DIAGNOSIS AND MANAGEMENT GUIDE FOR KERATOCONJUNCTIVITIS SICCA IN PETS

Keratoconjunctivitis Sicca (KCS) is an inflammatory disease of the ocular surface (cornea and conjunctiva) secondary to the deficiency of some of the phases of the tear film (1) and which has a prevalence in dogs of 0.4% according to a study recent study carried out by the BSAVA (British Small Animal Veterinary Association) (2).

THE TEAR LAYER

The tear film provides lubrication and hydration to the ocular surface. It is a source of oxygen, immunoglobulins, lysozymes, lactoferrin and defensins. The tear film is made up of three different layers.

The mammalian tear film is made up of three layers: the innermost layer is composed of mucin, which is secreted by conjunctival goblet cells and whose function is to provide a smooth corneal surface to improve the spread of the tear film (3). The intermediate layer is the aqueous one, secreted by the lacrimal glands and responsible for the main metabolic and defensive functions of the tear (4).

Finally, the outermost layer is the lipid layer, secreted by the meibomian glands and whose main function is to limit evaporation, improve the adhesion of the tear film to the cornea and provide the necessary surface tension to prevent tear overflow (1).

KERATOCONJUNCTIVITIS SICCA (KCS)

Several different types of KCS can be identified depending on the layer of the tear film that is affected, although in any of them, the key pathological mechanism of the pathology will be tear hyperosmolarity, which will damage the ocular surface by causing an inflammatory process.

When there is a reduction in the production of the aqueous component of the tear film under normal evaporation conditions, it will be quantitative keratoconjunctivitis sicca. If there is excessive tear evaporation in the presence of a functional lacrimal gland, we will speak of qualitative keratoconjunctivitis sicca (5).

In any case, on many occasions in which there is a dysfunction of the lacrimal glands with reduced tear secretion, we will find in these patients a hybrid form in which both characteristics of aqueous deficiency and increased evaporation will be present (5).

In pets, the qualitative form can be observed in any breed, although it will be more common in brachiocephalic animals with lagophthalmos (inability to completely close one or both eyelids) or in pets in which, due to a deficiency in the lipid layer of tear film, greater loss occurs due to evaporation (6).

Dysfunctions in the meibomian glands as a result of marginal blepharitis, blepharoconjunctivitis, meibomianitis and dermatological diseases are the main causes that have been associated in both pets and humans for the qualitative form of the disease (7).

Even though it is relatively common, qualitative KCS often goes unnoticed in general clinics because the results of the Schirmer test (STT-1) are usually within normal values and the tests recommended for its diagnosis such as Tear Break-Up Time (TBUT) or the OSA-VET® (veterinary ocular surface analyzer) (1), are less widely used in non-specialized centers.

Therefore, many patients who suffer from this variant of the disease do not receive adequate and effective treatment, worsening their symptoms and quality of life.

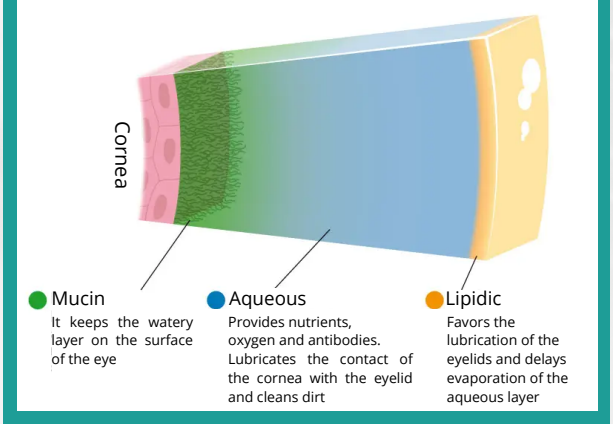

Figure 1. Layer scheme of the tear

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PATHOPHYSIOLOGY OF THE DISEASE

Tear deficiencies lead to chronic inflammation of the ocular surface, secondary infections, dehydration and malnutrition of the corneal and conjunctival epithelium. This pathology can progress to an ulcer or infection.

Chronic inflammation of the ocular surface will cause conjunctival hyperemia, squamous metaplasia of the surface epithelium, and thickening of the corneal epithelium. Inflammatory cells and blood vessels penetrate the anterior stroma, depositing pigments, lipids, and calcium. These components stabilize the cornea to prevent possible ulceration, although their presence can cause loss of visual acuity.

KCS QUANTITATIVE

There are a variety of causes for the development of a quantitative KCS, including:

- Chronic conjunctivitis
- Immune-mediated lacrimal adenitis
- Distemper virus
- Leishmaniasis
- Congenital
- Iatrogenic
- Trauma to tear glands or nerves

Any alteration that may affect the production or secretion of tears will cause this quantitative KCS.

The diagnosis will be based on a reduction in the value in the Schirmer test (normal values should be greater than 15mm/min).

KCS QUALITATIVE

Some of the causes of this form of KCS are:

- Chronic blepharitis with meibomianitis causing reduced lipid production
- Infectious causes such as Staphylococcus, Candida or Malassezia
- Reduction in the number of goblet cells and their mucin layer

Clinical signs of the disease include blepharospasm, corneal neovascularization, and increased production of ocular mucus.

The diagnosis, as we have already mentioned, will be based on the TBUT (Tear Break-Up Time) test, since the Schirmer test has normal values in these patients. To perform the TBUT test, a drop of fluorescein is applied to the eye while holding the eyelids open. Once the cornea has been impregnated with fluorescein, the eye is closed, opened and illuminated with a cobalt blue lamp.

he result of TBUT will be the time that tear stability is maintained without any breakage of the fluorescein layer.

The normal TBUT result should be greater than 20 seconds. Some quantitative KCS may have TBUT <5s which indicates a qualitative deficit in turn (8).

Table 1. Structure of the tear film and its deficiencies

	Production	Function	Type of deficiency	Diagnostic test
Lipidic	Meibomian glands	Limits evaporation Binds tear film to cornea Provides surface tension to prevent tear film overflow	Qualitative	Decrease in TBUT
Aqueous	Lacrimal glands	Provides corneal nutrition, surface lubrication, and smooth surface for optical clarity Removes waste material and bacteria	Quantitative	Decrease in Schirmer test
Mucin	Goblet cells	Enhances spread of tear film	Qualitative	Decrease in TBUT

MANAGEMENT OF THE KCS

Medical therapy is used in both quantitative and qualitative KCS and is based on the use of tear secretion stimulants and tear supplementation through eye drops. Topical antibiotics and anti-inflammatories are also commonly used. In most patients with KCS, long-term, even chronic, topical therapy is required and the client has to be well aware of this disease.

The stimulation of tear secretion is normally achieved with drugs such as Cyclosporine, Tacrolimus or Pilocarpine in eye drops (9,10).

Tear replacement therapy provides lubrication until secretion stimulants are effective. Tear replacement therapy may be necessary for life in pets that do not respond to the drugs listed above. These medications are available in the form of solutions, gels, and ointments, and have a wide variety of components.

More recently, especially focused on qualitative KCS, veterinary nutraceuticals with Omega-3 fatty acids (DHA/EPA), lactoferrin, vitamins (C and E) and minerals (Zinc and Copper) have begun to be used, such as Lacrimalis+ from Dr+Vet® as a complement to the previously exposed treatments.

The oral intake of fatty acids helps reduce inflammation of the meibomian glands and helps improve the quality of the tear lipid layer. In the comparison of clinical cases carried out by Paco Simó and María Simó of the IVO, the results of the use of the product are reflected (11).

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