used in the present study satisfactorily controlled the postoperative pain and inflammation (Munro, 2001 and Pirie *et al.*, 2011).

Multivitamins supplemented in all the cases of ulcerative keratitis promoted corneal epithelial healing (Startup, 1984).

Supportive care of the cornea with tear substitute topical 0.5% carboxy methyl cellulose sodium in dogs with relative exophthalmos helped to control corneal drying and enabled protection against corneal desiccation and exposure keratopathy (Wilkie and Whittaker, 1997 and Hartley, 2010)

Elizabethan collar used in all the surgically treated cases were well tolerated by the patients and was found useful in preventing self trauma, until complete healing (Herring, 2003).

The temporary tarsorrhaphy sutures and the skin sutures were removed on the seventh postoperative day.

5.4 MAIN ITEMS OF OBSERVATION

5.4.1 Signalment

The average age of the Chinese pug dogs with ulcerative keratitis selected in the study were 14.00 ± 2.45 , ranging from 2 months to 36 months. This was less when compared to the average age of all the dogs selected in the study. The higher incidence of corneal ulcer in Chinese pugs at an early age was also noticed by Bussieres *et al.* (2004), Raji (2006) and Chinchu (2010). Brachycephalic

breeds, in general, were found to develop ulcerative keratitis at any age (Wang *et al.*, 2008). However, no sex predisposition was noticed among dogs with ulcerative keratitis as observed by Bentley *et al.* (2001), Janssens (2007) and Chinchu (2010). Among the dogs with ulcerative keratitis Dog X, with concurrent bilateral KCS, was a puppy of 2 months. KCS typically affect smaller dogs since they have inherently lower basal tear production (Berger and King, 1998) and results in ulcerative keratitis (Startup, 1984; Mandell and Holt, 2005 and Kim *et al.*, 2009).

The average age of the dogs with glaucoma selected for the study was 18.10 ± 2.74 , ranging from 8 months to 36 months. Even though Kato *et al.* (2006) reported early onset of glaucoma in Chinese pugs compared to other breeds of dogs, the dogs belonging to all age groups were equally affected (Gelatt and Mackay, 2004a). Usually, glaucoma develops in middle aged dogs (Crispin *et al.* 2008). Out of the 10 dogs with glaucoma, 9 were females. The predisposition of females to glaucoma was also observed by Slater and Erb (1986) and Gelatt and Mackay (2004a).

The dogs with pigmentary keratitis selected for the study were older compared to the selected group of dogs. According to Magrane (1977), some Pekingese and pug blood lines showed congenital predisposition to corneal pigment deposition at an early age.

The Dog XVIII with cherry eye was a puppy of 3 months age. The condition is commonly noticed in puppies (Christmas, 1992).

The average duration of illness of dogs with ulcerative keratitis ranged from 2 to 11 days. The corneal ulcers that persisted for a long period were considered as refractory ulcers. Re-epithelialisation of uncomplicated ulcers needed three to five days (Ollivier, 2003 and Mandell and Holt, 2005), but if they do not heal within this period could be considered as refractory ulcers (Whitley, 2000). Most of the deep and full thickness defects were complications of simple ulcerative keratitis (Startup, 1984; Moore, 2003 and Mandell and Holt, 2005).

The previous treatment with corticosteroids in Dogs III, V, XI and XV increased the risk of progression of ulcer and delayed healing (Wilkie and Whittaker, 1997 and Tolar *et al.*, 2006).

In dogs with pigmentary keratitis, the pigmentation of cornea was observed by the owners over a period of 6 to 7 months before presentation. The pigmentation of cornea was suggested as a sign of chronic inflammation by Peiffer *et al.* (1987) and Gilger *et al.* (2008).

5.4.2 Physiological parameters

Except for the rise in temperature that was noticed in Dog XIII and XVI on the first day of observation, none of the animals showed any signs of systemic involvement throughout the observation period. These dogs were screened positive for microfilaria.

The physiological parameters were evaluated to rule out the presence of any concurrent systemic illness. As the ocular disease condition with which the animal was presented might be a manifestation of the indwelling systemic disturbances, the general examination preceded the ophthalmic examination (Felchle and Urbanz, 2001).

5.4.3 Clinical examination

5.4.3.1 General condition of the patient

All the dogs selected in the study were in good health except Dog XVII, which had poor condition on the first day and recovered good health in later observations. Multivitamins were administered in all the cases.

According to Gum (1991), the glycogen on the corneal epithelium act as

the main source of energy under stressful conditions like trauma or surgical wounds and therefore, if the glycogen stores get depleted, normal healing of epithelium and cellular locomotion over the surface would be inhibited. Depletion of glycogen reserve may occur especially in, immunosuppressive diseases such as demodicosis, dermatophytosis and, renal failure (Pardo *et al.*, 2005) and in endocrinopathies like diabetes mellitus, hyperadrenocorticism and hypothyroidism (Williams, 2008).

5.4.3.2 Wet film examination

Moving blood parasites were not detected in any of the cases selected for the study except in Dog XIII with ulcerative keratitis and Dog XVI with pigmentary keratitis. Massa *et al.* (2002) described uveitis associated with common infectious organisms like *Dirofilaria* sp. as one of the most common cause of blindness in dogs. However, the moving parasites detected were inert considering ocular involvement. Oral administration of ivermectin at the rate of 50 µg/kg body weight helped to clear the larval nematode (Bowman and Atkins, 2009) as evidenced by the reexamination on the 25th day.

5.4.3.3 Condition of the eye

5.4.3.3.1 Appearance of eye, eyelids and adnexa and cornea

A variable degree of buphthalmos was noticed in Dogs IV, XVII, XIX, XX and XXII with glaucoma (Woerdt, 2001 and Storm *et al.*, 2011). Buphthalmos developed in most of the dogs with intraocular pressure above 40 mm of Hg (Gelatt, 1997). The young dogs rapidly developed buphthalmia, which was reversible by the medications easily (Gelatt, 1981). Even though mild elevations in IOP were noticed on subsequent days during the course of treatment, clinical signs were not observed (Mughannam *et al.*, 2004).

The phthisis bulbi that was noticed in the right eye of Dog XVI was the sequel of a complicated deep ulcer the animal had previously (Ollivier, 2003).

Epiphora, blepharospasm and blepharodema displayed by the dogs with ulcerative keratitis indicated trigeminal irritation and associated pain (Bedford, 1987 and Mandell and Holt, 2005). These signs disappeared in the due course of treatment.

Episcleral vessel congestion was a constant finding noticed in all dogs with glaucoma on the first day of observation (Kallberg, 2007) and resolved completely during the subsequent days of observation. According to Helper (1989), the vortex veins within sclera were affected by initial increase in pressure and thus anterior ciliary vein would take over their work and become distended in the process.

Pigmentation of the cornea was noticed in both the dogs with pigmentary keratitis. This was due to the mechanical irritation by the abnormalities in eyelids and adnexal structures (Peiffer *et al.*, 1987). As observed by Roberts (1954), the pigment covered the entire cornea. The left eye of the Dog XVI with pigmentary keratitis had nasal fold trichiasis and the left eye of the Dog XVII with pigmentary keratitis had lower eyelid entropion. In both the dogs with pigmentary keratitis, accumulation of hair, dirt and debris was noticed on the central cornea in addition to xerophthalmia as a result of poor tear film distribution (Renwick, 2007) and due to absence of an effective blink (Kaswan *et al.*, 1995).

In all the dogs with keratitis, relative exophthalmos was noticed. Excessive corneal exposure greatly exaggerated the severity of corneal lesions and resulted in dense pigmentation and corneal ulceration (Kaswan *et al.*, 1995 and Renwick, 2007). Oedema associated with corneal lesion was observed in all dogs with ulcerative keratitis (Startup, 1984 and Mandell and Holt, 2005).

The corneal changes associated with glaucoma mentioned by Gelatt (1981) were noticed in Dog XXII, with glaucoma, which included pigmentation

and corneal oedema. In glaucomatous eyes, the corneal endothelium may stretch, breaking Descemet's membrane and lead to striae, characterized by white streaks in the cornea (Gionfriddo, 1995c) resulting in oedema (Woerdt, 2001 and Palmer *et al.*, 2008).

5.4.3.3.2 Nature of discharge

The nature of discharge was clear in all the cases except in Dogs IV, V, X, XIII and XV. In these dogs, the discharge was mucopurulent in nature. According to Sansom (1988), the purulent ocular discharge indicated a bacterial infection. Purulent discharge associated with melting ulcer in Dog IV was described by Wolfer and Grahn (1994). Sansom and Barnett (1985), Kaswan *et al.* (1995) and Davidson and Kuonen (2004) stated mucopurulent ocular discharge, which was noticed in Dog X diagnosed to have KCS, as the hallmark sign of this condition. The discharge in these dogs became clear in due course of treatment.

5.4.3.3.3 Type and extent of lesion

All the cases of ulcerative keratitis were categorized by the depth of corneal involvement as superficial ulcer, deep stromal ulcer, descemetocoele and corneal perforation based on the classification described by Gilger *et al.* (2008).

In Dogs I and IX, the ulcers were superficial, shallow and stationary. Both the cases responded well to medical treatment and immediate reduction in depth and extent were noticed. Superficial ulcerations with least stromal involvement heal quickly within three to five days with minimum scar formation (Ollivier, 2003 and Mandell and Holt, 2005).

The Dogs II, IV, X, XI and XIV had stromal ulcer. Corneal ulcers turn into deep stromal ulcers because of infection, tear film deficiency due to KCS (Williams, 2008) as in case of Dog X and inappropriate use of corticosteroids (Wilkie and Whittaker, 1997) as in case of Dog XI. According to Whitley (2000), deep stromal ulcers were potentially a threat to globe and vision. The dog X was treated medically and the Dogs II, IV, XI and XIV were treated surgically. Progressive reduction in depth and extent of ulcer was noticed in all the cases in due course of treatment. Deep stromal ulcerations required vascularization for healing and took three weeks to heal (Moore, 2003 and Mandell and Holt, 2005).

Stromal melting was observed in Dog IV with stromal ulcer. Whitley (2000) described melting ulcers as collagenase and protease associated ulcers. They were characterized by progressive stromal dissolution secondary to proteolytic activity due to imbalances between proteases and protease inhibitor levels (Vanore *et al.*, 2007). Although traumatic in origin, the ulcer gets infected by opportunistic bacteria, resulting in rapid melting (Wolfer and Grahn, 1994). According to Startup (1984), even though large areas of cornea was badly affected, the melting ulcer had little tendency to perforate the cornea as collagenase did not affect the Descemet's membrane. But, the ulcer was found to have progressed into a staphyloma without anterior synechiae by the tenth day of observation. Progressive reduction in the depth and extent of the ulcer was noticed on subsequent observations.

Among the three dogs with descemetocele (Dogs III, VIII and XII), Dog XII had an extensive stromal ulcer. Descemetocele are ocular emergencies impending perforation of globe and required immediate surgical intervention (Startup, 1984 and Whitley, 2000). All the dogs were treated surgically. Descemet's membrane being elastic in nature usually projects forward as descemetocele and acts as a significant barrier to perforation (Miller, 2001). The depth and extent of descemetocele in Dogs III and VIII considerably reduced by the 25th day of observation except in Dog XII. In this dog the descemetocele progressed into a small healing staphyloma. Also, due to the breakdown of blood

aqueous barrier and exudation of blood proteins into the eye, the associated uveitis was well established and the eye developed hypopion and finally resulted in permanent anterior synechiae (Gionfriddo, 1995d). The recurrent superficial ulcer noticed in Dog VIII when presented on the 60th day of observation could not be considered as a classical indolent ulcer because of the absence of characteristic epithelial lip and early healing (Whitley, 2000 and Moore, 2003).

The protruded iris was irreducible and involved a large area of cornea in all the dogs presented with staphyloma. According to Wilkie and Whittaker (1997), corneal perforation occurred as a result of a complicated corneal ulcer. In corneal perforation, the extrusion of iris through the corneal wound would seal the wound but provided a potent tract to intraocular infection and hence required immediate repair (Bedford, 1987). The iris was found to have covered with fibrin clot and debris in all the dogs, except in Dog VII. The iris was severely adhered to the cornea and it sealed the corneal wound in Dogs V, VI, VII and XIII. Seepage of aqueous humor was not noticed in any of the case. The dog V with staphyloma was presented with an already collapsed anterior chamber and anterior synechiae. The penetration of anterior chamber causes loss of aqueous humor and thereby shallowing of anterior chamber, uveitis, miosis, aqueous flare and hyphaema (Sansom, 2000) with subsequent anterior synechiae and phthisis bulbi (Massa *et al.*, 2002). The lesion in centre of cornea of all these dogs became shallow on tenth day of observation and by 25th day, the cornea healed completely. But, in Dog XIII, a small facet was noticed on the central cornea by 40th day of observation and in Dog V, the shape of the globe was not regained. Dog XV was presented with staphyloma in addition to extensive stromal injury and lens extrusion. Even though enucleation was advised in such cases with severe intraocular injury (Hollingsworth, 2003 and Kim *et al.*, 2009), the surgical treatment and postoperative care provided excellent result. Healing cornea with granulation was noticed in this dog by the 40th day of observation with an aqueous filled anterior chamber.

The scleral pigmentation was noticed in addition to the densely pigmented cornea in both the dogs with pigmentary keratitis. The presence of eyelid and adnexal abnormalities in these dogs indicated the associated chronic inflammation and irritation (Peiffer *et al.*, 1987 and Gilger *et al.*, 2008). Further accumulation of corneal pigments was not observed in these dogs once the medical treatment was started after the surgical correction of eyelid and adnexal abnormalities.

5.4.3.3.4 Visual function tests

Menace reflex was found to be sluggish in all the dogs with ulcerative keratitis on the day of presentation and was absent in Dogs V and XV, throughout the observation period. All the dogs selected in the study had normal palpebral and corneal reflexes throughout the observation period. Martin (2001) suggested to elicit a blink before performing menace test to assure whether the facial nerve was intact through the palpebral or corneal reflex. The presence of normal palpebral and corneal reflexes in dogs with ulcerative keratitis also ruled out the possibility of neurologic deficit (Hartley, 2010 and Mitchell, 2011).

The menace and pupillary light reflex in all the dogs with ulcerative keratitis was found normal when the cornea attained clarity except in Dogs V, XII and XV. The Dog X lost menace reflex by 40th day of observation due to complete bilateral corneal pigmentation.

In dogs with pigmentary keratitis, menace reflex was absent and, the pupillary light reflex could not be assessed in both the eyes throughout the observation period. Also, the blinking of eyes in these dogs was found to be incomplete due to lagophthalmos (Renwick, 2007 and Williams, 2008). According to Kaswan *et al.* (1995), in brachycephalic dogs, especially Chinese pugs that could not blink effectively, excessive corneal exposure greatly exaggerated the severity of corneal lesions.

The pupillary light reflex, found to be sluggish in all the dogs with glaucoma on the day of presentation, became normal in due course of treatment. The reflex could not be assessed in Dog XVII, with complete corneal pigmentation. The normal direct pupillary light response in these dogs in due course of treatment indicated an intact optic and oculomotor nerve (Moore, 2001). Also, mydriasis with absence of pupillary light reflex indicated severe optic nerve damage or disruption of the parasympathetic supply to the iris sphincter musculature or both (Bedford, 1987).

5.4.3.3.5 Corneal clarity

On the day of presentation, all the dogs with ulcerative keratitis showed variable degree of corneal opacity. The corneal opacity was found as a common symptom of keratitis (Bedford, 1982 and Moore, 2001). According to Krawitz (1963), low number of leukocytes usually found in the basal cells increased during keratitis that accounted for the corneal haze.

The cornea of all the dogs presented with superficial ulcers attained clarity by the 10th day after presentation.

Corneal clarity was noticed in all the dogs presented with stromal ulcers (Dogs II, X, XI and XV) except in Dog IV in which a central haziness remained on the 60th day of observation.

Among the Dogs with descemetocele (Dogs III, VIII and XII), moderate opacity remained in Dog XII on the 60th day of observation.

Except in Dog V, complete corneal clarity could not be observed in any of the dogs with staphyloma (Dogs VI, VII, XIII and XV) by the 60th day of observation.

According to Miller (2001), when the stroma was damaged, collagen manufactured by the fibroblasts gets deposited in a random manner producing scar. The disruption of the regular lamellar arrangement of stroma or changes in

the collagen type laid during the wound healing might lead to irreversible opacification of cornea and interfere with transparency (Wilkie and Whittaker, 1997). The factors essential to maintain corneal clarity included lack of vascularization or pigmentation and fibrosis (Morreale, 2003), regular arrangement of collagen in combination with the smooth non-keratinized squamous epithelium and tear film and relative dehydrated nature of cornea (Herring, 2003).

Since the observation was made for a maximum period of 60 days, complete clarity could not be observed in Dogs IV, VI, VII, XII, XIII and XV. Clearing usually requires more than three months. However, clarity was achieved in uncomplicated cases.

5.4.3.3.6 Corneal oedema

Corneal oedema was observed in all the ulcerative keratitis cases presented and progressive reduction was noticed during the subsequent observation with corneal healing. Corneal oedema was found to be a common symptom of ulcerative keratitis (Startup, 1984 and Mandell and Holt, 2005). The endothelium maintains a constant thickness, hydration and transparency of cornea (Rodrigues *et al.*, 2006). According to Wilkie and Whittaker (1997), any break in the dehydrated state of stroma maintained by corneal epithelium and endothelium resulted in corneal oedema. The imbibition of fluid by the epithelium or stroma and the failure of extrusion of electrolytes by the corneal endothelium increased the water content that led to corneal oedema and reduced transparency (Gilger *et al.*, 2008).

Corneal oedema in Dog XXII with glaucoma resulted from compression of stromal lamellae forcing water into epithelium. Chronically elevated intraocular pressure often damage endothelial cells and Descemet's membrane with focal striae and associated corneal oedema (Peiffer *et al.*, 1987).

5.4.3.3.7 Fluorescein staining test (FST)

Fluorescein dye strips were used for assessing the depth, extent and healing of corneal lesions. FST was positive in all the dogs presented with ulcerative keratitis on the day of presentation. The hydrophilic nature of corneal stroma was responsible for the retention of the water soluble sodium fluorescein dye (Wilkie and Whittaker, 1997).

Except Dogs XII, XIII and XV, all the dogs with ulcerative keratitis did not retain fluorescein stain on the 25th day of observation. The Dogs XII, XIII and XV were fluorescein negative by the 40th day of observation.

The Dogs with descemetocele (Dogs III, VIII and XII) were stained only at the borders. This was in accordance with the finding of Mitchell (2011).

5.4.3.3.8 Vascularisation of cornea

Vascularisation of cornea was noticed in all the dogs with ulcerative keratitis except in Dogs I and IX with superficial ulcer, Dog IV with stromal ulcer, Dogs III, VIII and XII with descemetocele and Dog VII with staphyloma.

Vascularisation of cornea represented an emergency reaction, to improve the nutrition of the cells of the cornea in response to various pathologic processes and, to support stromal healing process (Magrane, 1977). The vascular response to peripheral lesions was found to be more prominent compared to the central lesions (Bentley, 2005). Deep corneal vessels which formed 360^o perilimbal pattern noticed in Dog XIII suggested an associated uveitis (Moore, 2001).

Vascularisation was found to increase in intensity by the 10th day in dogs which already had it and developed in dogs which did not show any vascularisation on the first day. However, vascularisation was not noticed in dogs with superficial

ulcers. The neovascularization of cornea or intensification in vascularity following implantation of small intestine submucosa might be induced by surgery or initial traumatic event and got amplified during SIS integration rather than an immune rejection (Bussieres *et al.*, 2004 and Vanore *et al.*, 2007).

According to Wilkie and Whittaker (1997), the corneal vascularisation was depending upon the duration of ulcer and developed after three to five days. Deep stromal ulcers required vascularisation for healing if they involved more than one third of corneal thickness (Mandell and Holt, 2005). But, uncomplicated corneal ulcers healed without vascularisation (Slatter and Dieterich, 2003). Although corneal neovascularisation was beneficial in the early healing stages of ulcerative keratitis, an excessive degree causes ocular discomfort and corneal opacity (Featherstone *et al.*, 2001).

The vascularisation of cornea noticed in Dog XVI, presented with pigmentary keratitis was an indication of chronic inflammation (Peiffer *et al.*, 1987).

Complete regression of vascularisation was noticed in all the dogs once cornea was healed (Brunott *et al.*, 2007).

5.4.3.3.9 Conjunctival changes

According to Wilcock (1993), conjunctival reactions like hyperemia, cellular exudation and lymphofollicular hyperplasia might be due to release of inflammatory mediators of either microbial, leucocytic or tissue origin from the injured cornea. Conjunctival hyperaemia was noticed in all the dogs with keratitis on the day of presentation and resolved completely in due course of treatment.

5.4.3.3.10 Pigmentation of cornea

All the dogs in the study with keratitis showed varying degree of pigmentation by the 60th day of observation. Pigmentation was noticed in Dogs XV, XVI and XVII on the day of presentation itself.

The large and prominent eyes of Chinese pugs predispose the animal to pigment deposition that started at the corneal side of limbus and at times the pigment, melanin, covered the entire cornea (Roberts, 1954). The pigment deposition was an indication of chronic inflammation (Gilger *et al.*, 2008). According to Magrane (1977), uveal pigment migrated through nerve and vessel opening and deposited in the corneal stroma, following the development of anterior synechiae in corneal perforation as observed in Dog V.

Corneal pigmentation resulted from deposition of melanocytic cells in the basal epithelial cells and the anterior stromal tissue and accompanied vascularization, stromal inflammatory cell infiltration, and even granulation tissue formation as in Dog XV (Gilger *et al.*, 2008).

In Dog X and XVIII, the pigmentary keratitis was the devastating consequence of KCS (Gionfriddo, 1995b). Also, the pigmentary keratitis was so dense and resulted in blindness (Kaswan *et al.*, 1995). The pigmentation remained in these dogs till the last day of observation.

The pigmentary Keratitis noticed in Dogs XVI and XVII was an indication of chronic inflammation or mechanical irritation such as entropion or facial fold trichiasis (Peiffer *et al.*, 1987). Due to chronic exposure in these dogs, corneal epithelium reverted to skin pattern with keratinisation and pigmentation (Slatter and Dietrich, 2003). The pigmentary keratitis in these dogs was well controlled by the application of topical cyclosporine and topical 0.5% carboxy methyl cellulose (Townsend, 2007). The lateral lid shortening and modified Hotz-Celsus operation that was done in Dog XVII and the nasal fold resection that was done in Dog XVI

also helped to prevent further accumulation of pigments (Whitley *et al.*, 1991; Kaswan *et al.*, 1995 and Renwick, 2007).

In all the dogs with keratitis, relative exophthalmos increased the corneal exposure and it greatly exaggerated the severity of corneal lesions and resulted in dense pigmentation (Kaswan *et al.*, 1995 and Renwick, 2007).

The pigmentation noticed in the cornea of Dog XXII, with glaucoma remained as such till the final observation day (Gelatt, 1981).

5.4.3.3.11 Schirmer tear test (STT)

The STT remains as the most common test for pre-corneal tear film and it assess the quantitative production of the aqueous portion of the tear film (Moore, 2003).

The STT was indicated in dogs with corneal ulcer because a normal tear film was required to maintain an intact corneal epithelium (Miller, 2001). Also, the predisposition of Chinese pugs to KCS and tear film deficiencies increased the significance of Schirmer tear testing (Kaswan *et al.*, 1995 and Williams, 2008).

The STT readings of all the dogs were within the normal range except in Dogs X, XVI, XVII and XVIII. However, the STT values in dogs with ulcerative keratitis were comparatively higher on the day of presentation as a result of reflex tear production due to stimulation of the trigeminal nerve (Hamor *et al.*, 2000).

Dog X, with ulcerative keratitis, was diagnosed to have congenital KCS on the day of presentation itself (Kaswan *et al.*, 1995). According to Davidson and Kuonen (2004), the KCS resulting from a deficiency in the aqueous component of tear film predispose the ocular surface to infection. The KCS had developed in Dog XVIII, with the surgical excision of prolapsed third eyelid gland (Gionfriddo, 1995b).

In dogs with pigmentary keratitis, Dogs XVI and XVII, border line tear deficiency was noticed. This might be due to the ocular surface tear deficiency due to tear evaporation as a result of the relative exophthalmos (Renwick, 2007) and the inability to complete blink (Williams, 2008).

All the dogs with KCS and tear film deficiencies responded well to the treatment with the lacrimostimulant, topical cyclosporine (Whitley, 2000 and Williams, 2008) and lacrimomimetic, topical 0.5% carboxy methyl cellulose (Townsend, 2007) evidenced by the rise in STT values to normal during the subsequent observations. The lateral lid shortening that was done in Dog XVII improved the tear film distribution in this dog characterized by the absence of accumulation of ocular mucus, hairs and debris over the central cornea (Carrington *et al.*, 1989; Whitley *et al.*, 1991; Kaswan *et al.*, 1995 and Renwick, 2007).

5.4.3.3.12 Intraocular pressure (IOP)

A drastic reduction in IOP was noticed in all the dogs by the 10th day of observation and subsequent observations showed gradual reduction in IOP. All the dogs responded to the medical management using topical 2% dorzolamide and topical 0.5% timolol maleate (Wilkie and Latimer, 1991; Gelatt and Mackay, 2001 and Plummer *et al.*, 2006). By day 60, the mean IOP returned to almost normal values.

5.4.3.3.13 Ophthalmoscopic examination

Mild degree of reflex uveitis accompanied all the dogs with ulcerative keratitis as observed by Sansom (2000) and Mandell and Holt (2005) clinically manifested as anterior uveitis with a reflex miosis and ocular hyperemia (Wilkie and Whittaker, 1997). According to Giuliano (2004), uveitis secondary to

ulcerative keratitis was due to trigeminal nerve stimulation of the cornea and secondary axonal stimulation of inflammatory mediators like substance P, leading to increased vascular permeability, chemotaxis of neutrophils and miosis.

The uveitis in Dog XII got established and developed hypopion which lead to the formation of anterior synechiae (Gionfriddo, 1995d).

5.4.3.3.14 Other relevant observations

Xerophthalmia was noticed in Dogs XVI and XVII presented with pigmentary keratitis on the day of presentation. In brachycephalic breeds, exposure keratopathy might occur due to central tear film deficiency because of the marked exophthalmic configuration (Carrington *et al.*, 1989) and the inability to complete a blink reflex (Hartley, 2010). Both the dogs responded well to the medical treatment with artificial tear preparation, topical 0.5% carboxy methyl cellulose (Hartley, 2010). The lateral lid shortening that was done in Dog XVII improved the tear film distribution (Whitley *et al.*, 1991; Kaswan *et al.*, 1995 and Renwick, 2007).

5.4.4 Haematological parameters

A complete blood count was estimated in combination with thorough clinical examination to rule out any underlying systemic disease or associated uveitis (Sansom, 2000). A recheck was made in all the dogs during the subsequent days of observation throughout the course of treatment as suggested by Metzger and Rebar (2004). The haematological values of all the dogs selected in the study were within the normal range as mentioned by Schalm *et al.* (2000). The findings were similar to that of Bath and Dua (2006a), Resmi (2008) and Priya (2009).

5.4.5 Random blood glucose level

The random blood glucose levels were estimated since diabetic dogs have significantly altered keratoconjunctival characteristics compared to non-diabetic dogs (Cullen *et al.*, 2005) and to detect whether the ocular disease presented was a manifestation of this endocrinopathy. The mean random blood glucose level of all the dogs selected in the study was within the normal range. Diabetes mellitus was also found to be associated with decreased corneal sensitivity (Williams *et al.*, 2007), exposure keratopathy in brachycephalic breeds with lagophthalmos (Williams, 2008) and KCS (Whitley *et al.*, 1991 and Kaswan *et al.*, 1995).

5.4.6 Culture and sensitivity test of corneal swab

Culture and sensitivity test of corneal swab was carried out in all the cases presented with ulcerative keratitis and/or full thickness perforations and it helped in definitive diagnosis and selection of appropriate antimicrobial therapy (Wilkie and Whittaker, 1997).

The corneal specimens were collected from the centre and periphery of the epithelial defect using sterile swabs as described by Massa *et al.* (1999) before instilling any topical medications as they might inhibit bacterial growth (Morreale, 2003).

The predominant isolate was Gram positive cocci (53.3%) with colony characteristics of *Staphylococcus* spp., followed by Gram negative bacilli (20%) and Gram positive bacilli. The *Staphylococcus* spp. was reported to be the predominant isolate in corneal defects by Massa *et al.* (1999), Whitley (2000) and Prado *et al.* (2005). According to Sweeny and Irby (1996), the infectious ulcerative keratitis was due to opportunistic bacterial infection by the resident conjunctival flora after trauma and ulceration. Being a common isolate in normal

flora of canine eye (Ollivier, 2003 and Wang *et al.*, 2008), the presence of *Staphylococcus* spp. in ulcerated cornea accounts opportunistic infection. The Gram negative bacilli were isolated from Dogs II and X and Dog IV, with melting ulcer. The melting ulcers caused by Gram negative rods like *Pseudomonas* spp. were most frequently observed in brachycephalic breeds by Startup (1984) and Wolfer and Grahn (1994). Although both Gram positive and Gram negative organisms were capable of infecting the cornea, Gram negative rods produced proteases which could result in rapid progressive destruction or melting of corneal stroma (Mandell and Holt, 2005).

Ciprofloxacin was selected as the primary antibiotic and was continued in all the cases as all the isolates were found sensitive to ciprofloxacin. This was in accordance with the findings of Whitley (2000) and Ledbetter *et al.* (2007)

Ciprofloxacin was found to be resistant in Dogs II and X. However, clinical response was noticed in these dogs with the use of ciprofloxacin and hence the antibiotic was not changed.

No growth was observed in Dogs I, VI, VIII and IX. In Dogs VI and VIII, the absence of growth may be due to the medical treatment the animal received before presenting to the hospital. Topical use of antimicrobials interferes with the culture and sensitivity and thus in definite diagnosis (Massa *et al.*, 1999). Growth was not noticed in Dogs I and IX, with superficial ulcers as stated by Ollivier (2003).

5.4.7 Exfoliative cytology of impression smears of corneal ulcer

The cytological examination of the corneal smear was done in dogs with ulcerative keratitis on all observation days. Cotton swabs were used as suggested by Felchle and Urbanz (2001) and were found to be useful for the collection of exudates and scrapings.

Nucleated epithelial cells were seen in higher quantity in dogs with deep defects and decreased gradually as the healing progressed. Anuclear keratinized cells were seen till the completion of corneal healing. Polymorphonuclear cells were noticed only in deep and full thickness defects. The observations were similar to that reported by Lavach *et al.* (1977).

The use of cytological examination of corneal smear to establish the cause of ocular disease, to design an initial treatment regimen and to study the cellular response associated with specific disease condition was emphasized by Massa *et al.* (1999), Hamor (2001) and Morreale (2003).

5.4.8 Complications

Partial disruption of temporary tarsorrhaphy sutures were noticed in Dogs IV, XII and XIII by the seventh postoperative day. Dislodgement of third eyelid flap was observed in Dog XIII by the seventh postoperative day. Similar complications were observed by Resmi (2008).

The melting ulcer in dog IV, and the descemetocoele in Dog XII, progressed into a small staphyloma by the 10th day of observation even though they show little tendency to perforate the Descemet's membrane (Startup, 1984).

Dog V had an already formed anterior synechiae and resulted in phthisis bulbi. Dog XII developed severe uveitis with hypopion by the seventh postoperative day. Anterior synechiae formation was noticed in this dog on the 25th day of observation and resulted in phthisis bulbi. The penetration of anterior chamber causes loss of aqueous humor and thereby shallowing of anterior chamber, uveitis, miosis, aqueous flare and hyphaema (Sansom, 2000) with subsequent anterior synechiae and phthisis bulbi (Massa *et al.*, 2002).

In Dog XV, the cornea was covered with deeply pigmented granulation tissue with resultant enophthalmos by the 60th day of observation. According to Miller (2001), enophthalmos was a serious threat after corneal perforation.

In Dog X, complete bilateral pigmentation and in Dog XI and Dog XIII, dense pigmentation was noticed in the cornea of affected eye by the 60th day of observation. In Dog X, pigmentation was the devastating consequence of KCS (Gionfriddo, 1995b). Also, the pigmentary keratitis was so dense and resulted in blindness (Kaswan *et al.*, 1995). Complete bilateral pigmentation persisted in Dogs XVI and XVII throughout the observation period.

In Dog XVIII, with cherry eye, decrease in tear production was noticed after the removal of third eyelid gland as observed by Kaswan *et al.* (1995).

Summary

6. SUMMARY

A study was conducted in Chinese pugs to evaluate the occurrence of different ophthalmic diseases, the clinical signs associated with each condition and the effectiveness of the various treatments adopted. The dogs of Chinese pug breed presented with eye affections to the surgery outpatient unit of University Veterinary Hospitals, Mannuthy and Kokkalai were included in the study. All the dogs were thoroughly examined and were categorised based on the disease condition for which they were presented or diagnosed. The dogs were subjected to detailed clinical and ophthalmic examination.

Among 578 Chinese pugs presented, 84 dogs had ocular affections and accounted 14.53% of the total dogs presented. Out of the 84 Chinese pug dogs with ocular diseases, 40 dogs had ulcerative keratitis, 10 dogs had glaucoma, 10 dogs had concurrent glaucoma and ulcerative keratitis, 12 dogs had conjunctivitis, seven dogs had pigmentary keratitis, 2 dogs had concurrent pigmentary keratitis and glaucoma and one dog each had proptosis, cherry eye and, concurrent ulcerative keratitis and keratoconjunctivitis sicca (KCS).

In total, the incidence of ulcerative keratitis, glaucoma and pigmentary keratitis in Chinese pugs in the present study was 60.71% 26.19% and 10.71% respectively. About 11.9% of the dogs had concurrent ulcerative keratitis and glaucoma and 2.38% of dogs had concurrent glaucoma and pigmentary keratitis. All the dogs were treated either medically and/or surgically.

Out of the 84 dogs presented with ocular diseases, cases of 22 dogs, the post treatment observations of which were available for a period of 60 days were selected for the study. Among the 22 Chinese pug dogs with ocular diseases selected for the study, 15 dogs had ulcerative keratitis, 10 dogs had glaucoma, five dogs had concurrent glaucoma and ulcerative keratitis, two dogs had pigmentary

keratitis, one dog each had concurrent pigmentary keratitis and glaucoma, cherry eye and, concurrent ulcerative keratitis and KCS.

The average age of the Chinese pug dogs with ulcerative keratitis and glaucoma selected in the study were 14.00 ± 2.45 and 18.10 ± 2.74 months respectively. Sex predisposition to glaucoma and age predisposition to KCS and pigmentary keratitis were noticed in selected cases. 90% of the dogs with glaucoma were females. KCS was noticed in a puppy with corneal ulcer and pigmentary keratitis was noticed in older dogs with eyelid and adnexal abnormalities. The previous treatment with corticosteroids in four dogs increased the risk of progression of ulcer and delayed healing.

Epiphora, blepharospasm, blepharoeidema and conjunctival hyperaemia were displayed by the dogs with ulcerative keratitis. A variable degree of buphthalmos and episcleral vessel congestion were noticed in all the dogs with glaucoma. Complete corneal pigmentation, xerophthalmia and mechanical irritation of cornea by either nasal fold trichiasis or medial lower lid entropion were noticed in dogs with pigmentary keratitis. In all the dogs with keratitis, relative exophthalmos and excessive corneal exposure greatly exaggerated the severity of corneal lesions. The physiological parameters, haematological parameters and random blood glucose levels of all the dogs were found to be within the normal range.

Corneal oedema was observed in all the ulcerative keratitis cases presented and progressive reduction was noticed during the subsequent observation with corneal healing. Corneal oedema that was noticed in a dog with glaucoma indicated endothelial damage. Vascularisation was noticed in all the dogs with stromal ulcers and full thickness defects. Vascularisation was found to increase in intensity by the 10th day in dogs which already had it and developed in dogs which did not show any vacularisation on the day of presentation. Pigmentation of cornea was noticed in most of the dogs presented with keratitis.

Specialized investigation tests like ophthalmoscopy and diagnostic tests like Schirmer tear test, fluorescein dye test, tonometry, exfoliative cytology of lacrimal smear and culture and sensitivity tests were found effective. The reflex uveitis associated with ulcerative keratitis and the hypopion and anterior synechiae associated with established uveitis were appreciated with ophthalmoscopic examination. Fluorescein dye test was helpful in assessing the depth, extent and healing of corneal lesions. The predisposition of Chinese pugs to KCS and tear film deficiencies increased the significance of Schirmer tear testing.

Culture and sensitivity test helped in definitive diagnosis and selection of appropriate antimicrobial therapy. The predominant isolate was Gram positive cocci (53.3%) with colony characteristics of *Staphylococcus* spp.. The other isolates included Gram negative bacilli (20%) and Gram positive bacilli. All the isolates were found sensitive to ciprofloxacin except that of two dogs which however showed clinical response to ciprofloxacin.

The medical treatment for ulcerative keratitis included topical instillation of ciprofloxacin, furbiprofen and tropicamide in addition to oral cephelexin and multivitamins. The topical antibiotic selected initially was ciprofloxacin and was continued in all the cases as culture and sensitivity and clinical response suggested the same. Flurbiprofen counteracted the clinically manifested anterior uveitis secondary to ulcerative keratitis. The use of topical analgesic and mydriatic agent, tropicamide helped to improve animal's comfort, reduced the postoperative pain and thereby the tendency for self-trauma. Administration of systemic antibiotic, cephalixin was effective in controlling the infection. Multivitamins promoted corneal epithelial healing. Topical 0.5% carboxy methyl cellulose sodium helped to control corneal drying and enabled protection against corneal desiccation and exposure keratopathy.

All the dogs with glaucoma were treated medically with topical 0.5% dorzolamide and 0.2% timolol maleate. All the dogs in the study showed considerable reduction in IOP to a safer level during the observation period.

Significant side effects of clinical importance were not noticed with topical carbonic anhydrase inhibitors and β -blockers during the period of treatment. Topical 0.5% carboxy methyl cellulose sodium helped to prevent corneal drying and exposure keratopathy due to buphthalmos.

For the dogs with KCS, topical lacrimomimetic, 0.5% carboxy methyl cellulose sodium and topical lacrimostimulant, 0.1% cyclosporine were advised. Topical instillation of cyclosporine improved the tear production. The use of 0.5% carboxy methyl cellulose, simulated natural tears, helped to improve lubrication and provided comfort until sufficient tear production was attained.

The medical treatment for dogs with pigmentary keratitis included topical ciprofloxacin, 0.1% cyclosporine and 0.5% carboxymethyl cellulose sodium. Even though topical cyclosporine improved the tear production, it could not control the progression of corneal pigmentation. Also, the pigmentary keratitis in these dogs was so dense and they remained blind throughout the observation period.

All the animals tolerated the medications and responded satisfactorily. Elizabethan collar was used in all the cases and were found to be useful in preventing self-trauma, until complete healing was observed

All the dogs with deep and full thickness corneal defects and eyelid and adnexal abnormalities were subjected to surgical treatment. All the surgical procedures were carried out under general anaesthesia with proper preoperative preparation. The anticholinergic treatment was beneficial in prevention or reversal of oculocardiac reflex. All the surgical procedures were conducted with the help of an operating microscope, with a magnification of 0.4X.

The surgical treatment for ulcerative keratitis included debridement and/or third eyelid flap or collagen sheet placement and temporary tarsorrhaphy. Iridectomy was done in full thickness defects. Nasal fold resection for nasal fold trichiasis, modified Hotz-Celsius operation for medial entropion and lateral lid shortening for exposed globe were done in dogs with concurrent pigmentary

keratitis. Removal of third eyelid gland was done in a dog with cherry eye. Postoperative medications were continued.

Debridement stimulated corneal healing and possessed little risk to the eye. Third eyelid flap was found to be useful for corneal ulcers where a large area was involved and provided direct support to the weakened cornea. Iridectomy reduced the chances of intraocular infections and anterior synechiae in full thickness defects.

The application of collagen sheet prepared from small intestine submucosa enabled to attain optimum results, preserved vision and promoted tissue healing with negligible scar formation. It acted as a scaffold for the repair and provided valuable tectonic support and helped in epithelialisation. Also, it helped to maintain corneal transparency and preservation of corneal integrity.

Temporary tarsorrhaphy that was done in all the surgical cases with ulcerative keratitis was found to be an effective bandaging technique after intraocular surgeries. Also, it helped in providing short-term protection of the surgical site and supported the corneal healing.

The lid shortening that was done in a dog with pigmentary keratitis improved the blink function and the tear film distribution and decreased the tendency for corneal desiccation. The smaller palpebral fissure thus created enabled better elimination of ocular mucus, hairs and debris, redistribution of the tear film lipid layer and reduced the evaporation rates over the central cornea and the opportunity for the development of ulcerations. Even though complete resolution of clinical signs could not be achieved, the technique improved the comfort of the patient.

The nasal fold reduction was effective in reducing chronic irritation and inflammation of cornea due to nasal fold trichiasis. Reduction of fold improved the comfort of the patient. The modified Hotz-Celsus operation reduced the degree of entropion.

Postoperative medications were continued in all the cases and all the animals recovered uneventfully.

The complications encountered were partial tarsorrhaphy suture disruption, dislodgement of third eyelid flap, hypopion, anterior synechiae and phthisis bulbi. Decrease in tear production was noticed after removal of prolapsed third eyelid gland.

The study concluded that the increased incidence of ocular diseases in Chinese pugs were due to the anatomical peculiarities and breed predisposition.

The Chinese pugs has prominent globe because of shallow orbits and large palpebral fissures. Their relative exophthalmos, in addition to poor corneal sensitivity or glaucoma resulted in poor blinking and central corneal tear film deficiency, led to excessive corneal exposure. This, along with the presence of concurrent KCS in juveniles, greatly exaggerated the severity of corneal lesions and increased the tendency to develop exposure keratitis, vascularization and ultimately, frank ulceration. The lid abnormalities like entropion and nasal fold trichiasis along with exposure due to large palpebral fissure, lagophthalmos, poor tear film distribution and presence of foreign materials resulted in corneal pigmentation.

Early diagnosis and proper treatment, together with adoption of protective and preventive measures to control exposure keratopathy, enabled the successful management of ocular diseases in Chinese pugs.

References

7. REFERENCES

- Anderson, J.F., Gelatt, K.N. and Farnsworth, R.J. 1976. A modified membrana nictitans flap technique for the treatment of ulcerative keratitis in cattle. *J. Am. Vet. Med. Assoc.* 168: 706-708
- Anoop, S., Chinchu, J., Syam, K.V. and Amma, T. S. 2010. Efficacy of collagen sheet for the management of staphyloma in dogs. In proceedings of world academy of science, engineering and technology, Singapore. 69: 131-133
- Barnett, K.C. 1985. The diagnosis and differential diagnosis of cataract in the dog. *J. Small Anim. Pract.* 26: 305-316
- Bath, G.S. and Dua, K. 2006a. Glaucoma in dogs. *Indian J. Vet. Med.* 26: 152-153
- Bath, G.S. and Dua, K. 2006b. Diagnosis and treatment of keratitis in dogs. *Indian J. Vet. Med.* 26: 160-161
- Bedford, P. 1980. Small Animal Clinic - Ophthalmic surgery in the dog and cat. *In Pract.* 2: 5-14
- Bedford, P.G.C. 1982. The diagnosis of ocular disease in the dog and cat. *Br. Vet. J.* 138: 93-119
- Bedford, P.G.C. 1987. Ocular emergencies in the dog and cat. *Br. Vet. J.* 143: 489-497
- Bentley, E. 2005. Spontaneous chronic corneal epithelial defects in dogs: A review. *J. Am. Anim. Hosp. Assoc.* 41: 158-165
- Bentley, E., Abrams, G.A., Covitz, D., Cook, C.S., Fischer, C.A., Hacker, D., Stuhr, C.M., Reid, T.W. and Murphy, C.J. 2001. Morphology and immunohistochemistry of spontaneous chronic corneal epithelial defects (SCCED) in dogs. *Invest. Ophthalmol. Vis. Sci.* 42: 2262-2269

- Bentley, E. and Murphy, C.J. 2004. Thermal cautery of the cornea for treatment of spontaneous chronic epithelial defects in dogs and horses. *J. Am. Vet. Med. Assoc.* 224: 250-253
- Berger, S.L. and King, V.L. 1998. The fluctuation of tear production in the dog. *J. Am. Anim. Hosp. Assoc.* 34: 79-83
- Bharathi, S., Raghavender, K.B.P. and Kumar, V.G. 2011. A study on the incidence of eye diseases in dogs. *Indian Vet. J.* 88: 20-21
- Bharathi, S., Raghavender, K.B.P., Kumar, V.G., Srilatha, C. and Rao, T.C.S. 2010. Diseases of the eyelid in dogs. *Indian J. Vet. Surgery.* 31: 146-148
- Blocker, C. and Woerdt, A. 2001. A comparison of corneal sensitivity between brachycephalic and domestic short-haired cats. *Vet. Ophthalmol.* 4: 127-130
- Bouhanna, L., Liscoet, L.B. and Letron, I.R. 2008. Corneal stromal sequestration in a dog. *Vet. Ophthalmol.* 11: 211-214
- Bowersox, J. and Croix, N.L. 2004. Examining the posterior segment of the eye in small animals. *Vet. Med.* 79: 800-804
- Bowman, D. D. and Atkins, C. E. 2009. Heart worm biology, treatment, and control. *Vet. Clin. Small Anim.* 39: 1127–1158
- Broadwater, J.J. Schorling, J.J, Herring, I.P. and Elvinger, F. 2008. Effects of body position on intraocular pressure in dogs without glaucoma. *Am. J. Vet. Res.* 69: 527-530
- Brooks, D.E., Komaromy, A.M. and Kallberg, M.E. 1999. Comparative optic nerve physiology: implications for glaucoma, neuroprotection, and neuroregeneration. *Vet. Ophthalmol.* 2: 13-25

- Brooks, D.E. and Ollivier, F.J. 2004. Matrix metalloproteinase inhibition in corneal ulceration. *Vet. Clin. Small Anim.* 34: 611-622
- Brunott, A., Boeve, M.H. and Velden., M.A. 2007. Grid keratotomy as a treatment for superficial nonhealing corneal ulcers in 10 horses. *Vet. Ophthalmol.* 10: 162-167
- Bussieres, M., Krohne, S.G., Stiles, J. and Townsend, W.M. 2004. The use of porcine small intestinal submucosa for the repair of full-thickness corneal defects in dogs, cats and horses. *Vet. Ophthalmol.* 7: 352-359
- Carrington, S.D., Bedford, P.G.C., Guillon, J.P. and Woodward, E.G. 1989. Biomicroscopy of the tear film: The tear film of the Pekingese dog. *Vet Rec.* 124: 323-328
- Carter, R.T. 2009. The role of integrins in corneal wound healing. *Vet. Ophthalmol.* 12: 2-9
- Chandler, H.L., Gemensky-Metzler, A.J. Bras, I.D., Robbin-Webb, T.E., Saville, W.J.A. and Colitz, C.M.H. 2010. In vivo effects of adjunctive tetracycline treatment on refractory corneal ulcers in dogs. *J. Am. Vet. Med. Assoc.* 237: 378-385
- Chinchu, J. 2010. Efficacy of collagen sheet for the management of corneal ulcers in dogs. MVSc. Thesis. Kerala Agricultural University. Thrissur. 110p
- Christmas, R. E. 1992. Common ocular problems of Shih Tzu dogs. *Can. Vet. J.* 33: 390-393
- Collins, B.K., Gross, M.E., Moore, C.P. and Branson, K.R. 1995. Physiologic, pharmacologic and practical considerations for anesthesia of domestic animals with eye diseases. *J. Am. Vet. Med. Assoc.* 207: 220-230

- Cook, C.S. 1997. Surgery for glaucoma. *Vet. Clin. Small Anim.* 27: 1109-1129
- Crispin, S.M. 1988. Uveitis in the dog and cat. *J. Small Anim. Pract.* 29: 429-447
- Crispin, S., Gould, D., Ellis, S., Mould, J. and Renwick, P. 2008. Hereditary eye disease and the BVA/KC/ISDS Eye Scheme: an update. *In Pract.* 30:2-14
- Cullen, C.L., Ihle, S.L. Webb, A.A. and McCarrille, C. 2005. Keratoconjunctival effects of diabetes mellitus in dogs. *Vet. Ophthalmol* 8: 215-224
- Davidson, H.J., and Kuonen, V.J. 2004. The tear film and ocular mucins. *Vet. Ophthalmol* 7: 71-77
- Deehr, A.J. and Dubielzig, R.R. 1998. A histopathological study of iridociliary cyst and glaucoma in Golden Retrievers. *Vet. Ophthalmol.* 1: 153-158
- Ekesten, B. and Narfstrom, K. 1991. Correlation of morphologic features of the iridocorneal angle to intraocular pressure in Samoyeds. *Am. J. Vet. Res.* 52: 1875-1878
- Featherstone, H.J. and Sansom, J. 2000. Intestinal submucosa repair in two cases of feline ulcerative keratitis. *Vet. Record.* 146: 136-138
- Featherstone, H.J., Sansom, J. and Heinrich, C.L. 2001. The use of porcine small intestinal submucosa in ten cases of feline corneal disease. *Vet. Ophthalmol* 4: 147-153
- Felchle, L. and Urbanz, L.J. 2001. Examining the anterior segment of the eye in small animals. *Vet. Med.* 96: 792-799
- Geasey, S.D., Cerro, M.D., Aquavella, J.V. and Viola, R.S. 1992. Collagen shields as a vehicle for collecting and studying migratory cells on human corneas. *Invest. Ophthalmol Vis. Sci.* 33: 298-303
- Gelatt, K.N. 1981. The canine glaucomas. *Veterinary Ophthalmology* (ed. Gelatt, K.N.). First edition. Lea and Febiger, Philadelphia, pp. 390-434

- Gelatt, K.N. 1991. Ophthalmic examination and diagnostic procedures. *Veterinary Ophthalmology* (ed. Gelatt, K.N.). Second edition. Lea and Febiger, Philadelphia, pp. 195-235
- Gelatt, K.N. 1997. Visual disturbance: where do I look? *J. Small Anim. Pract.* 38: 328-335
- Gelatt, K.N. and Mackay, E.O. 1998. Distribution of intraocular pressure in dogs. *Vet. Ophthalmol.* 1: 109-114
- Gelatt, K.N. and Mackay, E.O. 2001. Changes in intraocular pressure associated with topical dorzolamide and oral methazolamide in glaucomatous dogs. *Vet. Ophthalmol.* 4: 61-67
- Gelatt, K.N. and Mackay, E.O. 2002. Effect of single and multiple doses of 0.2% brimonidine tartrate in the glaucomatous Beagle. *Vet. Ophthalmol.* 5: 253-262
- Gelatt, K.N. and Mackay, E.O. 2004. Prevalence of the breed-related glaucomas in pure-bred dogs in North America. *Vet. Ophthalmol.* 7: 97-111
- Gelatt, K.N. and Mackay, E.O. 2004. Secondary glaucomas in the dog in North America. *Vet. Ophthalmol.* 7: 245-259
- Gerding, P.A., McLaughlin, S.A. and Troop, M.W. 1988. Pathogenic bacteria and fungi associated with external ocular diseases in dogs: 131 cases (1981-1986). *J. Am. Vet. Med. Assoc.* 193: 242-244
- Gibson, T.E., Roberts, S.M., Severin, G.A., Steyn, P.F. and Wrigley, R.H. 1998. Comparison of gonioscopy and ultrasound biomicroscopy for evaluating the iridocorneal angle in dogs. *J. Am. Vet. Med. Assoc.* 213: 635-638
- Gilger, B.C., Ollivier, F. J. and Bentley, E. 2008. Disease and surgery of the canine cornea and sclera. *Essentials of Veterinary Ophthalmology* (ed. Gelatt, K.N.). Second edition. Wiley-Black well, Iowa, pp. 119-152

- Gilmour, M.A. and Lehenbauer, T.W. 2009. Comparison of tepoxalin, carprofen and meloxicam for reducing intraocular inflammation in dogs. *Am. J. Vet. Res.* 70: 902-907
- Gionfriddo, J.R. 1995a. Identifying and treating conjunctivitis in dogs and cats. *Vet. Med.* 90: 242-253
- Gionfriddo, J.R. 1995b. When red eyes are due to preocular tear film diseases. *Vet. Med.* 90: 256-264
- Gionfriddo, J.R. 1995c. Recognizing and managing acute and chronic cases of glaucoma. *Vet. Med.* 90: 265-275
- Gionfriddo, J.R. 1995d. The causes, diagnosis, and treatment of uveitis. *Vet. Med.* 90: 278-284
- Gionfriddo, J.R. and Powell, C.C. 2001. Traumatic glaucoma in a dog. *Vet. Med.* 96: 830-836
- Giuliano, E.A. 2004. Nonsteroidal anti-inflammatory drugs in veterinary ophthalmology. *Vet. Clin. Small Anim.* 34: 707-723
- Gum, G.G. 1991. Physiology of the eye. *Veterinary Ophthalmology* (ed. Gelatt, K.N.). Second edition. Lea and Febiger, Philadelphia, pp. 124-161
- Hamor, R.E. 2001. Techniques for collection and interpretation of tissue samples in ocular disease. *Clin. Tech. Small Anim. Pract.* 16: 17-21
- Hamor, R. E., Roberts, S.M., Severin, G.A. and Chavkin, M.J. 2000. Evaluation of results for Schirmer tear tests conducted with and without application of a topical anaesthetic in clinically normal dogs of 5 breeds. *Am. J. Vet. Res.* 61: 1422-1425

- Hansen, P.A. and Guandalini, A. 1999. A retrospective study of 30 cases of frozen lamellar corneal graft in dogs and cats. *Vet. Ophthalmol.* 2: 231-244
- Hartley, C. 2010. Treatment of corneal ulcers. When is surgery indicated? *J. Feline Med. Surg.* 12: 398-405
- Hartley, C., Williams, D.L. and Adams, V.J. 2006. Effect of age, gender, weight, and time of day on tear production in normal dogs. *Vet. Ophthalmol.* 9: 53-57
- Hasegawa, T., Doki, K. and Yanase, J. 2001. Long term management of a glaucomatous eye in a dog treated with medical therapy alone. *J. Vet. Med. Sci.* 63: 1323-1325
- Helper, L.C. 1981. Canine nictitating membrane and conjunctiva. *Veterinary ophthalmology* (ed. Gelatt, K.N.). Second edition. Lea and Febiger, Philadelphia. pp: 330-342
- Helper, L.C. 1989. *Magrane's Canine Ophthalmology*. Fourth edition. Lea and Febiger, Philadelphia. 297p
- Hendrix, D.V.H. and Cox, S.K. 2008. Pharmacokinetics of topically applied ciprofloxacin in tears of mesocephalic and brachycephalic dogs. *Vet. Ophthalmol.* 11: 7-10
- Hendrix, D.V.H., Ward, D.A. and Barnhill, M.A. 2001. Effects of antibiotics on morphologic characteristics and migration of canine corneal epithelial cells in tissue culture. *Am. J. Vet. Res.* 62: 1664-1669
- Herrera, D. 2005. Surgery of the eyelids. In proceedings of the 30th world congress of the World Small Animal Veterinary Association, Mexico City, Mexico.

- Herring, I.P. 2003. Corneal surgery: Instrumentation, patient considerations, and surgical principles. *Clin. Tech. Small Anim. pract.* 18: 152-160
- Hollingsworth, S.R. 2003. Corneal surgical procedures. *Clin. Tech. Small Anim. pract.* 18: 161-167
- Janssens, G. 2007. Indolent ulcers in dogs' eyes. *Euro. J. Compan. Anim. Pract.* 17: 280-284
- Johnson, B.C. and Miller, W.W. 1990. Recognizing ocular signs of systemic diseases in dogs. *Vet. Med.* 85: 1076-1090
- Jones, G. 2004. Ophthalmic examination equipment- toy or tool? *In Pract.* 26: 332-335
- Kallberg, M.E., Brooks, D.E., Gelatt, K.N., Garcia-Sanchez, G.A., Szabo, N.J. and Lambrou, G.N. 2007. Endothelin-1, nitric oxide, and glutamate in the normal and glaucomatous dog eye. *Vet. Ophthalmol.* 10: 46-52
- Kaswan, R.L., Bounous, D. and Hirsh, S.G. 1995. Diagnosis and management of keratoconjunctivitis sicca. *Vet. Med.* 90: 539-560
- Kaswan, R. L. and Martin, C. L. 1985. Surgical correction of third eyelid prolapse in dogs. *J. Am. Vet. Med. Assoc.* 186: 83
- Kato, K., Sasaki, N., Matsunaga, S., Nishimura, R. and Ogawa, H. 2006. Incidence of canine glaucoma with goniodysplasia in Japan: A retrospective study. *J. Vet. Med. Sci.* 68: 853-858
- Kim, J.Y., Won, H. and Jeong, S. 2009. A retrospective study of ulcerative keratitis in 32 dogs. *Intern. J. Appl. Res. Vet. Med.* 7: 27-31
- Krawitz, L. 1963. Practical anatomy and physiology of the canine eye. *J. Am. Vet. Med. Assoc.* 142: 770-775

- Krohne, S. G. 2008. Medial canthus syndrome in dogs – Chronic tearing, pigment, medial entropion and trichiasis. In *Corneal diseases*. Proceedings of international symposium by Schering-Plough Animal Health. pp 1- 14
- Lavach, J.D., Thrall, M.A., Benjamin, M.M. and Severin, G.A. 1977. Cytology of normal and inflamed conjunctivas in dogs and cats. *J. Am. Vet. Med. Assoc.* 170: 722-72
- Ledbetter, E.C., Hendricks, L.M., Riis, R.C. and Scarlett, J.M. 2007. Invitro fluroquinolone susceptibility of *Pseudomonas aeruginosa* isolates from dogs with ulcerative keratitis. *Am. J. Vet. Res.* 68: 638-642
- Ledbetter, E.C., Mun, J.J., Kowbel, D. and Fleiszig, S.M.J. 2009. Pathogenic phenotype and genotype of *Pseudomonas aeruginosa* isolates from spontaneous canine ocular infections. *Invest. Ophthalmol. Vis. Sci.* 50: 729-736
- Ledbetter, E.C., Munger, R.J., Ring, R.D. and Scarlett, J.M. 2006. Efficacy of two chondroitin sulphate ophthalmic solutions in the therapy of spontaneous chronic corneal epithelial defects and ulcerative keratitis associated with bullous keratopathy in dogs. *Vet. Ophthalmol.* 9: 77-87
- Maehara, S., Ono, K., Ito, N., Tsuzuki, K., Seno, T., Yokoyama, T., Yamashita, K., Izumisawa, Y. and Kotani, T. 2004. Effects of topical nipradilol and timolol maleate on intraocular pressure, facility of outflow, arterial blood pressure and pulse rate in dogs. *Vet. Ophthalmol.* 7: 147-150
- Magrane, W.G. 1977. *Canine Ophthalmology*. Third edition. Lea & Febiger, Philadelphia. 305p
- Malmberg, G.J. and Lupo, R. 2004. Compounding in veterinary ophthalmology. *Vet. Clin. Small Anim.* 34: 825-838

- Mandell, D.C. and Holt, E. 2005. Ophthalmic emergencies. *Vet. Clin. Small Anim.* 35: 455-480
- Mangan, B.G., Al-Yahya, K., Chen, C., Gionfriddo, J.R., Powell, C.C., Dubielzig, R.R., Ehrhart, E.J. and Madl, J.E. 2007. Retinal pigment epithelial damage, breakdown of the blood–retinal barrier, and retinal inflammation in dogs with primary glaucoma. *Vet. Ophthalmol.* 10: 117-124
- Martin, C.L. 1971. Effect of vitamin-A, antibiotic, mineral oil and subconjunctival corticosteroid on corneal epithelial wound healing in the dog. *J. Am. Vet. Med. Assoc.* 159: 1392-1399
- Martin, C.L. 2001. Evaluation of patients with decreased vision or blindness. *Clin. Tech. Small Anim. Pract.* 16: 62-70
- Massa, K.L., Gilger, B.C., Miller, T.L. and Davidson, M.G. 2002. Causes of uveitis in dogs: 102 cases (1989-2000). *Vet. Ophthalmol.* 5: 93-98
- Massa, K.L., Murphy, C.J., Hartmann, F.A., Miller, P.E., Korsower, C.S. and Young, K.M. 1999. Usefulness of aerobic microbial culture and cytologic evaluation of corneal specimens in the diagnosis of infectious ulcerative keratitis in animals. *J. Am. Vet. Med. Assoc.* 215: 1671-1674
- Metzger, F.L. and Rebar, A. 2004. Three-minute peripheral blood film evaluation: preparing the film. *Vet. Med.* 99: 1020-1024
- Miller, P.E. 2003. Glaucoma. *TextBook of Small Animal Surgery Vol. II* (Ed. Slatter, D.). Third edition. W.B. Saunders Co., Philadelphia. pp 1454-1477
- Miller, W.W. 1996. Using polysulphated glycosaminoglycans to treat persistent corneal erosions in dogs. *Vet. Med.* 91: 916-922

- Miller, W.W. 2001. Evaluation and management of corneal ulcerations: A systematic approach. *Clin. Tech. Small Anim. Pract.* 16: 51-57
- Miller, W.W. and Crenshaw, K.L. 1988. The basics of in-clinic ophthalmic examinations. *Vet. Med.* 83: 1154- 1161
- Mitchell, N. 2011. Approach to ocular examination in small animals. *In Pract.* 33: 146-154
- Montiani-Ferreira, F., Cardoso, F.F. and Peterson-Jones, S. 2004. Basic concepts in statistics for veterinary ophthalmologists. *Vet. Ophthalmol.* 7: 79-85
- Moore, P.A. 2001. Examination techniques and interpretation of ophthalmic findings. *Clin. Tech. Small Anim. Pract.* 16: 1- 12
- Moore, P.A. 2003. Diagnosis and management of chronic corneal epithelial defects (indolent corneal ulcerations). *Clin. Tech. Small Anim. Pract.* 18: 168-177
- Morgan, R. V. 2004. Common corrective and protective eyelid surgeries. *Vet. Med.* 99: 354-373
- Morgan, R. V. 2004. Preparations for eyelid surgeries. *Vet. Med.* 99: 346-350
- Morgan, R. V., Duddy, J. M., and McChung, K. 1993. Prolapse of the gland of the third eyelid in dogs: a retrospective study of 89 cases (1980-1990). *J. Am. Anim. Hosp. Assoc.* 29: 56-60
- Morreale, R.J. 2003. Corneal diagnostic procedures. *Clin. Tech. Small Anim. Pract.* 18: 145-151

- Mughannam, A.J., Cook, C.S. and Fritz, C.L. 2004. Change in intraocular pressure during maturation in Labrador Retrievers. *Vet. Ophthalmol.* 7: 87-89
- Munro, E. 2001. Advances in ocular therapeutics in dogs and cats. *In Pract.* 23: 316-327
- Murphy, C.J., Marfurt, C.F., McDermott, A., Bentley, E., Abrams, G.A., Reid, T.W. and Campbell, S. 2001. Spontaneous chronic corneal epithelial defects (SCCED) in dogs: Clinical Features, innervation, and effect of topical SP, with or without IGF-1. *Invest. Ophthalmol. Vis. Sci.* 42: 2252- 2261
- Ollivier, F.J. 2003. Bacterial corneal diseases in dogs and cats. *Clin. Tech. Small Anim. Pract.* 18: 193-198
- Ollivier, F.J., Gilger, B.C., Barrie, K.P., Kallberg, M.E., Plummer, C.E., O'Reilly, S., Gelatt, K.N. and Brooks, D.E. 2007. Proteinases of the cornea and precocular tear film. *Vet. Ophthalmol.* 10: 199-206
- Ollivier, F.J., Plummer, C.E. and Barrie, K.P. 2008. Ophthalmic examination and diagnostics. *Essentials of Veterinary Ophthalmology* (ed. Gelatt, K.N.). Second edition. Wiley-Black well, Iowa, pp. 3-22
- Palmer, C.L.E., Byers, C.G. and Caruso, K. 2008. Acute glaucoma. *Comp. Cont. Educ. Pract. Vet.* 10: 1-8
- Peiffer, R.L. and Gelatt, K.N. 1980. Aqueous humor outflow in Beagles with inherited glaucoma: Gross and light microscopic observations of the iridocorneal angle. *Am. J. Vet. Res.* 41: 861-867
- Peiffer, R.L., Gelatt, K.N., Jessen, C. R., Gum, G.G., Gwin, R.M. and Davis, J. 1977. Calibration of the Schiottz tonometer for the normal canine eye. *Am. J. Vet. Res.* 38: 1881-1889

- Peiffer, R.L., Nasisse, M.P., Cook, C.S. and Harling, D.E. 1987. Surgery of the canine and feline orbit, adnexa and globe part 6: Surgery of the cornea. *Companion Anim. Pract.* 1: 3-13
- Pirie, C.G., Maranda, L.S. and Pizzirani, S. 2011. Effect of topical 0.03% flurbiprofen and 0.005% latanoprost, alone and in combination, on normal canine eyes. *Vet. Ophthalmol.* 14: 71-79
- Plummer, C. E., Mackay, E.O. and Gelatt, K.N. 2006. Comparison of the effects of topical administration of a fixed combination of dorzolamide-timolol to monotherapy with timolol or dorzolamide on IOP, pupil size, and heart rate in glaucomatous dogs. *Vet. Ophthalmol.* 9: 245-249
- Prado, M.R., Rocha, M.F.G., Brito, E.H.S., Girao, M.D., Monteiro, A.J., Teixeira, M.F.S. and Sidrim, J.J.C. 2005. Survey of bacterial microorganisms in the conjunctival sac of clinically normal dogs and dogs with ulcerative keratitis in Fortaleza, Ceara, Brazil. *Vet. Ophthalmol.* 8: 33-37
- Priya, P. 2009. Evaluation and management of glaucoma in dogs. MVSc. Thesis. Kerala Agricultural University. Thrissur. 93p
- Raji, T.A. 2006. Processed collagen graft for the treatment of corneal lesions in dogs. MVSc. Thesis. Kerala Agricultural University. Thrissur. 73p
- Renwick, P. 1996. Diagnosis and treatment of corneal disorders in dogs. *In. Pract.* 18: 315-328
- Renwick, P. 2007. Eyelid surgery in dogs. *In. Pract.* 29: 256-271
- Resmi, T.S. 2008. Evaluation and management of keratitis in dogs. MVSc. Thesis. Kerala Agricultural University. Thrissur. 100p
- Roberts, S.R. 1954. The nature of corneal pigmentation in the dog. *J. Am. Vet. Med. Assoc.* 124: 208-211

- Roberts, S.M., Severin, G.A. and Lavach, J.D. 1986. Antibacterial activity of dilute povidone-iodine solutions used for ocular surface disinfection in dogs. *Am. J. Vet. Res.* 47: 1207-1210
- Rodrigues, G.N., Laus, J.L., Santos, J.M., Rigueiro, M.P. and Smith, R.L. 2006. Corneal endothelial cell morphology of normal dogs in different ages. *Vet. Ophthalmol.* 9: 101-107
- Salisbury, M.A.R., Kaswan, R.L. and Brown, J.B. 1995. Microorganisms isolated from the corneal surface before and during topical cyclosporine treatment in dogs with keratoconjunctivitis sicca. *Am. J. Vet. Res.* 56: 880-884
- Sansom, J. 1988. Antibacterials in the treatment of ocular infections. *J. Small Anim. Pract.* 29: 487-499
- Sansom, J. 2000. Diseases involving the anterior chamber of the dog and cat. In *Pract.* 22: 58-70
- Sansom, J. and Barnett, K.C. 1985. Keratoconjunctivitis sicca in the dog: a review of two hundred cases. *J. Small Anim. Pract.* 26: 121-131
- Schalm, O.W., Feldman, B.F., Zinkl, J.G. and Jain, N.C. 2000. *Veterinary Haematology*. Fifth edition. Lippincott Williams and Wilkins, Philadelphia. 1344p
- Schoofs, S. 1999. Prolapse of the gland of the third eyelid in a cat: a case report and literature review. *J. Am. Anim. Hosp. Assoc.* 35: 240-242

- Slatter, D. and Dietrich, U. 2003. Cornea and Sclera (Ed. Slatter, D.). *TextBook of Small Animal Surgery Vol. II*. Third edition. W.B. Saunders Co., Philadelphia. pp 1368-1396
- Slater, M.R. and Erb, H.N. 1986. Effects of risk factors and prophylactic treatment on primary glaucoma in the dog. *J. Am. Vet. Med. Assoc.* 188: 1028-1030
- Smedes, S.L. and Dubielzig, R.R. 1994. Early degenerative changes associated with spontaneous glaucoma in dogs. *J. Vet. Diagn. Invest.* 6: 259-263
- Smith, L.N., Miller, P.E. and Felchle, L.M. 2010. Effects of topical administration of latanoprost, timolol, or a combination of latanoprost and timolol on intraocular pressure, pupil size, and heart rate in clinically normal dogs. *Am. J. Vet. Res.* 71: 1055-1061
- Stanley, R.G., Hardman, C. and Johnson, B.W. 1998. Results of grid keratotomy, superficial keratotomy and debridement for the management of persistent corneal erosions in 92 dogs. *Vet. Ophthalmol.* 1: 233-238
- Stanley, R.G. and Kaswan, R.L. 1994. Modification of the orbital rim anchorage method for surgical replacement of the gland of the third eyelid in dogs. *J. Am. Vet. Med. Assoc.* 205: 1412-1414
- Startup, F.G. 1984. Corneal ulceration in the dog. *J. Small. Anim. Pract.* 25: 737-752
- Stiles, J., Honda, C.N., Krohne, S.G. and Kazacos., E.A. 2003. Effect of topical administration of 1% morphine sulphate solution on signs of pain and corneal wound healing in dogs. *Am. J. Vet. Res.* 64: 813-818

- Storm, A.R., Hassig, M., Iburg, T.M. and Spiess, B.M. 2011. Epidemiology of canine glaucoma presented to University of Zurich from 1995 to 2009. Part 2: secondary glaucoma (217 cases). *Vet. Ophthalmol.* 14:127-132
- Sweeny, C.R. and Irby, N.L. 1996. Topical treatment of *Pseudomonas* sp-infected corneal ulcers in horses: 70 cases (1977-1994). *J. Am. Vet. Med. Assoc.* 209: 954-957
- Tolar, E.L., Hendrix, D.V.H., Rohrbach, B.W., Plummer, C.E., Brooks, D.E. and Gelatt, K.N. 2006. Evaluation of clinical characteristics and bacterial isolates in dogs with bacterial keratitis: 97 cases (1993-2003). *J. Am. Vet. Med. Assoc.* 228: 80-85
- Townsend, W.M. 2007. Ophthalmic drugs: what's new?. *Vet. Med.* 102: 540-545
- Townsend, W., Bedford, P. and Jones, G. 2009. Abnormal appearance (eds. Peiffer, R. and Jones, P.). *Small Animal Ophthalmology - A problem oriented approach*. Fourth edition. Saunders- Elsevier, Philadelphia, pp. 67-113
- Vanore, M., Chahory, S., Payen, G. and Clerc, B. 2007. Surgical repair of deep melting ulcers with porcine small intestinal submucosa (SIS) graft in dogs and cats. *Vet. Ophthalmol.* 10: 93-99
- Venugopal, S. K. 2011. Management of corneal injuries in Chinese pugs. *Indian J. Canine Pract.* 3: 52-56
- Wang, L., Pan, Q., Xue, Q., Cui, J. and Qi, C. 2008. Evaluation of matrix metalloproteinase concentrations in precorneal tear film from dogs with *Pseudomonas aeruginosa* -associated keratitis. *Am. J. Vet. Res.* 69: 1341-1345

- Wang, L., Pan, Q., Zhang, L., Xue, Q., Cui, J. and Qi, C. 2008. Investigation of bacterial microorganisms in the conjunctival sac of clinically normal dogs and dogs with ulcerative keratitis in Beijing, China. *Vet. Ophthalmol* 11: 145-149
- Whitley, R.D. 2000. Canine and feline primary ocular bacterial infections. *Vet. Clin. Small Anim. Pract.* 30: 1151-1167
- Whitley, R.D., McLaughlin, S.A. and Gilger, B.C. 1995. Update on eye disorders among purebred dogs. *Vet. Med.* 90: 574-592
- Whitley, R.D., McLaughlin, S.A., Gilger, B.C. and Lindley, D.M. 1991. The treatments for keratoconjunctivitis sicca. *Vet. Med.* 86: 1076-1093
- Wilcock, B.P. 1993. The eye and ear. *Pathology of Domestic Animals*. (eds. Jubb, K.V.F., Kennedy, P.C. and Palmer, N.). Fourth edition. Academic Press, San Diego, pp. 441-529
- Wilkie, D.A. and Latimer, C.A. 1991. Effects of topical administration of timolol maleate on intraocular pressure and pupil size in dogs. *Am. J. Vet. Res.* 52: 432-435
- Wilkie, D.A. and Whittaker, C. 1997. Surgery of the cornea. *Vet. Clin. Small Anim.* 27: 1097-1107
- Williams, D.L. 2008. Immunopathogenesis of keratoconjunctivitis sicca in the dog. *Vet. Clin. Small Anim.* 38: 251-268
- Williams, D.L., Pierce, V., Mellor, P. and Heath, M.F. 2007. Reduced tear production in three canine endocrinopathies. *J. Small Anim. Pract.* 48: 252-256
- Willis, A.M., Diehl, K.A. and Robbin, T.E. 2002. Advances in topical glaucoma therapy. *Vet. Ophthalmol.* 5: 9-17

Woerd, A.V. 2001. The treatment of acute glaucoma in dogs and cats. *J. Vet. Emerg. Crit. Care.* 11: 199-205

Wolfer, J. and Grahn, B. 1994. Diagnostic ophthalmology. *Can. Vet. J.* 35: 124-125

**EVALUATION AND MANAGEMENT OF EYE
AFFECTIONS IN CHINESE PUGS**

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**Abstract of the thesis submitted in partial fulfillment of the
requirement for the degree of**

**MASTER OF VETERINARY SCIENCE IN
VETERINARY SURGERY AND RADIOLOGY**

**Faculty of Veterinary and Animal Sciences
Kerala Veterinary and Animal Sciences University**

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ABSTRACT

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The study was conducted in Chinese pugs to evaluate the occurrence of different ophthalmic diseases, the clinical signs associated with each condition, and the effectiveness of the various treatments adopted. The study was conducted during the period from June 2011 to May 2012.

Among 578 Chinese pugs presented, 84 dogs had ocular affections and accounted 14.53% of the total dogs presented. Among the 84 Chinese pug dogs, 40 dogs had ulcerative keratitis, 10 had glaucoma, 10 had concurrent glaucoma and ulcerative keratitis, 12 dogs had conjunctivitis, seven dogs had pigmentary keratitis, two dogs had concurrent pigmentary keratitis and glaucoma and one dog each had proptosis, cherry eye and, concurrent ulcerative keratitis and keratoconjunctivitis sicca (KCS). All the dogs were treated either medically and/or surgically.

Out of the 84 dogs presented with ocular diseases, cases of 22 dogs, the post treatment observations of which were available for a period of 60 days were selected for the study. Sex predisposition to glaucoma and age predisposition to KCS and pigmentary keratitis were noticed in selected cases. 90% of the dogs with glaucoma were females. KCS was noticed in a puppy with corneal ulcer and pigmentary keratitis was noticed in older dogs with eyelid and adnexal abnormalities.

The medical treatment for ulcerative keratitis included topical instillation of ciprofloxacin, furbiprofen and tropicamide in addition to oral cephelexin and multivitamins. All the dogs with glaucoma were treated medically with topical 0.5% dorzolamide and 0.2% timolol maleate. Topical 0.5% carboxy methyl cellulose sodium was also advised. For dogs with KCS, topical 0.5% carboxy methyl cellulose sodium and 0.1% cyclosporine were advised. The treatment for pigmentary keratitis included topical ciprofloxacin, 0.1% cyclosporine and 0.5% carboxymethyl cellulose sodium. All the animals tolerated the medications and

responded satisfactorily. Topical cyclosporine improved tear production but couldn't control the progression of corneal pigmentation.

The surgical treatment for ulcerative keratitis included debridement and/or third eyelid flap or collagen sheet placement and temporary tarsorrhaphy. Iridectomy was done in full thickness defects. Nasal fold resection for nasal fold trichiasis, modified Hotz-Celsus operation for medial entropion and lateral lid shortening for exposed globe were done in dogs with concurrent pigmentary keratitis. Removal of third eyelid gland was done in a dog with cherry eye. All the surgical procedures were done under general anaesthesia after preoperative topical medication. Postoperative medications were continued. All the animals recovered uneventfully.

The complications encountered were partial disruption of tarsorrhaphy sutures, dislodgement of third eyelid flap, hypopion, anterior synechiae and phthisis bulbi. The excision of prolapsed third eyelid gland reduced the tear production.

The study concluded that the increased incidence of ocular diseases in Chinese pugs were due to the excessive globe prominence because of shallow orbits and large palpebral fissures. Their relative exophthalmos, in addition to poor corneal sensitivity or glaucoma resulted in poor blinking and central corneal tear film deficiency, led to excessive corneal exposure. This, along with the presence of concurrent KCS in juveniles, greatly exaggerated the severity of corneal lesions and increased the tendency to develop exposure keratitis, vascularization and ultimately, frank ulceration. The lid abnormalities like entropion and nasal fold trichiasis along with exposure due to large palpebral fissure, lagophthalmos, poor tear film distribution and presence of foreign materials resulted in corneal pigmentation.

Early diagnosis and proper treatment, together with adoption of protective and preventive measures to control exposure keratopathy, enabled the successful management of ocular diseases in Chinese pugs.

